

TEXTBOOK OF SURGERY

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Evolution of Modern Surgery

Preface to Third Edition

The completion of the materials for this third edition is significant, as the coverage and content approximate for the first time that intended when the project was originally planned in 1945. We have profited greatly from the constructive suggestions obtained during the past year in response to a questionnaire sent to teachers in all medical schools of Canada and the United States. Their comments, together with those of numerous teachers and students at home and abroad, have helped to establish the underlying editorial policy which is reflected in this new volume. For all such assistance the editor wishes to express his sincere gratitude.

The reader and reviewer will note that the materials have been completely revised, expanded, and brought up to date. In addition, an important new chapter on Pediatric Surgery, written by Dr. Gordon M. Karn and Dr. Harvey E. Beardmore of The Montreal Children's Hospital, has been included in this edition. This indicates a departure from past policy which tended to restrict the contributors to the staff of the Royal Victoria Hospital and certain allied departments of McGill University, whereas the editor's original plan was to make this textbook representative of the teaching and practice of the three hospitals associated with the surgical staff of McGill University.

Since the most rapid changes have occurred in the surgery of the heart, great vessels, and lungs, these sections have been completely rewritten and extended. Dr. Arthur M. Vineberg and Dr. David R. Murphy have contributed the new chapter on Cardiac Surgery, and in this section valuable assistance has been given by Dr. A. Dobell and Dr. G. D. Hooper. Dr. Darrell D. Munro collaborated in extending and revising the chapter on Thoracic Surgery.

Additional new contributors are Dr. Lloyd G. Stevenson, who assisted in the chapter on Evolution of Modern Surgery; Dr. Gardner C. McMillan, who has rewritten the chapter on Inflammation and Repair; Dr. Roger W. Reed, who has joined Dr. Gertrude G. Kalz in bringing the chapter on Surgical Bacteriology and Chemotherapy up to date; Dr. Alan B. Noble and Dr. Philip R. Bromage, who have collaborated with Dr. Richard G. B. Gilbert in the chapter on Anesthesia; Dr. William R. Foote, who has worked with Dr. George A. Simpson on the enlarged chapter on the Female Genital Tract, which replaces the restricted coverage of the Abdominal Aspects of Gynecology in the first two editions, and Dr. Martin A. Entin, who has written the section on Open Wounds and Soft Tissue Injuries of the Hand that is included in the editor's chapter on Fractures and Disorders of the Upper Extremity. Dr. Edward J. Tabah has extended his coverage of the Face, Mouth, and Neck and has assisted Dr. Munro and the editor with the enlarged chapter on the Esophagus.

In compliance with repeated requests, the editor has extended the coverage of the ankle and foot to correspond with that for the hip and knee and, for this, twenty-one additional color plates have been included. Five further color plates bearing on the surgery of the great vessels will be found in Dr. Josephus C. Luke's section with the kind permission of Dr. M. E. DeBakey. Four additional color plates are included in the chapters on the Liver, Biliary System, and Female Genital Tract. In all, there are thirty

PREFACE TO THIRD EDITION

additional plates, making a total of 108 color plates in this new edition. One hundred sixty-eight additional black and white photographs and drawings have been added to maintain the profusion of visual aids which has been an essential feature of this textbook

The editor wishes to take this opportunity to thank all the contributors for their part in the production of this volume. Our gratitude also extends to Ciba Pharmaceutical Products, Inc., for their continued support which has lightened the editor's burden in each edition. Further acknowledgment is given to Miss Olive Hosmer, who not only has made a generous gift to assist this edition but has also recently endowed the Department of Medical Illustration at the Royal Victoria Hospital. This is significant in that the Department was founded in order to illustrate the first edition of the *Textbook of Surgery* and has functioned since that time as the center from which further editions have been produced

The greatest credit must go to Miss Helen MacArthur, our medical illustrator, who has assisted the editor and contributors in every phase of this major academic endeavor. Mrs. Max Slapack has been responsible for the preparation of the manuscript, Mr. Harold Coletta for the photography, and Mrs. F. D. Peart for the checking of many of the references

Since the second edition was published we have lost our valued friend, Miss Marguerite Stadelhofer, of The C. V. Mosby Company, with whom we worked so happily on the first two editions, and we hereby acknowledge the valuable assistance she has given us

Montreal

H. FRID MOSBY

Preface to First Edition

The writing of a textbook to contain the principles of surgery and the amount of factual information required by students at the time of their graduation and during their early period as interns was proposed by me in this center several years ago.

In order better to understand the approach to be followed, student forums were held. These indicated that the students, following the present trends in visual education, wished a profusely illustrated volume. They asked that sections on the Eye, Ear, Nose and Throat, and on Gynecology, apart from that required in the differential diagnosis in abdominal conditions, be omitted, since separate texts were used in these subjects. It was also decided that anatomy, physiology, and pathology should be integrated in the text and that no attempt be made to cover in detail these fields, since they are more adequately dealt with in their respective books.

To ensure the integration of the materials, the sections have been written by members of the Surgical Department of the Royal Victoria Hospital and of the Departments of Neurosurgery, Obstetrics and Gynecology, and Pathology, all associated with McGill University. Great overlap and cooperative discussion were required to achieve this end. Many difficulties presented from the rapid specialization even within special fields and the recent great changes due to chemotherapy, anticoagulants, and improved surgical technique. Balancing the proportions of various sections has been a great problem, and it is hoped that further critical evaluation will assist in this respect.

A book of this type represents the collective support of many individuals, and on behalf of the contributors I would like to acknowledge our debt to Dr. G. Gavin Miller, our Surgeon-in-Chief, who has placed the facilities of his Department at our disposal, to Drs. J. C. Armour, A. L. Wilkie and C. A. MacIntosh, Senior Consultants in General Surgery, to Dr. Newell Philpott, Director of the Montreal Maternity Hospital; to Dr. Wilder Penfield, Director of the Montreal Neurological Institute; to Dr. Emerson Smith, Urologist-in-Chief; to Dr. F. A. H. Wilkinson, Anesthetist-in-Chief, to Drs. Carleton Pearce and E. C. Brooks, Senior Radiologists of the Royal Victoria Hospital, and to Dr. Lyman Duff, Director of the Pathological Institute, all of whom have given valuable advice and criticism in the respective sections. We are also indebted to Dr. J. C. Meakins and Dr. E. H. Mason of the Department of Medicine for assistance on medical problems.

The photographic work is largely the product of Harold Coletta of the Pathological Institute. The references have been checked by Mrs. F. D. Peart of the Medical Library. The manuscript has been meticulously prepared by Mrs. Max Slapack.

Great credit is due to Miss Helen MacArthur of the Department of Medical Illustration of this Hospital, who, during the past three and one-half years, has worked assiduously to prepare the illustrations for this textbook and who has also assisted greatly in its organization and integration.

The editor wishes to thank all his colleagues who have given up their leisure time to write their individual sections and to contribute to the correlation of the materials. It is our hope that this textbook will assist students at all stages in their investigation of the fascinating study of surgery.

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Montreal

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TEXTBOOK
OF
SURGERY

Chapter 1

Evolution of Modern Surgery

E. Walter Workman, MD, Lloyd G. Stevenson, MD,
and H. Fred Moseley, DM.

The practice of surgery, growing more scientific and more successful year by year, has undergone many changes during the last century, following a very long period in which it was essentially a traditional craft, slow to alter. The surgeon, although his skills slowly improved and his social and professional status rose, was in many ways a stock figure, recognizably the same in the 17th century as in antiquity. Some of the characteristics of this relatively unchanging surgeon of old could still be discerned in his modern counterpart of the early 20th century.

THE "OUTSIDE DOCTOR"

The distinction between physician and surgeon was not well marked in the Hippocratic era; nevertheless, the decrying of all manual endeavor as vulgar and low, a form of snobbery peculiarly Greek, was one of the reasons for the subsequent lowly status of the surgeon. In afterages and during many centuries of Western European history, the surgeon was a mere artisan, a common mechanic who had little association with the learned professions, of which medicine was one. Much the inferior in education of the higher-ranking physician, he became, in two senses, the "outside doctor." Doctor he was not, if the word is taken to mean a learned teacher, but doctor he certainly was in the modern popular sense of one who ministers to the sick and injured; and standing as he did for so long outside the regular profession of physic, he was also the

"outside doctor" in having only the outside of the patient—the wounds, the outward herniations, the diseased eyes, the hemorrhoids, the ulcers and the other skin lesions—as his special province.

It is not surprising, in view of the old distinction between surgery and physic, or "internal medicine," that dermatology and syphilology once formed a branch of surgery rather than one of the medical specialties. Jonathan Hutchinson, who lived until 1913 and who is best remembered for his contributions to our knowledge of syphilis, was a surgeon-dermatologist. So was Francis J. Shepherd, Osler's contemporary and friend, who used to teach anatomy at McGill University in the winter and operate at the Montreal General Hospital in the summer. He was Montreal's first dermatologist.

MEDICINE AND SURGERY

The historical relationship between medicine and surgery, a subject of great interest and value, has been investigated by Sir Clifford Allbutt and more recently by Owsei Temkin. According to Temkin, the advances of surgery played a significant part in the rise of modern medical thought. For centuries surgery depended upon an objective anatomic diagnosis; it was not until the 18th century, however, that medicine adopted the surgeon's outlook of a localized pathology in the diagnosis and treatment of disease.

EVOLUTION OF MODERN SURGERY

PRACTICE AND THEORY

Before the 18th century the practicing surgeon was almost invariably an ignorant man of the people and it was only very gradually that the picture changed. True, the famous surgeons of the long robe in 14th century France were in some measure exceptions to the rule, but most surgeons everywhere were ill-educated for another 400 years at least. And men there were, in all parts of Europe, who knew a great deal about surgery in theory who never touched a knife. In the 18th century we find Albrecht von Haller lecturing on surgery at Göttingen University and bringing a vast bibliography of surgical writings, still a useful reference, but never practicing the art. All of his surgical knowledge, great practical anatomist and physiologist though he was, was merely book learning. A practical literature did indeed exist, but it was based on ancient sources and was unendingly repetitious. Only about this time was it beginning to reach out toward science.

As for the practicing surgeon, innocent of Latin and not dependent on books, he had learned the techniques of his craft as an apprentice, like his master before him. For this reason we can seldom be sure that the first account of any operation in the older literature was not preceded by a long unwritten history. Who invented the method of treating fracture of the clavicle with a sandbag between the shoulder blades as described in the Edwin Smith Papyrus of 1700 B.C.? We are reasonably sure that much of the other knowledge preserved in this venerable work is older by centuries than the presumed date of the document itself. Throughout antiquity technical information was largely in the public domain; nobody was much concerned about the "secrets." Operative treatment for hernia, bladder stone, and cataract is of very ancient lineage. Several centuries before the birth of Christ, the treatment of fractures and dislocations had already reached an advanced stage of development among the Hippocratic surgeons of Greece. We know that the plastic surgery of the Renaissance, as set forth by Gasparo Tagliacozzi, was the outgrowth of traditional practice, often carried on by successive members of the same family.

The practicing surgeon languished for centuries outside the pale of the regular profession. In London he belonged, from the 16th century, to the same guild as the barbers and not until nearly the middle of the 18th century did the Surgeons' Company establish itself as a separate entity. This should not be taken to mean that barbers and surgeons were identical. Even in the 16th century London guild, barbers took no part in the examination of surgeons. Nevertheless real barber-surgeons carried on their dual business everywhere in Europe. The surgeons in the armies of Frederick the Great were required to shave the officers, and at about the time when the London surgeons were setting up the Surgeons' Company, Frederick gave permission to Prussian executioners to treat fractures, wounds and ulcers, to the great indignation of his surgeons.

THE BASIS OF MODERN SURGERY

When practical men were ignorant and learned men were pedantic and antiquarian, progress was slow. English medical education escaped from the grip of scholasticism in the London hospitals, where surgery gradually came into its own. Oxford and Cambridge even more firmly rooted in scholastic tradition than the universities of the Continent, were remote from clinical experience. In 18th century Edinburgh, the university and hospital could be happily wedded, partly because they existed side by side in the same urban center.

It was in the universities of Italy, however, rather than in the hospitals, that the groundwork had been laid for modern surgery by the anatomic investigations of Andreas Vesalius and some of his contemporaries of the 16th century. The year 1543 had seen the publication of the first modern anatomic text, the *De Humani Corporis Fabrica* of Vesalius. Professor in the University of Padua. Two centuries later, when pathology began to rival normal human anatomy as the basic discipline of the surgeon, the hospitals, whether linked with universities or not, made the chief contribution. Physiology, on the other hand, coming to the aid of surgery in still more recent times, has grown up in a university setting. University and hospital, with the disciplines peculiar to each, have flourished best when in close association with each other.

The rise of modern anatomy was the first preliminary to the development of modern surgery. It replaced the long-venerated teachings of Galen, a Greek doctor who worked in Rome in the 2nd century of our era. Galen was an expert anatomist but unfortunately had little opportunity to work with human materials and had to be content with apes, oxen, and other animals. His skill in experimental physiology proved harder to match.

SCIENCE AND PRACTICE

While surgery may be said to have waited on the growth of the sciences which nourish it, surgeons did not, and could not wait, their work continued, and not without improvement. Even in the early Middle Ages, the "Arabians"—all those who wrote in Arabic—were responsible not only for transmitting the knowledge of antiquity but for adding to it, especially in pharmacology and ophthalmology. By the 13th century the names of Theodoric and Henri de Mondeville remind us that the non-suppurative treatment of wounds was attempted very early. The concept of "healing by first intention" is, however, a Galenic concept, a fact to remember when we read that Galen believed pus to be necessary for the healing process. John of Arderne, in the 14th century, perfected an early technique for operating on *fistula-in-ano*. In surgery, as in other crafts, our medieval forbears did not lack in ingenuity, and some part, at least, of the glory of the Renaissance is literary artefact.

Renaissance surgery, fortified by the "new anatomy," nevertheless continued to progress on the old practical lines. Good things lost from sight had to be brought back. Ambrose Paré re-introduced and popularized (he did not invent) the use of the ligature to control hemorrhage. Like other great surgeons, he made constant use of the experience of daily practice. His adventurous, controlled experiment in treating some patients with gunshot wounds with boiling oil, at that time the standard procedure, and others with a bland ointment taught him that such wounds are not "poisoned" by gunpowder and not helped by cauterization. Paré is the supreme example, among many others, of the innovating military

surgeon. His contemporary, Pierre Franco, was probably the first to attempt the surgical treatment of strangulated hernia.

The 17th century carried forward the same means of advance. Knowledge of anatomy was of benefit in the treatment of fractures, in the emergencies of hemorrhage, and in various other ways, but for more than two centuries longer, surgical procedures were still to be very much limited by the necessity for speed, so that fancy disarticulations and so forth were not of much practical importance, though the state of anatomy permitted such undertakings. Seventeenth and many 18th century surgical texts, despite all improvements, still manifestly belong to the era of the old surgery. Amputation remained too often the first and not the last of the surgeon's resources.

Still, observation and ingenuity counted for much. In 1752 Jacques Daviel substituted cataract extraction for the age-old operation of couching, or downward dislocation of the cataractous lens on the end of a needle. Like many innovations this was the result of a happy accident in practice, seized upon and followed up by a keen practitioner. At the same time surgery was drawing more and more upon experimental science, John Hunter, a great 18th-century Scot practicing surgery in London, seems to have learned from purely scientific experiments on deer how rapidly collateral circulation, already known, may take over the function of the main artery of a part, and this knowledge he applied in devising a new and successful operation for popliteal aneurysm. Comparative anatomy he employed as another approach to physiology. He was likewise a good pathologist. In the 17th century, and increasingly in the 18th century, surgery began to be more than an empirical craft. Simultaneously the great hospital schools were developing and the surgeon was rising in social status and professional esprit.

The great surgeons of the late 18th and early 19th centuries, like Astley Cooper and James Syme, were primarily concerned with applied anatomy. Astley Cooper contributed not only by his great works on fractures and dislocations and on diseases of the breast, but also by his contributions to vascular surgery. He was the first to ligate the aorta successfully. James Syme

will be remembered for his resections of the capula, the forequarter, the lower jaw, and the major joints, as well as for the amputation that bears his name Jacques Lisfranc was in many ways the French counterpart of Syme. But pathology, too, played its part. Dupuytren, for example, was a first-class surgical pathologist. Karl Ferdinand von Gräfe in Germany and Philibert Roux in France extended the domain of plastic surgery. Orthopedics and ophthalmology progressed. Gynecologic surgery made a fresh beginning in America with Ephraim McDowell's ovariectomy and the pair of vesicovaginal fistula by Marion Sims. But surgeons of every sort pulled up helplessly in the face of the two problems of greatest significance to all—pain and sepsis.

ANESTHESIA

The introduction and development of anesthesia preceded antiseptics and asepsis. By 1850 anesthesia was in general use. Although ether had been known since the 16th century, it was first used in surgical practice on March 30, 1842, by Crawford Williamson Long, who did not publish his results at the time of his case. However, ether did not become popular until October 16, 1846, when W. T. G. Morton, a dentist, successfully demonstrated the use of ether on a patient of John Collins Warren at the Massachusetts General Hospital. In 1847 *chloroform* (discovered in 1831) was first used by Sir James Y. Simpson on obstetric cases. It soon became the usual anesthetic in Great Britain. Chloroform has been utilized more than ether in Europe, while the reverse is true in America. *Nitrous oxide* was isolated in 1776 by Joseph Priestley. Humphry Davy noted the effect of the gas when inhaled, he suggested its use during surgical operations but never attempted to employ it. Horace Wells, a dentist, administered nitrous oxide while extracting teeth in 1841. One of Wells' patients died during nitrous oxide administration after which he gave up his researches and the practice of dentistry. Nitrous oxide became a safe anesthetic when Edmund W. Andrews in 1868 introduced a mixture of oxygen with the gas. *Ethylene*, discovered in the 17th century, did not come into use until introduced by Luck-

hardt and Bailey in 1923. Although this agent approached the ideal anesthetic since it was fast acting and caused minimal disturbance of carbohydrate metabolism, ethylene did not continue to be popular because it was highly explosive and did not induce adequate relaxation. *Cyclopropane* discovered by August von Freund in 1882, studied as an anesthetic by G. H. W. Lucas and V. E. Henderson in the 1920's, was first administered clinically in 1930 by Ralph M. Waters and his associates. This agent proved to be a great advance because in addition to the advantages of ethylene it produces a greater degree of muscular relaxation and permits the use of a high percentage of oxygen. It is, however, explosive and has a tendency to produce ventricular fibrillation. While the development of intravenous anesthesia commenced with the synthesis of the barbiturates in 1904, the introduction of *hexobarbital* (Evinpan) in 1932 and *thiopental sodium* (Pentothal sodium) two years later established this form of anesthesia. Intravenous thiopental sodium has now come into general use for minor surgical procedures and in combination with other types of anesthesia. The use of local anesthesia came after antiseptics and asepsis. In 1883 *cocaine* was used to abolish pain in eye operations. Halsted was the pioneer in local cocaine infiltration anesthesia, and in 1885 his work was well established. With the discovery of *novocaine* by Einhorn and its clinical use by Braun in 1905, local anesthesia came into general use. Although August Bier developed and popularized spinal anesthesia in 1893, the first spinal anesthesia in man was performed in 1885 by James Leonard Corning. There have been further developments with the introduction of epidural anesthesia (1920), continuous spinal anesthesia (1940), and, more recently, catheter spinal anesthesia.

Muscular relaxants were brought into anesthesia in 1942 with the introduction of *curare* by H. R. Griffith and G. F. Johnson. This drug was used for centuries by the Orinoco Indians of South America as their arrow poison.

Recent physiologic studies of the anesthetics have led to their more selective application and to their use in combination to obtain the optimum result.



Fig 1—Lord Lister (1827-1912)

THE LISTERIAN ERA

In 1864 Pasteur proved that fermentation depended upon living organisms and thus began the conquest of sepsis and the era of modern surgery. In 1867 Joseph Lister published his paper on "The Antiseptic Principle in the Practice of Surgery." He laid the foundation for *asepsis* and *antiseptis*. In 1871 he introduced the carbolic spray which was in use until 1887. The purpose of the spray was to kill germs in the air. In 1880 he introduced the absorbable catgut ligature. The science of bacteriology was advanced by Robert Koch when in 1878 he showed that each type of wound infection was due to a specific organism. This placed surgery upon a firm scientific basis. The antiseptis of Lister was replaced by aseptic methods when Bergmann in 1886 introduced sterilization by boiling and Halsted in 1891 introduced the use of rubber gloves for operations. Asepsis made possible the extraordinary development of abdominal surgery at the end of the 19th century as exemplified by Billroth.

X-RAYS

Direct examination of the body cavities by endoscopic means was in use before the advent of x-rays. Methods of intubation of the larynx

were known since the time of Hippocrates and preceded the direct examination of it. At about the middle of the 19th century the laryngoscope became a popular instrument and laryngoscopy was established as a procedure for diagnosis and treatment. The introduction of the cystoscope in 1879 initiated rapid progress in endoscopic techniques for other parts of the body.

Following anesthesia, antiseptis, and asepsis came the discovery of x-rays in 1895 by Wilhelm Konrad Röntgen. After this event there was rapid development and 10 years later the specialty of radiology was established. X-rays were first used to visualize foreign bodies and, later, fractures. Advances in methods of diagnostic radiology came with the development of procedures using radiopaque media. The first of these was utilized by Walter B. Cannon when he gave a radiopaque meal of bismuth subnitrate to visualize the gastrointestinal tract. Later barium sulfate was generally adopted for this purpose. In 1924 Graham and Cole reported their development of cholecystography, using tetrabromphenolphthalein intravenously. This was an event of far-reaching importance in the study of biliary tract and gall bladder disease. Since that time compounds for oral use have been perfected. Recently a substance, Cholografin, has been developed for intravenous use that is especially valuable for studies of the biliary tract after cholecystectomy. The historic considerations of the radiologic visualization of the urinary tract are described with the development of urology. Other advances in the use of contrast techniques have been the introduction of ventriculography in 1918 by Walter E. Dandy and the development of cerebral angiography in 1931 by Egas Moniz and Almeida Lima. More active interest in vascular visualization came in 1937 when Robb and Steinberg reported the injection of large quantities of radiopaque material for visualization of the chambers of the heart and the pulmonary circulation. Aortography and visualization of the peripheral vascular system have now come into general use. Thus by the use of contrast media the scope of radiologic diagnosis has been extended and offers possibilities for continued development.

The field of radiotherapy began when the x-rays were discovered in 1895 and when Henri Becquerel in 1896 found that uranium salts

3 radiations. Early in the history of radiology the x-rays were used therapeutically for skin malignancies, skin diseases, leukemia, and lymphadenoma. The invention of the mechanical rectifier by Clyde Snook in 1905 initiated the production of higher voltage apparatus. A great advance was made when the hot cathode filament tube was developed by W. D. Coolidge in 1913. This Coolidge tube, although modified, is still standard in principle. A super-voltage x-ray generator, having a capacity of 1,000,000 volts, was built in 1935 by R. A. Röntgen and his associates. An evaluation of this form of therapy indicated that it is a refinement rather than a great improvement. In 1911 the betatron developed by Kerst generated 20,000,000 electron volts. This apparatus has been used successfully in the treatment of cancer.

Radium was discovered in 1898 by Pierre and Marie Curie and is still used today, although more in Europe than in North America. Radium in conjunction with x-rays is used in the treatment of cancer of the cervix.

The era of artificial radioactive materials commenced when Ernest Orlando Lawrence constructed the cyclotron in 1932. In 1942 Fermi was the first to build a nuclear reactor for the production of isotopes. Isotopes have been used for tracer studies and the treatment of cancer. Cobalt⁶⁰ is now used extensively in the form of cobalt bomb as a means of teletherapy and in needles and tubes for interstitial and intracavity therapy.

While radiotherapy alone has been curative and palliative in the treatment of cancers, it has often been used in conjunction with surgery. The therapeutic effect of radiation depends, in addition to other factors, upon the radiosensitivity of the lesion.

Thus by the end of the 19th century the three great pillars were placed and modern surgery was established.

SURGERY IN THE TWENTIETH CENTURY

The 20th century may be divided into two periods, 1900-1936 and 1936 until the present. The first period was influenced by World War I and the discovery of insulin. The second period was influenced by World War II and



Fig. 2 - Sir Frederick Banting (1891-1911)

the advent of the *sulfonamides* and the *antibiotics*.

As a result of World War I, antiseptics, germicidal but less destructive to tissues, were developed. Débridement of wounds and prophylactic inoculation against tetanus were introduced. In 1921 Banting, Best, Macleod, and Collip discovered insulin. This discovery had a profound influence on the surgical treatment of diabetes. Diabetic gangrene was more easily controlled and operations on the diabetic could be performed with the same degree of safety as on the nondiabetic.

The Age of Chemotherapy and the Antibiotics

The age of chemotherapy had its inception in 1906 when Paul Ehrlich discovered Salvarsan, but it was not until 1936 that the sulfonamides came into use for the treatment of infections. With this the present era was ushered in. Gelmo in 1908 synthesized para-aminobenzene-sulfonamide (sulfanilamide) which was used in the dye industry. It was not until 1933 that Foerster in Germany reported upon the first clinical use of a sulfonamide compound known as Prontosil. Domag

in 1935 reported the first experimental work on Prontosil in which he proved that it prevented fatal hemolytic streptococcal infections in mice. Simultaneously there appeared clinical confirmation of Domagk's work in that the drug was effective in septic hemolytic streptococcal angina, erysipelas, puerperal sepsis, and other infections. In 1936 Colebrook and Kenny in England reported that Prontosil and a variant Prontosil S were of definite value in the treatment of puerperal sepsis. Interest in these compounds in the United States did not appear until 1936 when investigations were begun by Mellon and his associates, Rosenthal and Bauer, and Long and Bliss. Long and Bliss formed the hypothesis that sulfanilamide acted directly upon the invading microorganism and that this action was primarily one of bacteriostasis. They were convinced that Prontosil, Prontosil S, and sulfanilamide were of definite therapeutic value in streptococcal infections.

During the years of World War II there followed sulfapyridine in 1939, sulfathiazole in 1940, sulfadiazine in 1941, and sulfamerazine in 1943. Then followed Sulfasuxidine and Sulfathalidine. New sulfonamide compounds are continually being developed.



Fig. 3—Sir Alexander Fleming (1881-1955)

In 1929 Alexander Fleming discovered *penicillin* and suggested that it might be an effective bacteriostatic agent to penicillin-sensitive organisms. Fleming's discovery remained unnoticed until 1940 when interest in the clinical use of penicillin was aroused following the investigations of Chain, Florey, and their associates at Oxford University. Since that time, the other antibiotics, including *streptomycin*, *Aureomycin*, *chloramphenicol*, *erythromycin*, and *tetracycline* were discovered. New antibiotics are constantly being produced. From 1940 there has been greater progress in the control of infections than has taken place in all previous medical history. The use of the sulfonamides and the antibiotics has made practical many surgical procedures that before were not possible with any degree of success.

The Control of Blood Coagulation

The anticoagulants *heparin* and *Dicumarol* have been two extremely effective agents of therapy for venous thromboembolic disease. As late as 1934 surgery was helpless against thrombosis. Heparin was discovered by McLean in 1916. In 1933 Best and his co-workers solved the problem of preparing large quantities of heparin. Between 1935 and 1942, Gordon Murray in Canada and Crafoord in Sweden simultaneously and independently showed that thrombosis can be prevented by heparin post-operatively. Dicumarol was described, isolated, and synthesized by Link in 1940 and came into clinical use in 1941. It is interesting that the discovery of this agent was due to Schofield, who in 1922, in the *Canadian Veterinary Record*, described a disease of cattle in which there was a tendency to bleed after eating spoiled sweet clover. The use of the anticoagulants after major surgical procedures for prophylaxis and treatment has diminished the incidence and mortality of pulmonary thrombosis and embolism. With these two agents greater safety in vascular and cardiac surgery became possible. A method of increasing the coagulability of the blood and the prevention of post-operative hemorrhages in obstructive jaundice became available with the advent of *vitamin K* ("Koagulations-Vitamin"). This was discovered by Dam in 1934 and was used clinically in 1938.

Blood Transfusions

Although blood transfusion had its inception in the 17th century, further investigation of its possibilities awaited the turn of the present century. Blood transfusion was successfully performed on dogs by Richard Lower in England (1666). Because of the lack of knowledge of iso-agglutinins and the many consequent failures in human beings, it was forbidden and abandoned for approximately 150 years. With the introduction of blood grouping in 1900 by Landsteiner and independently by Shattock the modern era of blood transfusion began. World War I saw the introduction of the citrate method which allowed blood to be stored for a few days before use. After the successful use of cadaver blood in 1927 by W. N. Sharnov, Kostinkov, and S. S. Yudin of Russia, there followed the development of banks of preserved blood and plasma. Refined methods of blood grouping, matching, and storing are now in general use and better methods of combating shock have thus been developed.

SURGICAL SPECIALTIES

In the first half of the 20th century there have been such tremendous advances in surgical knowledge that increased specialization in the various branches was necessitated. Each of these surgical specialties in turn has become highly developed and technical. A brief consideration of their evolution and status may contribute to a picture of present-day surgery.

Abdominal Surgery

Abdominal surgery has developed rapidly since the beginning of this century. At that time the aseptic method of opening the peritoneal cavity was introduced and appendectomy had become a frequent operation. Gastroenterostomy, first performed by Wölfler in 1881, reached the height of its popularity in the late 1920's for the surgical treatment of peptic ulcer. This was later replaced by subtotal gastrectomy as the operation of choice. The first successful partial gastrectomy was performed for cancer of the pyloric region by Theodor Billroth on January 29, 1881. The Billroth I operation included an anastomosis of the cut end of the stomach to the cut end of the duodenum. In 1884 he introduced the Billroth II

operation in which he anastomosed the posterior surface of the stomach to the cut end of jejunum. This operation, modified by von Eiselsberg, Polya, Hofmeister, Finsterer, Moynihan, Balfour, and others, is the surgical procedure now most frequently used. In 1943 Dragstedt and Owens, advancing Pieri and Tanferna's technique, began to perform vagotomy for duodenal ulcer. Later they combined this operation with gastroenterostomy which proved to be of value in certain cases. Vagotomy alone has been used effectively for gastrojejunal ulcer after gastric resection and for early cases of ulcerative colitis. Progress has been made in the differentiation of gastric ulcer and gastric carcinoma. In addition to x-ray procedures there has been the development of gastroscopy by Schindler and others, exfoliative cytologic techniques, and electrogastrography.

In 1826 Antoine Lembert described his method of intestinal suture and laid the foundation of modern intestinal surgery. The anastomosis button devised by J. B. Murphy in 1892 was used successfully in gastroenterostomies but did not reduce the high mortality rate of primary colonic resection. As a result, a safer procedure, the exteriorization resection of bowel, was developed by Oscar Block (1892) and Paul (1895) and popularized by Mikulicz in 1903. The aseptic anastomosis introduced by Parker and Kerr in 1908 was only later accepted. The sulfa drugs and then antibiotic agents have made the operation of resection and primary anastomosis the procedure of choice. The control of infection has also allowed extensive one-stage large bowel surgery.

Marked advances have been made in the treatment of intestinal obstruction through the studies of Dragstedt (1917), Haden and Orr (1923), Gamble and Ross (1925), and others. In 1933 Wangenstein popularized intestinal decompression by suction; this has been modified by Miller, Abbott, and others and has proved epoch-making in abdominal surgery. Following investigations of the physiology and chemistry of intestinal obstruction, the prime importance of adequate fluid and electrolytic balance has been recognized. In 1908 Ernest Miles introduced the abdominoperineal resection for carcinoma of the rectum which, in principle, is still most widely used, despite the development of many other operations.

While resections of portions of the liver were done at the end of the last century, there were no further reports until 1921 when Peck performed a left lobectomy for an angioma. Since then this operation has been successfully used for primary neoplasms. It was not until 1919 that Wangensteen successfully removed the entire right lobe of the liver, which operation has since been repeated by others.

In biliary tract surgery since about 1921 cholecystectomy has replaced cholecystostomy. Commencing in 1924 more exact methods of diagnosis have been developed and are described elsewhere. Liver damage has been extensively studied and methods of therapy have been discovered. Vitamin K was introduced in preoperative preparation of the jaundiced patient. New techniques for repair of traumatic rupture of the common bile duct have been developed by Walters, Cole, Lahey, and Cattell. In surgery of the pancreas, the first pancreaticoduodenectomy was reported by Whipple, Parsons, and Mullins in 1935. Many procedures have been devised, including total pancreatectomy, but despite the reports of longer survivals the treatment of carcinoma of the pancreas is still unsatisfactory.

In splenic surgery, while trauma is the most common indication for splenectomy, hematologic studies have added many other indications. The treatment of portal hypertension was first devised in 1943 by Whipple, who used a shunt between a major portal tributary and a systemic vein. This form of surgical treatment has been advanced and modified by many others.

Gynecology

Operative gynecology was founded by Abraham McDowell, who performed the first ovariotomy in 1809. This stimulated other surgeons to perfect the technique of this operation, and in the 1850's, mainly through the efforts of

John A. Sampson for vesicovaginal fistula removed that condition from the list of incurable maladies. This achievement was made possible by using the Sims' position, by the invention of the duck-billed speculum (Sims' speculum) and

silver wire sutures, and by utilizing the retention urethral catheter.

During this century the field of gynecology has grown enormously. To the specialty has been added radiation therapy, endocrinology, and psychosomatic medicine. The type of operation has changed markedly. Whereas suspension of the uterus was once a frequent operation it is now advised only when the indications are clear cut. Fewer radical operations are now carried out for fibroid tumors. Vaginal plastic operations are gradually being replaced by vaginal hysterectomy and repair. Total hysterectomy has replaced supravaginal hysterectomy. While the repair of vesicovaginal fistula has been solved, the etiologic factors have changed—fewer fistulas follow obstetric difficulties and more are due to injury of the bladder during uterine operations and as a result of radiation. The management of sterility, together with the surgical means of studying this problem, is a new development. The importance of endocrinology to the gynecologist is great; e.g., the identification of endocrine tumors has changed the classification of ovarian tumors.

Endometriosis was first described by John A. Sampson in 1921 and is now well understood. One of the great advances in treatment is that the chocolate cyst in the ovary can be "shelled out" and the ovary saved. Radical surgery and radiation therapy in the treatment of cancer of the female pelvis have yielded improved results but the problem still remains unsolved.

Urology

The introduction of the cystoscope in 1877-1878 by Max Nitze inaugurated the era of cystoscopy, and urology as a specialty evolved. Since that time the Brown-Buerger cystoscope, presented in 1909, has with modifications proved to be the most adaptable and popular instrument.

Retrograde pyelography was initiated in 1906 by Voelcker and von Lichtenberg when they used colloidal silver to visualize the bladder and unexpectedly also saw the ureter and renal pelvis. Using sodium iodide, Rowntree in 1923 introduced intravenous pyelography. This method has been perfected and is now a routine procedure. Other diagnostic procedures include perirenal insufflation of oxygen, introduced in

1921 for the diagnosis of adrenal tumors; this has been replaced by presacral insufflation. Renal aortography was utilized in 1929 for the diagnosis of renal cysts and tumors. There have been great changes in therapy. The management of renal tuberculosis has been greatly improved by the use of antituberculous drugs in association with nephrectomy.

Among the advancements in bladder surgery has been the construction of substitute bladders. In 1950 Gilchrist and his associates devised an operation utilizing the cecum and ascending colon. Bricker introduced another method in which an isolated loop of terminal ileum is used as an ileal bladder. These methods offer promising possibilities.

The outstanding contributor to prostatic surgery since 1900 has been Hugh Young, who perfected the "punch" operation and developed the perineal operation and radical procedures for prostatic carcinoma. In addition to the development of the perineal, suprapubic, and retropubic methods of prostatectomy, there has been great interest in transurethral resection, which for practical purposes dates from 1909 when Young introduced the urethrosopic prostatic excisor. There have been many modifications in the 1920's, and with the development of the Stern-McCarthy visual electrotome, great interest was aroused and many improvements followed. This is now a well-established form of treatment for certain prostatic cases. A new approach to the treatment of prostatic cancer was introduced in 1939 by Huggins' work on the endocrine features; this resulted in the use of bilateral orchiectomy for inoperable prostatic carcinoma. Recently Huggins has been studying the relationship of the pituitary and adrenal to this condition.

Thoracic Surgery

The foundation for modern thoracic surgery was laid, with attempts to control pneumothorax in open operations of the chest, by Ferdinand Sauerbruch with his negative pressure cabinet and Willy Meyer with a positive pressure apparatus. Sauerbruch's cabinet, introduced in 1903, weighed 10,000 pounds and necessitated that the patient and the operators be inside the enclosure with their heads outside. The problem was solved in 1909, when

S. J. Meltzer and J. Auer introduced positive pressure endotracheal anesthesia. With the outbreak of World War I the era of modern thoracic surgery commenced. During the years 1910-1920 thoracic operations were mostly procedures for the drainage of empyema and lung abscess. There was, however, some interest in thoracoplasty for pulmonary tuberculosis. Advances between 1920 and 1940 followed improvements in anesthetic techniques, increased knowledge of respiratory physiology, control of shock, blood transfusions, maintenance of fluid balance, and chemotherapy. Improved methods for the treatment of suppurative diseases of the lung were developed.

In 1923 Fvarts Graham described his operation "pneumonectomy with cautery" which was actually a resection of a portion of lung tissue. This method persisted for the excision of suppurative lung tissue until 1929 when Harold Brunn successfully devised an improved technique of lobectomy for bronchiectasis. The mortality and morbidity have since been greatly lowered with the use of chemotherapy and the antibiotics. Before 1933 there were only six recorded cases of lobectomy for carcinoma of the lung with one-year survival. The first successful pneumonectomy was performed by Nissen in 1931 on a patient with bronchiectasis. Fvarts Graham performed the first successful pneumonectomy for lung cancer on February 27, 1933, and the patient is alive and well to day. At present the operation carries a relatively low mortality rate.

Surgery of pulmonary tuberculosis advanced during the 1920's, more rapidly than other thoracic surgery. The collapse therapy methods of Sauerbruch of Germany were advanced and practiced on this continent by Edward Archibald, John Alexander, and others. With the discovery of antituberculosis drugs, early segmental resection and lobectomy are now being advised.

The field of thoracic surgery is continually expanding and has progressed from a restricted specialty to one which includes many safe and effective procedures.

Cardiovascular Surgery

Cardiac surgery until recently was very limited. In 1913 Rehn and Sauerbruch operated

for constrictive pericarditis. There were early attempts to explore the cardiac valves. Doyen in 1913 operated for pulmonary stenosis and Tuffier in 1914 was temporarily successful in digitally dilating an aortic stenosis. From 1923-1928 Cutler, Levine, and Beck operated upon patients with mitral stenosis, using a tenotome and modifications thereof. Also in 1925 Souttar performed a successful digital valvulotomy for

lowed by the work of Bailey, Harken, and others. New techniques are rapidly developing in cardiac surgery, one of the most recent of which is the extracorporeal pump. This apparatus, pioneered by John Gibbon, Jr., and others, is continually undergoing improvements and holds much promise for open cardiac surgery. Successful cases are being reported with greater frequency.

The surgical treatment for coronary occlusion was begun in 1932 by Claude Beck, who has been perfecting various methods for myocardial revascularization. O'Shaughnessy's use of omental grafts soon followed. Recent developments include Beck's shunt from the aorta to the coronary sinus, and Vineberg's internal mammary artery implant.

The groundwork for vascular surgery was laid by Alexis Carrel's work from 1900-1910. In 1905 he developed a method of blood vessel anastomosis, and in 1908 he reported the successful transplantation of a 30-day preserved segment of a dog's aorta into the aorta of another dog. During this time Rudolph Matas and William Halsted were pioneering in the surgical treatment of aneurysms.

In 1938 Gross reported the first successful ligation for patent ductus arteriosus. Blalock in 1945 successfully operated upon a patient with tetralogy of Fallot by anastomosing a branch of the aorta to the pulmonary artery, also in 1945 Crafoord and Gross independently reported a successful resection for coarctation of the aorta. In 1946 Potts described the side-to-side anastomosis of the aorta to the pulmonary artery for the tetralogy of Fallot.

There have been most rapid advances in blood vessel surgery. Vessel grafts are now being used after resection of vascular lesions.

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The surgical treatment of both the infective and traumatic disorders of the hand was late in its development. Kanavel placed the therapy of hand infections on a sound basis, and during and following World War II, Bunnell and his co-workers standardized practically every aspect of the primary and secondary procedures for the traumatized hand.

The investigations of tendon anatomy and physiology by Biesalski and Mayer, Bunnell, and others have led to an atraumatic technique. This has contributed largely to the success of transplant procedures for the correction of paralytic deformities and the repair of traumatized tendons of the hand and other parts of the body.

Plastic Surgery

Although some aspects of plastic surgery have been practiced for many centuries, it developed into a separate specialty during World War I. Subsequently its leading exponents in

various countries created centers for system training.

During World War II great progress made in the more physiologic treatment of burn shock. The advent of antibiotic agents assisted in the control of the associated infection. Treatment by exposure superseded the occlusive pressure dressing except for certain regions such as the hand. The development of the dermatome by Padgett enabled the surgeon readily to resurface third-degree burns.

The observation that homografts survive permanently in patients suffering from congenital agammaglobulinemia has spurred research into methods of obtaining survival of homologous grafts of tissues and organs.

General adoption by plastic surgeons of the LeMesurier procedure for cleft lip repair greatly improved the esthetic results obtained. Recently devised operations for repair of cleft palate have resulted in better function of speech.



Fig. 4—Present-day surgery under the television camera. (Courtesy Smith, Kline & French Laboratories, Montreal, Canada)

gery, and better hemostasis. Consequently more conditions are now being treated.

Recently psychosurgery, the surgical intervention for the relief of psychoses, previously considered hopeless, has been developed. These operations are declining in frequency as a result, in part, of new drugs.

Traumatic and Orthopedic Surgery

Like surgery in general, orthopedics was profoundly influenced by two world wars. In both conflicts there were numerous injuries to the extremities which led to new and better methods of treatment. It has been stated that orthopedic surgery as we know it today had its birth in World War I and became of age in World War II.

At the end of the 19th century, orthopedists were concerned mostly with deformities of the spine, tuberculosis of bone and joint, clubfoot, and congenital dislocation of the hip. While there was a trend toward operative surgery in addition to mechanical therapy, there were many orthopedists adhering to the principle of braces and mechanotherapy alone.

By the beginning of the present century operative surgery was gaining momentum. Among its pioneers were Arbuthnot Lane, Albin Lambotte, Fred Albee, and Russell Hibbs. In the last decade of the 19th century and early part of the present century, Lane and Lambotte developed the treatment of fractures by open operative reduction and mechanical fixation of the fragments. The former introduced the metal-touch technique for the insertion of the metallic plates and screws. This method fell into disuse until the introduction of the inert metals following the researches of Venable and Stuck in 1937, which obviated the associated foreign body reaction of bone to the corroding metal previously employed. This, together with the use of antibiotics and improved standards of surgical technique, has firmly established the open operative treatment of fractures.

The application of this method is well seen in the treatment of the so-called "unsolved" fracture, i.e., of the neck of the femur, which was revolutionized in 1931 when Smith-Petersen introduced the three-flanged nail for its internal fixation. A further development for fractures of the shafts of long bones such as

the femur was the use of intramedullary splinting, employing a nail developed by Kuntscher in 1938.

The independent development of bone graft surgery by Albee and Hibbs has also led to great progress, chiefly in relation to spinal fusion and to nonunion of fractures. In recent years bone banks have been established for the preservation of refrigerated homologous bone.

Orthopedic surgery was firmly established as a specialty in Great Britain, largely due to the influence of Robert Jones, who disseminated the ideas of Hugh Owen Thomas. In addition to his many other contributions and outstanding work on joint injuries, he was responsible, after World War I, for the inclusion of the treatment of fractures as a part of British orthopedic practice.

The outstanding contribution to the functional treatment of fractures was made by Bohler. He stressed the concomitant treatment of the soft tissues. His ideas were disseminated in the English-speaking world by the translation of his textbook by Hey Groves and later by the inclusion of his ideas in the well-known text of Watson-Jones.

In recent years advances have been made in many directions. Arthroplastic procedures, especially in relation to the hip joint, have undergone many changes. In 1939 Smith-Petersen developed the Vitallium mold arthroplasty. The acrylic replacement of the femoral head, popularized by Jean and Robert Judet, has rapidly been replaced by prostheses of inert metal with an intramedullary shaft. Evaluation of such prostheses still remains to be determined.

Our understanding of spinal disorders with referred segmental pain has been enhanced by the practical application by Mixter and Barr of Schmorl's studies of the intervertebral disc. Most cases of lumbosacral pain and sciatica are now known to be due to disc degeneration and prolapse.

Our knowledge of shoulder lesions rests on the fundamental studies of Codman and Bankart, who established the underlying bases for the clinical disorders of the rotator cuff and for the recurrent dislocation of this joint. To Codman must also be given the credit for establishing the American Registry of Bone Sarcoma on which is based the modern concepts and treatment of these malignant neoplasms.

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Requirements for Higher Surgical Qualifications

- Booklet of Information, American Board of Surgery, Inc, 225 S 15th St, Philadelphia 2, Pa
- Regulations Relating to Examinations for the Diploma of Fellow, Royal College of Surgeons of England, Lincoln's Inn Fields, London, W C 2, England.
- Regulations and Requirements of Graduate Training Related to the Examinations of the Royal College of Physicians and Surgeons of Canada; Surgery and Surgical Specialties (Office of the Honorary Secretary, 150 Metcalfe St, Ottawa 4, Canada)

Hand surgery has developed as a specialty which combines the skills of the general, orthopedic, neuro-, and plastic surgeon. The use of skin grafts, pedicle flaps, and bone cement, combined with cancellous grafts and nerve suture, has salvaged many mutilated hands.

Plastic surgery, in conjunction with other specialties, has become interested in the problems of geriatric patients and has helped in solving the difficulties arising from neoplastic disease, mainly of the head and neck.

THE TRAINING OF A SURGEON

While surgery has developed by a process of evolution from an art to a science, the training of the surgeon has also changed. The surgeon should have a thorough grounding in the Humanities before he seeks a knowledge of the basic sciences, physiology, anatomy, biochemistry, bacteriology, and before he attempts to coordinate pathology, roentgenology, and internal medicine. In an address before the Royal College of Physicians and Surgeons of Canada, Van Wyck aptly emphasized the importance of the Humanities as part of a medical education. "Thus, the humanities promise much more than a mere living. They promise life, wisdom, a spiritual concept of existence, freedom of intellect and will, and the fruitful development of men's ineradicable religious instinct. And of these interests and studies of man, let us choose the three greatest because they include all the others—literature (especially poetry), history, and the arts." Sir Henric Ogilvie stated, "There are at least four stages in the forming of a surgeon: he must be found, he must be qualified, he must be trained, and he must be given opportunities." Of the four, the training stage requires further consideration.

The doctor who plans a career in surgery must have a definite schedule of postgraduate training. In addition the course should be planned to meet the requisites of the various certification boards. A suggested minimum course should include six months in pathology, one year of internship in an approved hospital, and two and one-half years in general surgery in an approved hospital. Those who plan to undergo a more thorough training and receive a Fellowship in one of the colleges should in

addition, include a year in one of the basic sciences or experimental surgery, a year at various clinics, and a residency in surgery.

Finally improved methods of visual surgical education are being developed by means of the motion picture and television. The surgical societies and the medical schools are gradually building up libraries of valuable teaching films. In some centers operations are being televised. The advantages of this medium are obvious.

In addition to a scientific training, a surgeon must possess and develop fine qualities of mind and body. Hutchison has beautifully described the attributes of a surgeon in his address to the students of the London Hospital Medical College, entitled "Seven Gifts."*

"Good health that sort of wiry constitution which is able to resist fatigue and infection"

"Luckiness pure luck is one of the chief factors making for happiness and success. Some men owe all or nearly all their success to luck, but if you have it not, remember that hard work and patience can make up to a great extent for the want of it."

"Brains but not too many. It is unnecessary—perhaps dangerous—in medicine to be too clever. But if I had not many brains to bestow I should make up for it by an extra gift of diligence."

"Equanimity there is no quality of mind more essential to you as doctors, for you will often have to face sudden and disconcerting emergencies and a fair share of it will also do much to preserve you from the corroding effect of those worries which are unescapable in practice."

"A sense of justice in the first place to your patient—Justice also to your professional brethren—and lastly a sense of what is just to yourself."

"A sense of beauty. Disease is ugly. You will need a sense of beauty as a compensation and a way of escape, as a sanctifying and steadying influence."

"My last and best gift would be a sense of humor. It will help you to bear with the vagaries of your patients and still more with those of their relations and to derive amusement instead of annoyance from the eccentricities of your colleagues."

Armed especially with the last three, a sense of justice, of beauty and of humor, you will be able to face with equanimity all the buffets and disappointments, all the weariness and ugliness which your lives as doctors may have in store for you.

*Hutchison, R. Seven Gifts, *Lancet* 2. 61-6. 1938

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Chapter 2

Inflammation and Repair

Gardner C McMillan, MD

The reactions of the body to injury may be localized or generalized. The localized reaction to injury is called *inflammation*. The generalized reaction has not been given a special name. It arises when the local reaction is sufficiently great or when the injurious stimulus does not remain sufficiently localized. Both the local and general reactions are complex and may find variable expression. Inflammation is divided into simple and granulomatous types. The generalized reaction is not so clearly categorized but is designated by such terms as shocked, toxic, stressed, adaptive, febrile, allergic, etc.

Among the reactions of the body to injury is that of *repair*. It is a local reaction the effect of which is to restore injured tissue and its function. It is frequently maintained that repair is an end stage of inflammation, but there is little justification for this view. Whereas inflammation results in the neutralization or removal of injurious agents and effects the disposal of dead tissue, repair has no direct action on injurious agents; indeed it is inhibited by them, and it fills the defects left by injury and inflammation. One of the cardinal signs of inflammation is loss of function. The subsidence of inflammation is followed by repair, but special forms of repair such as compensatory hypertrophy can occur in the absence of inflammation at the site of hypertrophy. It would seem reasonable to recognize that, while inflammation and repair frequently seem intimately related, they are separate processes of fundi-

mentally different nature arising in response to fundamentally different stimuli.

It should not surprise the reader to find that this concept is opposed to that expressed in the previous editions of this textbook. Professor T. R. Waugh wrote: "Moreover, some pathologists include under inflammation only that part of the local changes which is of exudative character, while all proliferation of cells and fibrosis taking place at the site of the injury to them constitute repair. These difficulties and differences arise from a didactic and teleological approach to the subject. The cells of the body at the point of injury undergo regressive and progressive changes purely as the result of their natural reaction to alterations in their environment. These individual cell responses may or may not be beneficial to the body as a whole. Very different combinations of these cell reactions are met with in the various types of inflammation, and many of the same cell changes are encountered in repair. To assume that the fibroblasts proliferate in one case to check the spread of an infection and in another to replace damaged tissue is drawing a purposeful distinction. Consequently, any attempt to sharply define inflammation and repair as separate processes must of necessity fall down on natural grounds." The view expressed by Dr. Waugh is held by many pathologists. However, I, along with other pathologists, hold that repair is related to cellular generation, growth, and differentiation rather than to inflammation. I would maintain that the fundamental biologic phenomena of repair are related to those encountered in the study of embryology, of growth, or of neoplasia rather than to those of inflammation, and I regard it as a conceptual error to fail to distinguish between inflammation and repair.

*Waugh, T. R. In Mosley, Textbook of Surgery, eds. 1 and 2, St. Louis, 1932, 1935, The C. V. Mosby Co., chap. 2.

A debate on these views cannot be undertaken here. Suffice it to point out that the very coexistence of such radically different concepts as those expressed above is a measure of how great is our ignorance about some of the fundamental principles of inflammation and repair.

SIMPLE INFLAMMATION

The immediate causes of inflammation must injure the tissues. The causal agents may be *physical* in nature, such as heat, cold, mechanical injury, or radiant energy, or *chemical* and caustic, corrosive, or poisonous. *Living agents* such as *microorganisms* or *parasites* are usually separately classified as causes of inflammation because of their special behavior in relation to the host and their ability to live and multiply in the host and to induce immunologic reactions.

The essential reactions of simple inflammation are an orderly sequence of events. The process is remarkably similar from example to example. It does not vary, except in detail, from patient to patient, from site to site, or under different etiologic circumstances, however, some of the variations in detail may be of clinical importance. The orderly and progressive inflammatory process may halt at any stage of its development and regress. It does not always go through its entire sequence of stages, although their order is constant. The latter stages of the process cannot occur in the absence of the earlier ones.

VASCULAR REACTIONS IN INFLAMMATION

In simple inflammation vasomotor effects and consequences are of very great importance. The process begins, if the insult is abrupt enough and affects a sufficient region, with a momentary vasoconstriction, which is followed by capillary and arteriolar dilatation. The region is the site of an active hyperemia and is redder than normal. If the injury is on or close to an external or peripheral part of the body, the lesion will be warmer than usual. Further capillary dilatation, with or without some subsequent arteriolar narrowing, causes a reduction in the linear rate of blood flow through the capillary bed, although the volume of flow continues to be greater than normal. The lesion is reddened, perhaps slightly cyanosed, and less

warm than previously. It will be surrounded by a margin of active hyperemia that remains a brighter red color. This is a peripheral zone where the capillary bed is less markedly dilated and where the flow remains relatively rapid.

Concomitantly with these phenomena the capillary endothelium becomes swollen and presents an increased resistance to the flow of blood along the capillaries. It also, while retaining some semipermeable qualities, becomes more permeable to fluids and crystalloids and allows larger molecules to pass through its wall in greater quantities than normal. These reflections of capillary injury are accompanied by the transfer of an increased amount of fluid and protein to the interstitial spaces and by a further reduction in the rate of blood flow in the dilated capillary bed. Capillary blood sludging or coagulation may occur in some vessels. The fluid that escapes into the interstitial area flows on the surfaces of cell membranes and fibrillar strands at first but soon diffuses freely into the ground substance gel and renders it more fluid. A new equilibrium is established between the outflow of fluid from the capillaries, interstitial edema, fluid return to venular capillaries, and lymphatic drainage. The site of injury is swollen.

The vasomotor effects of simple inflammation in the skin have both chemical and nerve reflex mechanisms. Sensory stimuli carried along axons from the area of injury pass antidromically to the arterioles about the area and cause them to dilate. Abolition of the axon reflex will prevent development of some of the active hyperemia normally associated with cutaneous inflammation so that the site fails to develop a prominent peripheral flare. The nervous system also exercises some control over the inflammatory response in a more general vasomotor effect. It is observed that section of sympathetic nerves with relaxation of vasoconstriction enhances inflammatory effects, while the abolition of vasodilator action restricts inflammatory processes. These more general aspects of nervous control, however, are not fundamental to the inflammatory process. The axon reflex is thought to be initiated by a histamine-like substance or substances acting on sensory nerve endings. Histamine-like material, the so-called *H substance*, is released by cells at the local site of injury. It affects directly the walls

of capillaries and minute vessels to cause their vasodilatation. It is the direct action of the histamine-like material on these blood vessels that is responsible for capillary dilatation and the consequent capillary hyperemia and slow rate of blood flow in the affected region. This hyperemia causes a dull red color in the central area of injury.

As we have noted, the vasomotor effects of inflammation, unless they are very slight, are accompanied by an increased permeability of the affected capillaries and by local edema. The causes of increased capillary permeability are not entirely established. Immediate effects are probably due to H substances for it can be shown that histamine not only causes vasodilatation but also rapidly increases capillary permeability to dye substances injected into the blood stream. A similar increase in capillary permeability results from a substance or substances found in inflammatory exudates and called *leukotaxine*. It is observed that crude leukotaxine causes endothelial swelling, increased capillary permeability, and emigration of leukocytes from blood vessels. The material appears to result from the partial lysis of protein and to be a mixture of peptides. It has been shown that peptides composed of a chain of 8-14 amino acids will cause increased permeability. They may be responsible for the increased permeability of prolonged inflammation but, since their formation is relatively slow, will not account for the immediate or H-substance effect. It should be remembered that the hemodynamic changes occurring in the capillary bed tend to increase the capillary blood pressure and hence to promote the passage of fluid from the capillary and to hinder its return at the venous end of the capillary. Similarly, with the escape of a fluid containing more protein than usual, the normal differential in osmotic pressure between the plasma and the interstitial fluids tends to decrease. Moreover, the capillary membrane itself is no longer so effective in the creation of osmotic effects. All of these phenomena tend to cause local edema.

CELLULAR REACTIONS IN INFLAMMATION

As the vascular phenomena described above develop and persist, the emigration of leukocytes from the blood to the interstitial tissues

occurs. The cellular reactions of inflammation first affect the local tissue histiocytes. These are phagocytic cells that belong to the reticuloendothelial system and occur in variable numbers in the connective tissues of the body. They have little ability to migrate and only a relatively modest proliferative activity. However, they are avid phagocytes and they constitute the first cellular line of defense in inflammation. They are rapidly stimulated in simple inflammation. In those areas where they are numerous, bacterial inflammations may be quickly confined and terminated.

If the inflammatory process progresses, emigration of leukocytes from capillaries occurs. The polymorphonuclear neutrophilic leukocytes are the first cells to do this. They are followed by mononuclear leukocytes within an hour or two, and many investigators believe that during the subsequent several hours very numerous lymphocytes also emigrate and rapidly transform into mononuclear macrophages. Cycles or waves of cellular emigration may occur. The phenomenon begins as the capillary endothelium becomes swollen and altered, as the linear rate of blood flow slows, and as endothelial permeability increases. Leukocytes tumbling over the surface of the endothelium tend to adhere to swollen endothelial cells and intercellular cement substance. There occurs a margination or pavementing of leukocytes, especially neutrophil polymorphonuclear cells, on the walls of the capillary vessels. If the injury is sufficient, many of these cells will migrate by amoeboid movement through the capillary wall between endothelial cells. Their passage is aided by the intracapillary blood pressure.

The direction of motion of migrating leukocytes may be influenced by the presence of substances that are said to be chemotactic. *Chemotaxis* is a phenomenon of obscure mechanism but of widespread biologic occurrence. It is exhibited by such things as the spermatozooids of certain plants, by certain myxomycetes, amoebae, and other organisms, as well as by leukocytes of various sorts in various species of animals. There is little reason to believe that the pavementing of leukocytes or their emigration from the capillaries is influenced by chemotaxis, since there is no evidence that chemotactic material can act across the wall of the blood vessel. However, it remains possible

septa within the abscess of the fat-pad need division with a scalpel (fig. 58). The lips of the cutaneous incision should be trimmed elliptically to prevent premature closing of the skin.

Deep Plantar Abscess.—The central plantar space situated deep to the plantar fascia is arranged like an apartment house of four stories, each of which is occupied by the muscles that continue the flexors of the toes. Infection of the various floors becomes increasingly less common as one proceeds from the ground floor, upwards. For drainage of the central plantar space an incision parallel to, and just above, the medial border of the foot in the neighbourhood of the instep, is made (fig. 59).

Infections of the Dorsum of the Foot

The dorsal subcutaneous space usually is infected by extension from a subcutaneous interdigital space or a web space, while the dorsal subaponeurotic space is infected either from a direct puncture or from involvement by extension from the deep plantar space. To drain the former space the incision should be placed distal to the dorsal venous arch, but in the line of the digital vessels and nerves, in order to avoid them. To drain the latter space it is best to confirm the presence of pus by attempting aspiration, and if diagnostic aspiration is positive, to make a longitudinal incision alongside the needle.

Leprosy not infrequently attacks the toes, and the initial manifestation may simulate a chronically infected corn (fig. 60). Leprosy is discussed on p. 25.

Fig. 60.—Leprosy of the little toe (O. D. Adnan, F.R.C.S., Bombay)



Fig. 61.—Madura foot. (Dr. M. M. Schapiro, Honduras)



Madura foot (syn. mycetoma pedis) is a chronic granulomatous disease encountered especially in tropical countries, notably in certain parts of India but with increasing frequency in territories where hitherto it has been unrecognized, such as the Southern United States, South America, and Cuba. The disease is caused by a genus of filamentous fungus (*Odopora madurae*) resembling actinomycosis and abounding in road dust. In nine out of ten cases the organism gains entrance through a prick in those who go about barefooted. The first manifestation is a firm, painless, rather pale nodule. Soon other nodules appear. Later the nodules become surmounted by vesicles that burst to form discharging sinuses. In the watery discharge granules can be discovered, sometimes only with perseverance. On some

Fig. 59.—Incision for draining the central deep plantar space.



that pavementing of leukocytes occurring at the venous end of the capillary, where fluid is returning across the vessel wall from the interstitial region to the blood, might be enhanced by chemotaxis, and it is possible that leukocytes that have already migrated partially across a capillary membrane to lie both within and without the capillary may be directed to complete their emigration by chemotaxis. In any case, the role of chemotaxis becomes definite once the responsive leukocytes gain the interstitial spaces. Many substances appear to exert a chemotactic influence on leukocytes, and many bacterial and such a substance as starch grains possess this quality. On the other hand, there are differences of opinion as to whether or not dead tissue and tissue products resulting from autolysis or partial enzymatic digestion may be chemotactic for leukocytes. In this connection it may be noted that the prominent chemical necrosis of cells in the pancreatic islets that follows an intravenous injection of alloxan into experimental animals occurs without any emigration or accumulation of leukocytes. Indeed within 24-48 hours after such an injection, many necrotic cells have been entirely removed from the islets without the participation of leukocytes. Similarly in the human aorta diseased by medionecrosis, an appreciable amount of necrotic tissue may be seen in the media of the aorta in the absence of any leukocytic accumulation.

With the emigration and accumulation of leukocytes the inflammatory process has reached its full development. More emigration of cells may occur with suitable stimulation, but further changes involve the phagocytosis of particulate material, the disintegration and disappearance of polymorphonuclear leukocytes, the disappearance of mononuclear cells of various sorts, and the subsidence of the vascular phenomena. In the course of these changes host tissue may be destroyed and removed and pus may form and be absorbed or require drainage. The final regressing phases coincide with the initiation of healing.

Whether or not one regards the inflammatory process as purposive, its elements have certain predetermined effects in the local area of reaction. The vascular phenomena are essential to the formation of the fluid and cellular exudates. Hyperemia and the accompanying

edema serve to dilute and flush away toxic substances and waste materials. Hyperemia and edema increase the circulation of antibodies, nutrients, and antibacterial drugs in the area. As capillary permeability increases, large molecules such as fibrinogen escape into the tissues and fibrin may precipitate interstitially. The formation of a fibrin clot in the tissues assists phagocytes to capture and engulf particulate matter by surface phagocytosis. It also provides a partial mechanical barrier to the spread of infection and may reduce drainage of fluid from the lesion.

Role of the Cellular Exudate

The function of the several types of cells that participate in simple inflammation is not well understood. The *tissue histiocytes* are phagocytic to particulate matter and bactericidal for most organisms. They may successfully contain and limit an appropriate etiologic agent; obviously they cannot do this if the causal agent of the inflammation is a true chemical solution, a mechanical force, or is radiant energy. The *neutrophil leukocytes* emigrate promptly, are actively motile, and are phagocytic and bactericidal for many bacteria. Their life span in an inflammatory focus is 3-5 days or less. Many of them disintegrate rapidly to release their enzymes into the milieu of the reaction, assisting in the digestion of protein matter and forming pus. Others round up, acquire a pyknotic nucleus, and appear somewhat like lymphocytes in histologic preparations. If the region of inflammation is sufficiently large or if it is a smaller lesion that cannot drain but develops in the tissues under pressure, a systemic leukocytosis may occur. The products of certain bacteria, nucleoprotein, and possibly cytoplasmic breakdown products absorbed from the lesion are effective stimulants to cause leukocytosis, but the mechanisms of leukocytosis in inflammation are not understood. *Eosinophil leukocytes* are mobile, attracted by chemotaxis, and phagocytic. Their function in the body is not known in detail. Eosinophil leukocytes appear especially in inflammatory reactions of allergic etiology, but they are not limited to them. For example, in the inflammatory reaction occurring in acute myocardial infarction a brief influx of eosinophil leukocytes

is observed (see Fig. 5) The blood monocytes emigrate into inflammatory foci in considerable numbers and transform with considerable speed into large macrophages. They respond to chemotactic substances in much the same way as neutrophil leukocytes and may show an especially strong attraction to organisms such as *Mycobacterium tuberculosis*. They are phagocytic, bactericidal for many bacteria, and tend to survive as scavengers. They are

capable of multiplication at the site by mitotic division. They either migrate away from a subsiding area of inflammation, remain as tissue macrophages fixed in the area, transform to epithelioid cells, to giant cells, or possibly to fibroblasts, or they may disintegrate. The function of lymphocytes in inflammatory lesions is a subject of debate. Many pathologists believe that their local function is unknown; others, the author among them, maintain that lympho-

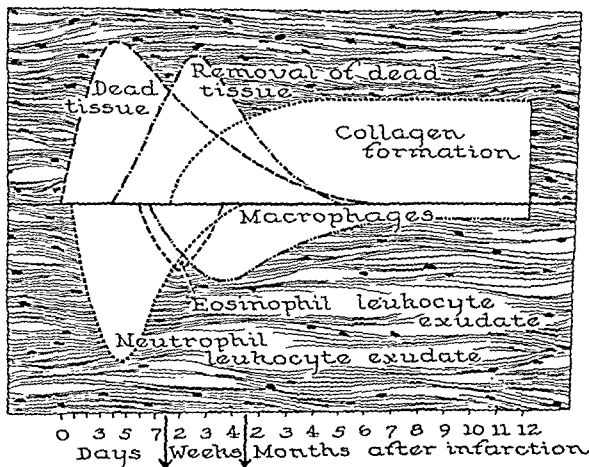


Fig. 5. The diagram represents some of the sequence of events that occurs at the site of a myocardial infarct. Following ischemic infarction and the death of a portion of myocardium there develops locally a simple acute inflammatory reaction. There is an emigration of neutrophil leukocytes that reaches its height within about 4 days and subsides in about 3 weeks. The removal of debris from the infarcted region is apparent about 3 days after infarction, it reaches its height in about 2½ weeks and may continue for several months. Occasionally in large infarcts a sequestrum of dead tissue may be left. Eosinophil leukocytes are seen in the lesion during the second and third weeks. Macrophages become prominent in the lesion toward the end of the first week and are most numerous between the third and fourth weeks. A small number of macrophages, some of which may contain hemosiderin, and lymphocytes may persist in the lesion for many months. Fibroblastic proliferation can be seen within a few days after infarction, appreciable production of collagen begins during the second week, increases rapidly during the third week, and is almost completed within 3 months following infarction. The interval of time that must elapse before collagen formation is completed will depend on the size of the infarct. The scar that is formed is always smaller in volume than the portion of myocardium that was infarcted. (Based on the data of Mallory, G. K. White P. D., and Salcedo Salazar, *J. Am. Heart J.* 18, 617, 1939.)

The experimental reaction

GRANULOMATOUS INFLAMMATION

COMMENT ON THE INFLAMMATORY REACTION

THE INFLAMMATORY REACTION

We have described inflammation as a local reaction to injury and have implied that it is a constant and orderly process. While this is a valid generalization, it is also true that the inflammatory reaction may be considerably modified by the constitutional and pathologic

status of the patient in whom the reaction is progressing. Some of the aberrations that may occur in the inflammatory reaction are of clinical interest.

SIMPLE INFLAMMATION

Influence of Tissue Structure

The types and numbers of cells that participate in an inflammatory reaction will depend on the nature and site of the tissue affected, on its vasculature, on the nature of the causal agent, and on the patient's hematologic status. Some parts of the body such as the liver, spleen, lymph nodes, and bone marrow contain great masses or filters of tissue macrophages. These are the main organs of the reticuloendothelial system. In them simple inflammation is modified by the presence of enormous numbers of tissue histiocytes. Indeed, so numerous are the phagocytes and so available is their supply that one seldom sees more than a small number of polymorphonuclear leukocytes in a lymph node that is the site of an acute inflammatory reaction. The acutely inflamed node is commonly swollen and soft with lymph fluid and hyperplasia of phagocytic cells, but only uncommonly will it be found to be suppurative. Similarly, the surfaces of the pericardial, pleural, and peritoneal spaces are richly supplied with tissue macrophages and these cells participate generously in simple inflammatory responses. However, in these spaces exudates can spread and accumulate, and since the tissue macrophages are relatively fixed, the cellular accumulation is largely hematogenous in type. It exudes from the rich vascular networks and lymphocytic tissues that lie close beneath the serous membranes. The inflammatory responses of the alveolar sponge of the lung are very much like those of the serosal spaces. The tissue presents an enormous surface area, richly vascularized and abundantly furnished with phagocytic cells. The latter are somewhat mobile and participate vigorously in the inflammatory reaction. However, the inflammatory exudates that spread and accumulate in the alveolar spaces contain chiefly cells of hematogenous origin. There is evidence that the lung, both normally and pathologically,

may act as a remarkable filter for the sequestration of polymorphonuclear leukocytes. This phenomenon will contribute to the cellular population in simple inflammation of the lung. The lung can drain its exudates through the bronchial tree. It is a curious and, so far as one is aware, unexplained fact that exudates of inflammatory cells in joint spaces lined with synovium contain many polymorphonuclear leukocytes, even in chronic disease. In the general connective tissues of the body, as in the subcutaneous tissues, histiocytes are relatively sparse and the cells that participate in simple inflammation are derived in great majority from the blood.

Influence of the Patient's Hematologic Status

Obviously the course of the inflammatory process at any site will depend on the nature and number of cells available. In general, the process is fairly uniform from patient to patient and whatever differences are noted lack apparent explanation. Major and explicable differences are occasionally observed. The most obvious of these occur in patients in whom the hematopoietic organs fail to produce polymorphonuclear leukocytes. In the most severe form of chemically (drug) induced acute agranulocytosis or malignant neutropenia, the polymorphonuclear cells, and to a lesser extent other leukocytes, may be so deficient in the blood that they are absent or nearly absent from foci of acute inflammation. The body's defenses against infection are correspondingly deficient and the patient becomes acutely ill with chills, fever, and mucosal ulceration. Lesser degrees of leukopenia and neutropenia occur under many circumstances, e.g., typhoid fever, measles, malaria, profound septicemia, aplastic anemia, systemic lupus erythematosus, and others, but, in general, the effect on the inflammatory process is clinically slight or negligible. The resultant leukopenia may delay transiently the proportionate accumulation of leukocytes at a site of inflammation, but adequate numbers of cells usually do accumulate rapidly. Nevertheless it may be noted that such leukopenic patients may be more susceptible to infective complications such as furunculosis, pneumonia, and other infective lesions. The larger a focus of

simple inflammation is, the more will it depend on the hematopoietic organs to supply hematogenous leukocytes. In some patients the bone marrow is incapable of responding adequately because it is replaced by fibrosis, by lymphomatous cells, or by carcinoma; the number of leukocytes circulating in the blood may be normal or reduced. An unexplained anergic state of the marrow is not unusual in elderly patients and may be encountered rarely in younger individuals. The number of leukocytes circulating in the blood may be normal and the bone marrow apparently normal, yet these patients do not respond with leukocytosis or with fever to such stimuli as bacterial pneumonia or peritonitis. In such patients the cellular processes of inflammation are initiated, but the usual reaction to injury is not sustained.

It is also observed that the accumulation of cells in a focus of acute inflammation will be partially governed by their proportions in the blood. For example, in rabbits with an experimentally induced blood monocyto- sis the number of monocytes that enter into an inflammatory focus is roughly proportional to their number in the blood. Similarly, it has been reported that the blood monocytes were an important source of macrophages in a simple inflammatory process in a patient with a form of chronic leukemia in which there were numerous circulating monocytes. The microscopic examination of simple inflammatory foci from patients with a prominent eosinophilia commonly discloses a slight increase in eosinophil leukocytes in the inflammatory lesions. It should be observed, however, that since the clinically significant inflammatory reaction usually lasts several days and since repeated waves of cellular immigration may occur, the proportionate distribution of leukocytes in the blood seldom affects the cellular composition of the inflammatory focus in more than a transient or minor fashion.

Influence of Functional Variations of Leukocytes

Of great interest are those patients in whom the leukocytes are functionally deficient. Leukocytes are complex metabolic units that have numerous enzyme systems and many functions in the body's economy. In the inflammatory

process the ability of leukocytes to migrate to a focus, the ability of monocytes and probably lymphocytes to transform into macrophages, and the phagocytic, bacteriocidal, and proteolytic functions are all of great importance. Similar sorts of leukocytes in different patients are not all identical in these properties. For example, neutrophil leukocytes have a fragility that is said to be inversely proportional to their content of vitamin C; immature granulocytes have little phagocytic activity, although young mature granulocytes are the most effective of phagocytes; there is a decrease of phagocytic activity in animals that are deficient of protein, ascorbic acid, or certain of the B vitamins; excessive amounts of cortisone or corticotrophin adversely affect phagocytosis and diminish the ability of the host to resist invasion by microorganisms; it has been suggested that there may be a deficiency in leukocytic bacteriocidal action in diabetic animals. The most striking deviations in leukocytic function in relation to inflammation are seen in patients with leukemia. Many such patients retain some production and circulation of mature granulocytes and other leukocytes and are capable of producing an inflammatory response. Other leukemic patients may lack sufficient normal leukocytes to respond adequately. A patient with an acute myelogenous leukemia, for example, may possess only primitive cells that migrate slowly, respond poorly to chemotactic stimuli, possess little or no phagocytic activity, have poor bacteriocidal properties, and yield low proteolytic activity. The pathologist is accustomed to see at autopsy what he terms agranulocytic pneumonia or agranulocytic abscesses in such patients. The modification of the cellular reactions of simple inflammation may be so extreme in leukemic diseases as to prevent the formation of pus or the localization of the reaction.

Influence of Vascular Supply

The roles of vasomotor activity, hemodynamic effects, semipermeability of membranes and fluid exchange, and of lymphatic drainage are important in the development of simple inflammation. They may vary to an extent that may markedly influence the general inflammatory process.

Influence of Ischemia

Obviously the acute inflammatory process cannot develop in the usual fashion in an avascular tissue, in a tissue deprived of the circulation of blood, or in an anoxic area. It will be modified by a pre-existing passive hyperemia, by edema, and by factors that affect vasomotor activity or capillary permeability. A large area of ischemic infarction may never show an inflammatory response in its central part because this area is too far removed from the circulation of blood at the periphery of the lesion. Instead, the central necrotic part may remain as a sequestrum or may liquefy. Lesions of this sort may be seen occasionally in large infarcts of the spleen or kidney and in ischemic gangrene of an extremity. The influence of a partial ischemia on the inflammatory reaction can be seen where areas of ischemic necrosis meet viable tissue that has an adequate circulation. The entire simple inflammatory process becomes attenuated and depleted in the more ischemic area. The tissue macrophages fail to function as a first line of defense while the leukocytes and hematogenous macrophages enter the more ischemic zone in reduced numbers and manifest a reduced function. The leukocytes and hematogenous macrophages may wander into the ischemic tissue beyond the threshold of adequate nutritional supply for their own maintenance and themselves die of ischemia. The depletion of inflammatory process to some degree by ischemia is not infrequent. The susceptibility to infection of the arteriosclerotic lower limb, of the lungs in pulmonary fibrosis, or of the skin affected by late changes due to irradiation, is, in part at least, a manifestation of the alteration induced by ischemia in the inflammatory response.

Influence of Edema

Excessive interstitial fluid or edema in an area also attenuates the inflammatory process. Edema fluid is, except for its content of immune bodies, a good bacterial culture medium; it is a diluent of toxins, but also of enzymatic and active substances released in the inflammatory process, it is associated with some degree of reduced blood flow in the area, and in anatomic circumstances where the swelling of

edema is rigidly confined, marked ischemia may result; it increases the distances across which respiratory and nutritional exchanges must occur; its fluid state makes more difficult the capture and surface phagocytosis of particulate matter; by separating tissues and membranes, by diluting the ground substance, and by the flow of its fluid, irritating matter may spread more easily and widely. It is observed that children with the nephrotic syndrome and hydropericardium, hydrothorax, and hydroperitoneum may succumb to an intercurrent infection of the fluid-filled serous cavities, commonly with pneumococci. The pathologist sees frequently at autopsy an early pneumonia in association with the hypostatic pulmonary edema that occurs so commonly a few hours or days before death in patients with some degree of terminal cardiac failure. The edematous lower limb of a patient with congestive heart failure and diabetes mellitus is susceptible to infection. An area of sacral edema is particularly liable to decubitus ulcer formation. Such examples of enhanced susceptibility to infection may show that edema may interfere with the inflammatory process, but it should be remembered that the inflammatory process is a complex one. For example, patients with chronic renal failure due to glomerulonephritis are commonly more susceptible to infection whether they are nephrotic or not; patients with ascites due to cirrhosis of the liver or peritoneal neoplasm do not seem especially susceptible to infective peritonitis unless infected during paracentesis; patients with pulmonary edema due to a transient episode of cardiac failure have only a slightly increased susceptibility to pneumonia, the edematous feet and ankles of a patient with congestive heart failure but without diabetes mellitus show only the most minor increase in susceptibility to infection; and patients with the indurated, chronic lymphedema of Milroy's disease probably do not manifest a significant disturbance in the inflammatory process in the affected limb even though lymph drainage is presumed to be deficient. It can be shown in experimental animals that the vasomotor nerves can influence the reaction rates of simple inflammation. For example, sympathectomy with consequent vasodilatation enhances the inflammatory process, while unbalanced sympathetic

nerve vasoconstriction delays and diminishes it. However, in clinical practice these effects seem minor or negligible. The neurosurgeon is not impressed with the enhanced inflammatory reaction in a previously normal limb of a patient who has undergone a lumbar sympathectomy. The vascular surgeon, however, may be considerably impressed with the more normal inflammatory reaction that may be possible following sympathectomy to improve the circulation of a previously ischemic limb.

GRANULOMATOUS INFLAMMATION

Granulomatous inflammation is somewhat simpler in the ebb and flow of its events than simple inflammation. Its relative cellular simplicity and its longer duration often create lesions of specific architecture, yet the process in general shows less of the sort of variability that has just been ascribed to simple inflammation. Nevertheless, examples of such variation are easily found; the susceptibility of the patient with uncontrolled diabetes mellitus or with silicosis to pulmonary tuberculosis is well recognized, although the causes are obscure. In granulomatous inflammation other kinds of variations may be prominent. Granulomatous inflammation is the form of inflammation in which the influences of natural and acquired immunities and hypersensitivities find their most complex morphologic expressions. It is only necessary to point to the enormous clinical and pathologic differences between a first infection and a second infection in pulmonary tuberculosis to demonstrate how effective these influences may be in modifying a granulomatous inflammatory process that is identical in its basic cellular reaction in both forms of tuberculosis. The value of an acquired immunity to tuberculosis is well recognized. It is rather characteristic, but by no means constant, for granulomatous inflammation to be most frequently localized in those tissues and organs that anatomically contain the most macrophages or through which monocytes and lymphocytes migrate in largest numbers. Again tuberculosis provides pertinent examples and exceptions. There is, for example, an unusual and clinically confusing form of tuberculosis in which the lesions are almost entirely confined to the lymph nodes and other reticulo-

endothelial organs of the body, the lesions may be widely disseminated in them. Contrarily, tuberculosis of the skin can occur following direct inoculation. Many of the lesions of granulomatous inflammation acquire importance because of recrudescence or chronicity leading to cumulative injury; many become important because of a multiplicity of small, focal, tumorlike lesions that ultimately obstruct or preclude some body function.

INTERRELATION OF INFLAMMATION AND CHEMOTHERAPEUTIC AGENTS

The nature of the inflammatory reaction to bacteria may influence the chemotherapeutic approach to a disease. Again it is commonly observed that the panoply of the inflammatory process may be obviated by modern chemotherapy. Antimicrobial drugs may be bacteriostatic or bactericidal or both, but in order to be effective they must contact the bacteria in suitable concentration, whether the latter are intracellular or extracellular and whether in viable or necrotic tissue. Some agents, such as the sulfonamides, are essentially bacteriostatic and require the active participation of leukocytes and macrophages for their effectiveness. Other agents, such as penicillin, Terramycin, and tetracycline, are bactericidal and can kill susceptible bacteria without the participation of leukocytes and macrophages, although, of course, all agents will be aided by the ordinary natural and acquired defenses of the body. In the case of many acute bacterial infections in which the bacteria are largely present in the extracellular fluids, where they live and grow, the organisms can usually be rapidly eliminated by suitable antimicrobial drugs. Since no drug kills all bacteria at once but may require several hours or a few days to accomplish this, several days of therapy are necessary. Nevertheless, the inflammatory reaction as such is usually much minimized by such therapy and will subside before its usual course is run. It should be noted that the vascular and permeability effects of simple inflammation that serve to increase the flow of interstitial fluid in an area of inflammation also serve to bring blood-borne chemotherapeutic agents to the site. Indeed, in some cases

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Influence of Ischemia

Obviously the acute inflammatory process cannot develop in the usual fashion in an avascular tissue, in a tissue deprived of the circulation of blood, or in an anoxic area. It will be modified by a pre-existing passive hyperemia, by edema, and by factors that affect vasomotor activity or capillary permeability. A large area of ischemic infarction may never show an inflammatory response in its central part because this area is too far removed from the circulation of blood at the periphery of the lesion. Instead, the central necrotic part may remain as a sequestrum or may liquefy. Lesions of this sort may be seen occasionally in large infarcts of the spleen or kidney and in ischemic gangrene of an extremity. The influence of a partial ischemia on the inflammatory reaction can be seen where areas of ischemic necrosis meet viable tissue that has an adequate circulation. The entire simple inflammatory process becomes attenuated and depleted in the more ischemic area. The tissue macrophages fail to function as a first line of defense while the leukocytes and hematogenous macrophages enter the more ischemic zone in reduced numbers and manifest a reduced function. The leukocytes and hematogenous macrophages may wander into the ischemic tissue beyond the threshold of adequate nutritional supply for their own maintenance and themselves die of ischemia. The depletion of the inflammatory process to some degree by ischemia is not infrequent. The susceptibility to infection of the arteriosclerotic lower limb, of the lungs in pulmonary fibrosis, or of the skin affected by late changes due to irradiation, is, in part at least, a manifestation of the alteration induced by ischemia in the inflammatory response.

Influence of Edema

Excessive interstitial fluid or edema in an area also attenuates the inflammatory process. Edema fluid is, except for its content of immune bodies, a good bacterial culture medium; it is a diluent of toxins, but also of enzymatic and active substances released in the inflammatory process, it is associated with some degree of reduced blood flow in the area, and in anoxic circumstances where the swelling of

edema is rigidly confined, marked ischemia may result; it increases the distances across which respiratory and nutritional exchanges must occur; its fluid state makes more difficult the capture and surface phagocytosis of particulate matter; by separating tissues and membranes, by diluting the ground substance, and by the flow of its fluid, irritating matter may spread more easily and widely. It is observed that children with the nephrotic syndrome and hydropericardium, hydrothorax, and hydroperitoneum may succumb to an intercurrent infection of the fluid-filled serous cavities, commonly with pneumococci. The pathologist sees frequently at autopsy an early pneumonitis in association with the hypostatic pulmonary edema that occurs so commonly a few hours or days before death in patients with some degree of terminal cardiac failure. The edematous lower limb of a patient with congestive heart failure and diabetes mellitus is susceptible to infection. An area of sacral edema is particularly liable to decubitus ulcer formation. Such examples of enhanced susceptibility to infection may show that edema may interfere with the inflammatory process, but it should be remembered that the inflammatory process is a complex one. For example, patients with chronic renal failure due to glomerulonephritis are commonly more susceptible to infection whether they are nephrotic or not; patients with ascites due to cirrhosis of the liver or peritoneal neoplasm do not seem especially susceptible to infective peritonitis unless infected during paracentesis; patients with pulmonary edema due to a transient episode of cardiac failure have only a slightly increased susceptibility to pneumonia; the edematous feet and ankles of a patient with congestive heart failure but without diabetes mellitus show only the most minor increase in susceptibility to infection; and patients with the indurated, chronic lymphedema of Milroy's disease probably do not manifest a significant disturbance in the inflammatory process in the affected limb even though lymph drainage is presumed to be deficient. It can be shown in experimental animals that the vasomotor nerves can influence the reaction rates of simple inflammation. For example, sympathectomy with consequent vasodilatation enhances the inflammatory process, while unbalanced sympathetic

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natural barriers to permeation may be partially broken. For example, the blood-brain barrier is normally impermeable to penicillin, but in the case of an acute meningitis the barrier may be partially broken and a small amount of penicillin and other nonpermeating substances may find their way into the cerebrospinal fluid. It should also be noted that agents that cannot penetrate cells in effective amounts cannot be expected to act on viable organisms within phagocytes or other cells. For example, the tubercle bacillus may survive and grow within macrophages, optimal chemotherapy requires the use of a drug that will reach these bacilli as well as those that are free in the interstitium or in necrotic tissue. Those inflammatory reactions in which extensive exudates, extensive tissue necrosis, or extensive areas of relatively avascular fibrous or cellular tissue develop may prove difficult to treat with antimicrobial drugs. While the increased permeability of vessels in areas of acute or subacute inflammation may allow the chemotherapeutic agent increased access to the area, the permeation of such agents into extensive, relatively avascular lesions, such as larger abscesses, empyemas, the vegetations of bacterial endocarditis, caseous foci in tuberculosis, impending arteriosclerotic gangrene of the lower limb, the diffuse interstitial fibrosis of extensive chronic pyelonephritis, the sequestra of chronic osteomyelitis, or the more avascular foci of neoplastic growth, is generally slow or ineffective in concentration. The surgical ablation of such lesions is usually warranted whenever practicable. It may be remarked that modern chemotherapeutic, antimicrobial agents have made great changes in therapy and now permit successful surgical procedures under conditions that were previously impossible, nevertheless, they in no way substitute for surgical competence or allow the relaxation of aseptic technique. In general, the more chronic a bacterial inflammatory process may be, the more unsatisfactory and prolonged is its treatment with antibiotics. Not only is it more difficult to bring antibiotics effectively to bear against the organisms in such lesions, but this difficulty makes more likely the evolution of resistant strains of bacteria that adapt to an environment containing the therapeutic agent.

It has been the purpose of this comment on the inflammatory reaction to emphasize some of the aspects of the inflammatory reactions to injury that may vary from site to site and from patient to patient. Obviously, the discussion has been fragmentary and important omissions have occurred, especially in relation to immunologic phenomena. However, it should be clear that the nature and course of an inflammatory reaction need not be constant, but that they may be modified by the constitution and status of the patient. Indeed, while present-day knowledge of the pathophysiology of inflammation is not great, nevertheless, it is often possible to recognize or anticipate certain deviations in the process in appropriate patients and to govern therapy accordingly.

REPAIR OF LESIONS

Following the demolition of injury and inflammation repair occurs. This may take place by regeneration in such a fashion that the tissue defect is occupied by the cells normal to the area in normal numbers, proportions, and arrangement and the region is entirely restored in structure and function. The blood is able to restore itself in this manner. Minor injuries to organs that involve the loss of scattered cells without damage to the reticulin and connective tissue scaffold in which they grow may heal with complete restitution. The repair of fractures of bone may be remarkably complete. More commonly, however, a defective area in an organ is replaced by connective tissue, largely fibrous tissue, that constitutes a scar. The repair process may combine both regeneration and replacement phenomena as in the healing of an incision of the skin.

Repair of Connective Tissue

Following an incision or wound of the skin and deeper structures of, say, the abdominal wall, the wound edges will gape. Approximation of the edges with sutures reduces the size of wound to be healed and will place structurally similar layers of the abdominal wall in apposition. The remaining small spaces between the edges of the wound are sealed by blood and plasma that exudes from damaged

and severed capillaries and is clotted in the presence of tissue juices from damaged cells. Following a period of vascular constriction that assists in hemostasis, vasodilation will occur and, if hemostasis is not adequate, further hemorrhage may occur. With the onset of hyperemia at the site of incision, a typical inflammatory process is established. In the case of a clean surgical incision with minimal cellular trauma, the inflammatory phenomena are also minimal. However, in wounds made by crude mechanical trauma with much cellular injury, the inflammatory response is marked. It will also be marked in infected wounds, in wounds contaminated by foreign material, or in wounds containing much dead tissue. Appreciable contamination of the wound will inhibit healing and, because of bacterial enzymes or as a result of inflammation, may bring about the solution of the clot material that initially unites the edges of the wound. Within 24-48 hours the fibrocytes that lie at the margins of a clean incision begin to multiply, and within 3-4 days from the time of injury they migrate into the clot material along fibrin threads. They are accompanied by macrophages and also by capillary buds that unite to form capillary arches. The clot is thus invaded by vascular connective tissue from all sides where it touches the host's tissue. The macrophages lyse the clot and remove it. Within 4-5 days the clot will have been replaced by vascular connective tissue growing in an organized manner. The process of organization joins the edges of the wound and replaces the previous fibrinous union by fibrous union. The wound rapidly gains tensile strength as the fibroblasts begin to assume the characteristics of fibrocytes and collagen is formed between them. A high level of strength is attained within 10 days. The tissue that unites the edges of the wound undergoes a subsequent maturation that may extend over several weeks or months. Much of the rich capillary supply to the newly organized tissue atrophies and only selected channels remain. The fibrous tissue develops a high content of collagen and a sparse content of elastic fibers. Many of the fibrocytes that were present disappear, a small number of macrophages remain as tissue histiocytes. A relatively avascular cicatrix results (see Fig. 5).

Repair of Surfaces

Repair at a body surface also involves *epithelial* or *mesothelial* regeneration. The epithelium of the skin will begin to proliferate as soon as it has a suitable surface over which to grow. In the healing of a clean approximated incision an exudate forms at the surface of the wound, clots, and dries to form a hard, relatively impervious, protective scab. With the organization and vascularization of the clot under the scab, a tissue develops that can support and nourish an epithelium. The epithelium at the immediate edges of the wound flattens and glides across the wound surface as a thin tongue beneath the scab by a mechanism called *thigmotaxis*. Mitotic activity occurs at a slight distance back from the immediate margin of the wound rather than at it. As the epithelium grows beneath the scab it loosens the scab so that it will fall off. Initially there may be some slight growth of epithelium on to the exterior surface of the scab at its periphery, but this is abortive. The epithelium grows across the wound as a covering, containing less than the normal number of cell layers, however, it matures to acquire them. It does not regenerate with complete restitution since appendages and rete pegs do not regrow.

Repair of Specialized Tissues

Other specialized tissues severed in the incision of the abdominal wall may or may not regenerate. *Adipose tissue* probably does not regenerate *sui generis* from adult or regressive fat cells but may grow rather by differentiation from connective tissue fibroblasts or reticuloendothelial cells. *Skeletal muscle* seldom shows mitotic activity, and while it may proliferate abortively to form syncytial, bulb-like terminations on the ends of severed fibers, continuity between severed fibers is only very rarely established. In the healing of a surgical incision of the abdomen it is not expected that any muscle continuity will be re-established. *Nerve fibers* that are severed degenerate distally and much of the free lipid that results from their disintegration is removed by macrophages during the subsequent several days. There occurs a similar, but much less extensive,

degeneration in the central stump of the nerve. Schwann cells proliferate along the course of the degenerated nerve, chiefly in relation to the peripheral fragment and, together with a lesser proliferation of Schwann cells from the central stump, bridge the gap between the severed portions. Axons begin to grow among the Schwann cells within a few days, and under favorable conditions can grow at the rate of 3-4 mm a day. The more accurately the proximal and distal stumps of the severed nerve are in apposition, the more prompt and successful will be the regeneration. The less accurate the apposition, the more disordered is the proliferation of Schwann cells at the point of severance and the greater is the possibility of the growth of an amputation neuroma. The *serosa of the peritoneal surface*, like the cutaneous epithelium, is brought into close apposition with itself when a surgical incision is closed. The remaining spaces in the wound are sealed with a blood or plasma clot. Since the peritoneal surfaces remain moist a dry scab does not form. The milieu of the peritoneal space is particularly suitable for the maintenance and growth of cells, and the single layer of mesothelial cells of the serosa can rapidly proliferate and glide across the wound and cover it. This may occur before organization of the clot has occurred. Excessive exudates of blood or plasma from the wound or excessive injury to the serosa may allow a clot to unite the peritoneum of the abdominal wall with that of some part of the gut before the mesothelium can cover the injuries. The subsequent organization of such connections results in fibrous adhesions. The adhesive bands are covered with mesothelium.

Repair of Larger Defects

The *healing of an open defect* in which the edges of the wound are not approximated differs somewhat from the repair of an incision such as described above. A craterlike defect must gradually fill with new connective tissues from its base and sides. The immediate surface of the wound presents a delicate layer of moist, clotlike exudate into which fibroblasts and capillaries grow. A new thin layer of exudate forms continuously at the advancing surface of organization. The manner in which

capillary arches form at the growth surface gives rise to a granular surface in which small elevations of fibroblastic tissue and exudate form at each point where there is a prominent leash of capillary vessels. When the surface of the wound is inspected it is found to consist of red, richly vascularized, moist tissue with a slightly granular surface. This is *granulation tissue*. The capillaries in it are subject to trauma and it can be easily made to bleed. If infection occurs, pus will be present in the surface exudate and the development of granulation tissue will be inhibited. With minor degrees of infection organization may still proceed; the infected surface of the wound is gradually reduced in extent and the defect is filled with granulation tissue. The epithelium at the edges of the wound lacks a suitable surface to grow across until the wound defect is filled. It proliferates only to a slight degree until this occurs. It then grows across the new surface. Occasionally it may grow down onto the surface of an incompletely filled defect but this is uncommon. In its essential biologic features the repair of a large open defect does not differ from that of an incision that has been approximated. In practice, however, the open wound requires much longer to heal because of the greater amount of tissue that must grow and because it is much more liable to contamination with bacteria or other substances that interfere with repair. Moreover, its surface may be so large that epithelium cannot cover it by growth from the periphery and skin grafts may be required. Granulation tissue covered by epidermis ceases to grow and the wound defect is normally repaired at a level similar to the adjacent undamaged tissue. However, occasionally this does not occur and an elevated mass of redundant connective tissue covered with epidermis grows at the site of an incision or wound. It is called a *keloid*. Certain individuals have a predisposition for this overgrowth of tissue in wound healing. The aberration is related to the constitution of the host rather than to the etiology of the wound.

Repair of Fractures of Bone

The *repair of fractures of bone* shares many of the features of simple wound healing. The fracture disrupts the blood channels within

and without the bone at the fracture site. Hemorrhage and hematoma formation follow. The periosteum may be partially elevated by blood. Repeated mechanical movement of the fracture site causes repeated vascular trauma. Injury to the blood supply of the area will cause some ischemia which, together with mechanical trauma, will result in the death of some tissue. Both bony and soft tissues may be affected. The hematoma, together with any necrotic tissue, is removed by inflammatory reaction and replaced by organization. It is not clear whether the connective tissue cells that organize the clot are fibroblasts that have the ability to differentiate into osteoblastic cells or whether there are specialized cells arising from the periosteum and endosteum that participate in the organization. In any case, the fracture site is united at first by clot and subsequently by a connective tissue mass. This mass forms an elliptical swelling at the site of the fracture. It may be contained within the original periosteum or it may extend beyond it. The structure is called a *provisional callus*. It is highly vascular. The fibroblasts (osteoblasts?) contained in the callus soon begin to elaborate a basophilic fibrillar and amorphous ground substance between them in large quantities. In this substance differentiation to form *cartilage* or *bone* occurs. What determines the differentiation is unknown. *Cartilage* tends to form more freely at the periphery of the callus or where there is much mechanical movement, but it also develops in an apparently random manner. *Bone* develops through a stage in which the ground-substance matrix becomes eosinophilic and compact. This material is called *osteoid*. It is subsequently calcified. The fracture becomes united by a callus formed of connective tissue, cartilage, and bone. The structure is preliminary and is subsequently modified by macrophages, by osteoclasts, and by bone formation until it is transformed into an architecture exactly resembling the normal bone. The swollen callus is thus replaced by normal bone. If surgical reduction of the fracture resulted in good alignment at the time the fracture was set, it may repair so well that no anatomic evidence of injury remains. If, however, perfect alignment of the fractured ends could not be obtained, some residual distortion will remain.

The factors that govern the remodelling of the callus and result in normal bone structure are unknown. Soft tissue injuries adjacent to a fracture are repaired by fibrous scars.

Both *cartilage* and *tendon* repair by the formation of a clot in the defect caused by an injury. Fibroblasts from the surrounding connective tissue grow into the clot. In the case of cartilage, a fibrocartilage is formed and it may subsequently undergo metaplasia to hyaline cartilage. In a tendon, fibroblasts tend to orient longitudinally and to provide tough fibrous union between the severed ends.

COMMENT ON REPAIR OF LESIONS

Regeneration

Regeneration is a remarkable phenomenon that may be organized to an incredible degree in many, but by no means all, primitive creatures. In man, however, it is a rather limited event that seldom results in an organ-like or tissue-like product. Regeneration must be preceded by certain regressive changes, including the completion of the local defense reactions, demolition of damaged cells and detritus, and dedifferentiation of the cells that will regenerate. If the basic stromal pattern of the area is left intact and the parenchymal cells are of a sort that can regenerate, they will replace the demolished cells unit for unit. Regeneration of this type is very common, and a constant cellular replacement goes on in the epithelia and parenchyma of the body. The healing or regeneration observed in toxic myocarditis, e.g., diphtheria, in liver necrosis, e.g., viral hepatitis, or in acute nephrosis, e.g., shock, is often this type. Regeneration in this fashion, however, is prevented if the stromal template of the cells is damaged. The damaged stroma will itself regenerate by fibroblastic growth and the production of new extracellular fibers, but only in an architectural pattern inappropriate for the accommodation of the parenchyma. Nevertheless, reconstitution toward normal may occur, although it should now be regarded in the more general sense as repair rather than regeneration. For example, after incision the repair of the duodenal mucosa may be remarkably detailed, but it is incomplete, since the muscularis does not regenerate. The repair of a wound of the skin

surface involving destruction of cutaneous appendages is well organized but, again, restoration is incomplete

Scar Formation

Extensive injuries to parenchymatous organs with much destruction of stroma heal merely by the formation of a scar that partially replaces the lost tissue but is without its specific functions. This happens in many infarcts in parenchymatous organs and in organs such as the brain where regeneration of specialized cells is not possible. Where possible, the remainder of the parenchymal organ, or the contralateral organ, may undergo some degree of hypertrophy, a specialized form of restitution.

The formation of a scar in a gross tissue defect effects mechanical repair. In many situations the result is satisfactory. However, at some sites scar formation may interfere with function. For example, scar formation about the joints may limit movement, a scar about the lower eyelid may cause its eversion, adhesion scars in the peritoneum may stimulate the gut, and a scar in the brain may stimulate epilepsy. The surgical treatment of injuries at such sites is directed toward the prevention of gross scar formation, but if a scar has formed in such a manner as to interfere with function its revision may be necessary. One of the serious sequelae to extensive infarction of the myocardium is the formation of large scars and fibrous aneurysms that may contribute to cardiac failure because they are noncontractile and because they stretch somewhat at each systolic impulse. In the absence of sufficient experimental data one can only speculate whether the surgical revision of large cardiac scars may be possible in the future and whether any benefit might be expected by the patient.

Influence of Blood Supply

The influence of the blood supply on both the demolition (inflammatory phase) that must precede regeneration and on the subsequent regeneration (repair phase) is of paramount importance. The metabolic requirements of inflammation and of repair are somewhat different, but both require an adequate blood supply

for their proper expression. It is not to be expected that an ischemic tissue that would not support an adequate inflammatory response will support a healing reaction. The dry, mummified tissues of a gangrenous extremity will not repair. If left, they will undergo autoamputation due to a healing process much like that occurring under a scab at its junction with viable tissue. Many examples of the general requirement for an adequate circulation may be given. It is surgically desirable to immobilize wounds or fractures until some tensile strength is gained, since the delicate capillary arches and sprouts at the advancing margin of organization are extremely fragile and rupture with the slightest force, but immobilization that increases circulatory stasis in the area is an adverse influence on healing. Patients with poor cardiorespiratory function will not heal as quickly as those with normal cardiac function. The stubborn refusal of varicose ulcers and decubitus ulcers to heal is attributable to local circulatory deficiency. The healing of myocardial infarcts may be somewhat delayed because the patient is suffering from some degree of cardiac failure as a consequence of the infarct itself. The sloughing of tightly placed sutures is partly an inflammatory reaction, partly an ischemic process, a wound apposed under extreme tension will be ischemic and will not heal. Tissues altered by severe radiation injury become ischemic and, due in part to their inadequate blood supply, do not heal well.

Influence of the Injurious Agent

It is obvious that regeneration and repair will require the cessation of the action of the etiologic agents of injury. Bacterial toxins and other agents will inhibit or destroy regenerating cells. The tubercles of tuberculosis and simple abscesses become encapsulated with fibrous tissue only after the infections have been arrested by the body's defenses. On the other hand, in some inflammatory reactions the irritant agent is present in such low concentration that it is capable of damaging only some of the cells. The surviving cells are more resistant and their regenerate daughter cells are also more resistant. Repair may then occur

that in adult life the effect of age on cell regeneration in wound healing is largely dependent on such a general factor as the blood supply to the area

Cortisone and pituitary adrenocorticotrophic hormone affect the processes of inflammation and healing. They have approximately similar effects. It is a matter of clinical experience that the hormones in sufficient dosage may reduce a patient's resistance to infection or the spread of infection and retard connective tissue repair. The mechanisms fundamental to the effects on the inflammatory process are not clearly established, but there is incomplete evidence that the hormones reduce the vascular responses of inflammation, retard and make incomplete the cellular sequences of inflammation, inhibit the phagocytic ability of neutrophil leukocytes, and depress macrophages of hematogenous origin. At the same time healing is defective. Fibroblastic proliferation is reduced, perhaps due to arteriolar spasm and a diminution in the formation of new vessels, and connective tissue fiber formation is retarded. The wound does not fill with connective tissue nor does it gain tensile strength at the normal rate. The effects of cortisone and of ACTH on both the inflammatory defenses of the body and on the reparative proliferation of healing are of considerable therapeutic importance.

Many of the fundamental biologic phenomena related to healing are poorly understood. Some, such as nutrition, are under the general control of the surgeon. He can assure a high protein diet of good general nutritional quality or he can take steps to assure that excessive amounts of protein are not lost to the body from such lesions as extensive cutaneous burns or ulcerative colitis. Other fundamental phenomena such as age cannot be altered. There are, however, many obvious factors, often mechanical in nature, that have clinical application in the repair of wounds. The wound should be protected from the further action of the etiologic agent. Irritant and dead tissue should be removed. Bacteria and chemicals may be removed by irrigation and bacteria by drainage or chemotherapeutic agents, and the wound may be protected from the subsequent entry of infective organisms. The size of the tissue defect to be repaired can

be reduced to as small a gap as possible by surgical revision and approximation. Certain tissue defects may be remedied by grafts or prostheses. Steps may be taken to assure that the blood supply is not further embarrassed by swelling of tissues beneath tight casts or by too much tension on sutures or tissues. Immobilization will prevent mechanical rending of delicate, newly formed tissues. Temporary or permanent re-epithelization may be obtained by grafting. The general mental and physical well-being of the patient may be sought.

The surgical principles of the treatment of wounds and similar defects are vital to the successful handling of such lesions. However, they are based on relatively subsidiary facets of the general biology of regeneration and repair. It may be hoped that further investigation will lead to more fundamental knowledge that may have application in the surgical treatment of disease, for our present knowledge is often inadequate to the demands that may, from time to time, be made upon it.

SYSTEMIC REACTIONS

The previous sections have considered the local reactions of inflammation and of repair and also the influence of the constitution and pathologic status of the patient on these local reactions. It is now necessary to comment briefly on the effect of the local reactions on the patient.

The complex but relatively constant nature of physiologic processes under changing environmental conditions has been known for many years. The ability of the body to maintain a relatively constant internal milieu in the presence of a moderately variable external environment has been expressed by the concept of *homeostasis*. The normal person has a considerable ability to adjust to variations in physical activity, temperature changes, alterations in atmospheric pressure and composition, food intake, etc. In the course of these adjustments a variety of physiologic states may be altered, and, while the individual may or may not be consciously uncomfortable, a new adjustment is soon accomplished and a new physiologic homeostatic state is established. The new homeostatic state is not a pathologic one; it cannot be described as a state of disease.

It is a state of adaptation to a changed environment. On the other hand, it is obvious that many changes in the environment do cause *disease*. In these circumstances either no homeostasis is possible in the continued presence of the environmental change, or, if homeostasis is possible, it can only occur together with the severe limitation or practical abolition of some physiologic function. There is a failure to adapt to the new environment and at the same time retain the functions and abilities that are normal to a state of health. The border line between successful adaptation and its failure is often difficult to establish.

Many environmental alterations elicit precise and specific physiologic responses. In these cases the stimulus and the response are intimately, sometimes uniquely, related. However, many other stimulus-response relationships lack this precise or unique quality. They are less specific. For example, a wide variety of events may be effective in stimulating the pituitary-adrenal axis. This hormone-mediated system has complex pathways of expression, but the general response to its stimulation is nevertheless approximately constant. Selye has recognized the *general response to a variety of stimuli* as something common to many disease states and largely independent of the special features of any given disease. The general response is said to represent the sum total of the nonspecific effects remaining for observation after the recognition of the specific responses peculiar to the particular disease that is being observed. Since this general response is often partially mediated by the hormones of the pituitary-adrenal axis, it frequently resembles the numerous physiologic effects that are based upon this axis. Selye regards the general response as adaptive.

Fever

Fever is one of the most common symptoms of disease. The temperature of the body is controlled from the hypothalamus where two incompletely separated centers maintain a balance between heat disposal and heat production so that the oral temperature approximates 98.6° F. Diurnal variations of 0.5°-2° F are common, and healthy individuals may occa-

sionally have oral body temperatures of as low as 96° F or as high as 103° F.

The production of heat derives from muscular activity such as exercise or shivering and from exothermic metabolic activity. An increase in body temperature of about 1° F. requires about 40 calories. The disposal of heat depends on the ability to transfer heat to the environment. It is mediated chiefly through the evaporation of sweat, the dilatation of surface blood vessels, and the respired air. If environmental temperature and humidity are too high, heat disposal mechanisms become ineffective and a fever of exogenous origin may be imposed on the body. This occurs in heat exhaustion or in artificial fever-cabinet therapy. Dehydration, especially in infants, may interfere with heat loss by diminishing sweating and peripheral vasodilatation. Damage to the heat disposal center in the hypothalamus may leave the ordinary activity of the heat production area unbalanced so that a neurogenic fever develops. Strenuous muscle exercise, especially in children, may cause transient fever. Hyperthyroidism may produce a sufficient increase in metabolic rate and muscular activity to cause a mild fever. Rarely, manic states may result in marked hyperpyrexia. Damage to the nerve tracts arising from the temperature control areas of the hypothalamus may render a patient poikilothermic.

The commonest examples of fever occur in infections and in diseases or injuries in which there is present a considerable amount of necrotic material. Many viral and bacterial infections cause fever, but others may be so localized that no systemic effect is noted. Severe sterile crushing injuries, internal hemorrhage, pulmonary or myocardial infarction or sterile neoplasms may all cause fever. The agents which cause the fever in these different examples are not known. It is possible to obtain from bacteria or from sterile inflammatory exudates substances called *pyrogens* that will elevate the body temperature. These substances are diverse and poorly characterized chemically. Their injection into the blood is not followed immediately by fever, but a latent period of considerable duration intervenes before the onset of pyrexia. Their mode of action on the

hypothalamus and medulla oblongata is entirely obscure. It does not appear to be mediated by the pituitary-adrenal gland axis. It is not established that the pyrogens that can be obtained from bacteria are responsible for the pyrexia of infections; indeed, it can be shown in a given case of infection that the pyrogens of the invading organisms do not account for more than a small fraction of the pyrexia that occurs. It is observed that if an individual is in a cool environment the development of fever will require vasoconstriction, cessation of sweating, and an increase in metabolic rate. However, if the individual is in a warm environment, the same degree of fever will be attained without any increase in metabolism. Apparently the hypothalamic centers can respond to the same pyrogenic stimulus in a different, adaptive manner according to the environmental circumstances.

Excessively elevated body temperatures are deleterious. The central nervous system does not well tolerate oral temperatures above 104°-105° F, and temperatures of 110° F, or sometimes considerably less may produce permanent cerebral injury. Insomnia is common even with mild fevers. Hyperpyrexia can cause hyperventilation and a degree of respiratory alkalosis that may be significant in infants and in some adult patients. The increased cardiac output that is usually required in hyperpyrexia may precipitate cardiac failure in patients whose cardiac reserve is small. Prolonged hyperpyrexia with its associated anorexia and elevation in metabolic rate causes protein wastage of significant amount and may occasionally cause ketosis. Liver failure may be precipitated in the patient who has a small hepatic reserve. Dehydration may both result from and further enhance hyperpyrexia.

With the exception of a few diseases, there is little evidence that hyperpyrexia promotes in any way the recovery of a patient from the infection, trauma, or infarction that may be causing fever. Nevertheless, a patient should respond to an appropriate stimulus by manifesting fever; the failure to respond appropriately is of serious prognostic import. Certain drugs have the ability to reduce fevers. Almost nothing is known about the mechanism of action of such antipyretic substances.

Leukocytosis and Leukopenia

Leukocytosis is a generalized reaction on the part of the hematopoietic organs that releases abnormal numbers of leukocytes into the circulating blood. It occurs in response to many stimuli. The ordinary fluctuations in the numbers of blood leukocytes under physiologic stimulation by muscular exercise, or to exposure to cold or heat, or in response to the pathologic stimuli of epileptiform seizures, pain, trauma, and similar stimuli are moderate. They consist of a leukocytosis that comprises chiefly neutrophil leukocytes and, after a short interval, the development of eosinopenia and slight lymphopenia. These fluctuations are similar to those produced by the injection of Adrenalin in the presence of an intact pituitary-adrenal gland axis. The reactions appear to be nonspecific and may be regarded as adaptive. The mechanisms of this form of leukocytosis are only partially understood. Adrenaline is released by the effective stimulus; adrenaline causes the pituitary to release ACTH which then causes the secretion of glucocorticoids from the adrenal glands. These hormones elicit leukocytosis in the pattern described.

The leukocytosis of various pathologic states occasionally resembles that described above, but it frequently differs so much that certain diseases are recognized to have particular, specific hematologic patterns or deviations. Overwhelming infections and chemicals, or irradiation that damage the hematopoietic organs, may cause leukopenia. Pyogenic bacteria and some nonpyogenic bacteria and viruses cause a marked neutrophilic leukocytosis. Other bacterial infections, such as typhoid fever or brucellosis, and many virus infections are accompanied by neutropenia and, frequently, also by lymphocytosis. Active tuberculosis stimulates lymphocytosis and monocytosis without affecting the number of circulating neutrophil leukocytes. Infectious mononucleosis stimulates profoundly the development of a mononuclear (lymphocytic) leukocytosis. Diseases of allergic nature are often accompanied by a marked increase in the number of eosinophil leukocytes circulating in the blood.

Almost nothing is known about the mechanisms concerned in the development of path-

ologic leukocytosis or leukopenia. Stimulation of the pituitary-adrenal gland axis would seem to be only one of several mechanisms that might operate in certain diseases. Nevertheless, the ability of the patient to react appropriately with the proper hematopoietic response is of the greatest importance. As in the case of fever, a failure to respond appropriately is of serious prognostic import.

Protein Metabolism

One of the common systemic reactions to injury is an increase in protein catabolism and the development of a transient period of negative nitrogen balance. With the onset of pain, fever, or a toxic state there is anorexia and an increased metabolic rate; protein is consumed faster than it is synthesized, and an excess of nitrogenous material is excreted in the urine. Confinement in bed for a sufficient time may lead to an appreciable disuse atrophy of skeletal muscle and protein loss.

Foci of tissue destruction caused by trauma, infarction, infection, or hemorrhage represent local areas of protein breakdown that contribute to systemic protein catabolism. The amount of protein that may be lost from hemorrhage to the exterior of the body or into the gastrointestinal tract may be large.

The protein loss from the surface of the colon during an active phase of ulcerative colitis may rapidly waste the patient and make him a poor operative risk. The amount of fluid and protein that may be dissipated from the weeping surfaces of extensive skin burns has been aptly called white hemorrhage. It is commonly accompanied by an excessive systemic protein catabolism. The cachexia of a chronic infection, such as tuberculosis, or of carcinomatosis usually represents the combined effects of local tissue destruction, of anorexia, and of increased systemic protein catabolism.

The excessive systemic catabolism of protein that commonly follows upon injury to the organism does not appear to assist the recovery of the patient. Indeed, if it is severe, protein catabolism may make the patient cachectic, lower resistance, deplete wound healing, and render him more liable to secondary infection.

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Chapter 3

Surgical Bacteriology and Chemotherapy

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The purpose of this chapter is to give the student a general survey of the infections he is most likely to encounter in surgical practice. No attempt is made to give a complete list of all the infections which may occur or to discuss details of the bacteriologic technique or of the physiologic and other characters of the bacteria. The microbes will be discussed only in their relation to disease, and those characteristics only will be selected for notice which are essential for the understanding of the disease process and for a rational approach to treatment with either antiserums or modern chemotherapeutics. It is obviously impossible to make a strict division into surgical, medical, or other infections, but certain types are by their very nature treated by the surgeon, whereas others, although they may occur in a surgical patient, are by convention treated by the physician. No consideration will be given to the latter type of infection in this chapter.

Surgery owes a great debt to Lord Lister (1827-1912) who was the first to realize the importance of Pasteur's work to the field of surgery and who made an important contribution by applying the knowledge of his time to the operating room. Modern asepsis is only a further development of those first important concepts of antiseptics. Due consideration is given to the whole problem of sterilization and asepsis in the chapter on Surgical Technique.

HOST-PARASITE RELATIONSHIP

The host-parasite relationship involves certain structural and physiologic features of the bacteria which play a part in their adaptation to parasitism and the responses of the invaded host to the bacteria and their products. To evaluate the risks of infection in the operating field the surgeon should recognize the natural presence of certain kinds of bacteria in the various parts of the body, as well as possible outside sources. He must, therefore, be familiar with the means of preventing bacteria from entering wounds and of treating already established infections.

Pathogenicity is the broad term used to express the concerted action of a number of different characteristics which together enable microorganisms to cause disease. The type of disease is determined by the specific organism and its products, but the responses of the individual host give character of clinical importance to the disease. A variety of factors in the host are of great individual and collective importance and determine the degree of susceptibility and resistance to the infecting organism. Other features, like the site of localization or the organ or system invaded, are also of great significance. Certain bacteria have become adapted in certain localities and are accepted by the body without eliciting any reaction and cannot be completely eliminated from the region. Thus, the flora of the intestinal

tract, of the mucous membranes, and of the skin are examples of a well-balanced state between host and parasite, which is not only harmless but as in the case of the intestinal flora, may also be beneficial in synthesizing essential substances (vitamin K and some of the B complex). However, if some of these bacteria stray from their usual site into different tissues or organs, they may and do cause disease, although not all bacteria found in and about the body are capable of doing this. In order to cause infection, it is essential for a pathogenic organism to gain access to the body tissues and to be able to multiply there to a sufficient degree. The mechanism by which specific organisms cause disease varies, some cause their effects by powerful toxins and/or enzymes absorbed from a site of localization (diphtheria, tetanus); in some cases produced outside the body (botulism); others produce disease by active multiplication and local injury at the site or sites of invasion (Staphylococcus abscess, impetigo). A third group of bacteria may combine both modes of attack (gas gangrene), and yet others characteristically cause rapidly generalized infection and septicemias (typhoid, syphilis). Whether or not an infection remains localized or becomes generalized depends to some extent upon the bacterial species involved and upon the defense mechanism of the host, and various degrees and characters of infection may be seen in different cases due to the same species of bacteria (*Mycobacterium tuberculosis*, streptococci, *Bacillus anthracis*). Certain bacteria show selective localization in particular organs or tissues.

DEFENSE MECHANISM OF HOST

The response of the host to the microbe is manifold, depending in part on the particular species of microbe and in part on several factors in the host itself. We have said above that a prime factor in the establishment of an infection is the successful penetration by the bacteria or their products into the tissues of the host. The natural barriers which man possesses have to be broken down before access can be obtained. The normal self-sterilizing ability of healthy skin is a very successful line of defense, but even in the unbroken skin

there are weak spots which the enemy can attack. The hair follicles and the ducts of sebaceous glands represent such openings, and the bacteria, particularly *Staphylococcus*, may take advantage. Certain bacteria, such as *Pseudomonas* and *Leptospira*, are thought to be able to penetrate uninjured skin and mucous membranes. Wounds or other injuries to the skin provide a point of attack for a variety of bacteria. The portal of entry for the *respiratory tract* is the nose and nasopharynx. The mucous membranes, phagocytes, and the various secretions of the nose, pharynx, and the mouth provide a fairly effective barrier. Lysozyme, a bacteriolytic enzyme occurring in nasal secretions, tears, and other body secretions, may afford some protection against certain bacteria, and a certain degree of resistance may be provided by antibodies in the mucosal secretions. If any of these natural defenses are altered, the penetration of pathogenic bacteria is facilitated and occur. The direct penetration of air-borne particles to the pulmonary alveoli is difficult, but the inspiration of infected materials and secretions may cause serious trouble.

Infections of the *intestinal tract* occur after the bacteria have passed several defense hurdles. In the mouth the saliva has an inhibitory action for some. The highly acid gastric juice will destroy another number of organisms, but many do pass this barrier in food which may act as a buffer or pass rapidly through the stomach in a fluid medium, and still others (*tubercle bacillus*) can survive for a certain length of time in the gastric juice. The type of flora found in the small and large intestine is determined by the anatomic structure, the secretions of the mucosa and accessory glands, and the dietary habits of the individual. Any upset in the normal physiologic conditions may permit the establishment of a number of intestinal pathogens.

In the healthy adult female the acid pH developing in the secretion and the type of epithelium of the *vagina* prevent fairly effectively the implantation of particular bacteria, e.g., coliforms, and may also restrict to some extent localization of gonococci, which can more easily establish themselves in the *vagina* of prepuberty children.

If bacteria penetrate these outer barriers, a second line of defense is provided by the process of phagocytosis and by "natural antibodies."

Phagocytosis represents an attempt of the body to rid itself of extrinsic matter and is highly successful with nonpathogenic organisms which reach the blood stream or tissues. If pathogenic organisms are introduced into the blood stream, the same mechanism is mobilized by the host, but the clearing is less successful because the ability to resist phagocytosis is a fairly common characteristic of virulent bacteria in general, and with some bacterial species it is the chief attribute of their pathogenicity. Even if pathogenic bacteria are taken up by phagocytic cells and a number of them are destroyed, the majority will escape after injuring or even killing the cells and start their process of multiplication or adaptation in various tissues. Most bacteria are not directly introduced into the blood stream but rather into some tissues or organs, and the immediate reaction of the body defenses is to localize and fix the injurious agent, the first clinical manifestation of the interaction between host and parasite is local inflammation. Polymorphonuclear leukocytes are mobilized from the blood stream and accumulate locally. After this initial wave of polymorphonuclear cells, which may be very brief in some infections, a second type of cells, the mononuclear macrophages, make their appearance and increase in numbers. Depending upon the bacterial species and its virulence, this mechanism is more or less effective. If it proves insufficient to destroy the majority of organisms, an invasion of lymph channels and blood vessels takes place and generalized infection will follow.

This cellular clearing mechanism is augmented and supported by components of the serum. It has been known for a long time that the serums of man and different animal species contain *antibodies* against various bacteria and red blood cells of other species. These antibodies which can render bacteria more susceptible to phagocytosis or can agglutinate or lyse microbes are connected with the serum globulin. They are often called "natural" or "nonspecific" or "physiologic" antibodies because they are present in the

serum of man or animals who are not known to have experienced previous infection with a given organism. The origin of these "natural" antibodies is still a hypothetical question, though they are more common and more abundant in the course of epidemics and are attributed by many to subclinical infections, by others to common antigens. The hemagglutinins that divide human blood into different blood groups are special examples of natural antibodies. The exact role of the natural antibodies in resistance to infection is not clearly established. Contrary to these antibacterial antibodies whose origin and exact value are still debatable, so-called "naturally" occurring antitoxins are almost certainly due to subclinical infection with the respective toxin-producing organisms, and they do confer antitoxic immunity of a specific type if present in sufficient amounts. This has been proved for diphtheria, tetanus, and scarlet fever. Of greater importance as defensive forces against infection are the antibodies which are formed in answer to a specific stimulus with the object of removing and neutralizing the bacterial or toxic substances or both. These antibodies are named "specific" or "immune" antibodies and they too are connected with the serum globulins. These antibodies are specific for the particular bacteria or their antigenic components, for instance, antibody produced against *Pneumococcus* Type I will only combine with Type I polysaccharide and is not effective against Type II, or antibody against *Haemophilus* Type B will only act on Type B and not on Types A, F, etc. Immune antibodies show a rising titer in recovery from an infection and can be transferred from man to man or animal to man and furnish protection against specific infection.

The formation and specificity of antibodies constitute one of the most fascinating subjects in biology. The site of formation of antibodies is still a controversial subject and cannot be discussed in this connection. Up to the relatively recent introduction of chemical agents into the treatment of infections, antibodies either actively produced or mainly passively introduced were the most important weapon in the fight against pathogenic microorganisms. But antibodies have definite limitations, they are effective only against bacteria

outside of tissue cells and cannot affect microbes inside the host cells. Antibody response has another shortcoming, namely, that on occasion it is accompanied by allergy. It is a controversial question whether "bacterial" allergy functions always to the disadvantage of the host. However, it is rather generally accepted that at least in certain infections (streptococcal) it would benefit the host if the allergic response could be suppressed and only the immune response retained.

In addition to the established value of serum antibodies, other components of the serum, namely *complement* and probably the *properdin system*, play a role in the defensive mechanism. Their exact interrelation and their relative values and functions are not entirely clear. In spite of available chemotherapeutic agents, antitoxins or antibacterial serums are still a valuable aid in the treatment of disease, and antitoxin is still pre-eminent in diphtheria and is essential in tetanus. Apart from the importance of antibodies as defensive forces, their appearance in response to infectious agents is of great significance in the diagnosis of a number of infectious diseases, as illustrated by the Widal reaction, brucella agglutination, and a series of other similar serologic tests based upon the demonstration of antibodies against specific microorganisms. The studies on the mechanism of antigen and antibody reactions have been tremendously stimulating and useful for decades and have led to a series of new concepts in immunity. The applications of highly specific antigen-antibody reactions have also resulted in the recognition of a variety of components in the bacterial cell which in turn has led to more exact identification of bacterial species, to new tools in the field of epidemiology, and to better appreciation of the processes of infection. An understanding of immunologic principles is of fundamental importance in every branch of medicine for diagnostic, preventive, and curative purposes.

The interaction between host and parasite in all its complexity determines the clinical course of an infection as well as the pathology of the lesions. The clinical signs and symptoms and the surgical treatment of various infectious processes are discussed in the appropriate chapters of this textbook. The de-

scription of tissue responses to bacterial invasion in general (inflammation, necrosis, fibrosis, etc.) or to certain specific microorganisms, e.g., tubercle bacillus, *Treponema pallidum*, fungi, is subject matter for pathology, and their discussion is omitted with the assumption that the student is thoroughly familiar with all the aspects. But, although redundant perhaps, a brief definition of terms which are commonly used in connection with infections is appended to prevent confusion of interpretation.

Abscess is a localized necrosis with a collection of pus confined by a wall of granulation tissue and may be acute or chronic. The furuncle or boil is a common form of abscess of the skin caused by *Staphylococcus pyogenes* which usually gains entrance through the hair follicle.

Cellulitis is a diffuse, often rapidly spreading inflammation of the subcutaneous tissues, frequently caused by the hemolytic streptococcus and involving the fascia. A serosanguineous exudate in which leukocytes are sparse is rather distinctive.

Bacteremia means the simple presence of bacteria in the blood stream, which may be transitory. This is a relatively frequent occurrence after tooth extraction or tonsillectomy without causing any serious symptoms.

Septicemia is a bacteremia with severe generalized symptoms, often accompanied by valvular lesions and other localizations.

Septicopyemia indicates a septicemia with abscess formation in various organs, usually initiated by infected emboli. These abscesses are known as "pyemic abscesses."

Toxemia signifies that the clinical signs and symptoms are due to a toxin rather than directly to bacterial multiplication. The best example of a toxemia is tetanus.

GENERAL ASPECTS OF THE TREATMENT OF INFECTIONS

Historic Introduction

The development in the treatment of infectious diseases can be divided roughly into three phases. The landmark in the first phase, which might be termed the *antitoxin-antisera phase*, was set by Roux and Martin (1894),

who immunized horses with diphtheria toxins and produced antitoxin. The extension and elaboration of this principle to other infections have been of immense value in the prevention and treatment of bacterial diseases. Behring and Wernicke (1892) actually treated for the first time a patient with diphtheria with serum from immunized animals. This marks the beginning of specific serum treatment. Infections in which treatment with antitoxin is still of prime importance will be discussed in the special part.

The second phase in the treatment of infections started with Ehrlich's work on chemotherapeutic agents (1904-1915), crowned by the discovery of "Compound 606" (1910), which as Salvarsan and all its related compounds has been the most effective weapon against syphilis for almost 35 years. The next important step in this phase was made with the introduction of Prontosil by Domagk (1935). The dramatic effect of Prontosil in streptococcal infections, particularly erysipelas, led to intensive investigations, and it was soon discovered by some French workers that the active principle of Prontosil was sulfanilamide. This compound was soon followed by more active and less toxic derivatives such as sulfapyridine, sulfathiazole, sulfadiazine, sulfamerazine, Gantrisin, and many others.

The usefulness of the sulfa drugs in surgery is somewhat limited by the fact that their activity is considerably decreased by the presence of pus and tissue debris in wounds. On the other hand, some sulfa drugs penetrate more freely into the spinal fluid than do antibiotics. They are therefore considered the drugs of choice in mild cases of meningococcal meningitis and are used in addition to an appropriate antibiotic in the treatment of other types of bacterial meningitis. Sulfa drugs are also employed in preference to antibiotics in the treatment of bacillary dysentery and some urinary tract infections.

The third and present phase in the treatment of infections, which logically is a continuation and part of the chemotherapeutic era, began with the discovery of penicillin by Fleming (1929) and its development for clinical application by Florey and Chain and many others (1910). This period of antibiotics, although of such recent origin, has

had the most stupendous effect on the whole field of medicine and allied sciences. The ever-increasing number of available antibiotic agents, all rather specific in their action, has put added importance on accurate diagnosis of bacterial infections for the proper choice and use of the most effective agent. It should be recognized and appreciated that our knowledge in this field is still limited and based to a large extent on empirical findings.

General Introduction

The exact mechanism by which the antibiotics exert their effect upon the microbial cells is not fully understood, although a certain amount of information is available. Our concepts and practices are therefore prone to changes, and statements as to choice of drugs, dosage, and route of application are only valid in the light of our present knowledge and experience. The choice of a particular antibiotic or of a combination of an antibiotic with other chemotherapeutic agents is dependent upon a number of factors such as the spectrum and relative toxicity, the available form for administration, concentrations obtainable, rate of diffusion, and excretion and stability. Other considerations are the type and severity of the infection and possible allergic reactions of the host.

The *antimicrobial spectrum* of an antibiotic refers to its relative selective activity against specific microorganisms. It has been realized recently that this expresses a quantitative rather than a qualitative characteristic. In the test tube most bacteria can be inhibited by practically all antibiotics, provided high enough concentrations are used. However, in the treatment of infections there are limits of obtainable concentrations in the body, and a definition of spectrum in this sense is still valid. As important, or even more important, from the practical point of view, are the variations in sensitivity from strain to strain within the same species or group of organisms. These variations are more pronounced in some bacterial species than in others. Antibiotics are either primarily *bacteriostatic* or *bactericidal* in their action although either characteristic is to some extent dependent upon the dosage and the microbe involved. It may be necessary to

use higher dosages and longer periods of treatment if antibiotics with mainly bacteriostatic activity are used in order to give the defense mechanisms of the body a better opportunity to eradicate the infection completely

Correlation Between in Vitro Findings and Clinical Effects

Sensitivity testing of isolated microorganisms has become a routine procedure in a diagnostic laboratory. It may well be asked how reliable and useful are such results with regard to the expected treatment response. To the alert reader the answer should already be obvious. By posing such a question it is implied that the correlation between the in vitro findings and the clinical effects is most certainly not absolute. The degree of correlation depends to some extent on the methods used for sensitivity testing, the microorganisms involved, the localization of the infection, and on a number of other factors in the host. Lacking a better foundation for the choice of a particular antibiotic, however, these tests have proved their practical value. It can be stated that, by and large, the effects in the body are better than those apparently indicated by the in vitro test, since humoral and cellular defensive forces enhance and aid the activity of the antibiotic. On the other hand, if the lesions are old, fibrotic, and poorly vascularized, the effect in the body may be considerably less than might be expected from in vitro sensitivity tests, since only small amounts of the antibiotic will actually reach the microorganisms. Furthermore, the designation of an organism as sensitive or resistant depends upon the methods and media used in different laboratories, and until standard methods and materials for testing of antibiotic sensitivities are developed, the results are not comparable. For all these and other reasons, the in vitro results should be used as a *qualitative* guide only for the choice and dosage of an antibiotic.

Development of Resistance

One of the most disturbing factors in antibiotic therapy is the development of *resistant strains*. This phenomenon has been observed

and studied by a great many investigators, and attempts have been made to counteract this limitation in the practical use of antibiotics. It is also one of the reasons for the constant search and development of new antibiotics.

Regarding the development of resistance of bacteria to antibiotics, there are two theories which have been proposed and which may in fact operate simultaneously. The *first theory* states that this phenomenon is due to spontaneous mutations which occur whether or not the antibiotic is present. When the antibiotic is present, it acts selectively to favor the survival of these resistant mutants. The significance of a mutation in every ninth or tenth generation becomes apparent only when the size of bacterial populations is considered, i.e., one ml of broth culture 24 hours old may contain up to 100,000,000 bacterial cells representing 30 generations. The number of bacteria present in the host at any time may be a very large multiple of this figure. Therefore enough mutants may appear to allow their survival and multiplication unless other factors such as body defenses can cope with them. An *alternate theory* states that resistance results from adaptation of organisms to sublethal concentrations of antibiotics. Eagle and his associates (1952) believe that the theories are not mutually exclusive. It is possible that slightly increased resistance following exposure to low concentrations of drugs may result from adaptation, while the highly resistant cells represent a spontaneous mutation.

However it develops, the resistance of bacteria, and in particular of staphylococci, to antibiotics is becoming a matter of great concern, particularly with respect to hospital-acquired infections. During the years 1946-1951, the number of hospital strains of staphylococci resistant to penicillin grew from 14% to about 70%. There has been no significant increase since then. During the same period, streptococci, pneumococci, and gonococci showed no such tendency. Staphylococci have shown a rapid development of resistance to streptomycin, chlortetracycline, and oxytetracycline but less to chloramphenicol due to its more restricted use. Since 25% or more of hospital staphylococci may be resistant to five or more antibiotics, the seriousness of the problem is immediately apparent. Resistant

staphylococci are not restricted to this continent but have been reported from all over the world where antibiotics of one type or another have been used for some time.

Various attempts have been made to counteract or circumvent the emergence of such resistant strains. One such approach has been to continue the search for new antibiotics effective against the multiple resistant strains. This search of course will have to be continued, since in time the staphylococci will become resistant to these new antibiotics as well. Another solution has been proposed by several authors, namely, to restrict the use of antibiotics to cases where there is a definite indication for their administration. As general restrictions of this nature are difficult to maintain, some hospitals have adopted a definite policy with regard to the use of one or two antibiotics. These antibiotics, still effective against staphylococci, are not used unless absolutely indicated in a particular case. The rationale of such decisions is based on the results of investigations which have clearly shown a marked difference in the incidence of resistant strains between hospital and outside patients. It is an established fact that cross-infections with resistant strains are common occurrences and responsible for the high incidence in hospitals. Spread occurs from patient to patient or via the hospital personnel acting as carriers. Reports from institutions where this policy has been carried out and maintained indicate that it is successful in preventing the emergence of resistant strains with respect to the chosen antibiotic or antibiotics.

In setting aside an antibiotic for restricted use, another problem must be kept in mind, the development of cross-resistance of an organism to more than one antibiotic. For example, in the tetracycline group of antibiotics, when an organism becomes resistant to, let us say, oxytetracycline, in most instances it also becomes resistant to chlortetracycline and to tetracycline itself although it may never have been exposed to the latter two antibiotics. The reason for this, of course, is the very close chemical similarity of the three drugs. Other instances of cross resistance have been observed and will be mentioned later. Therefore, if at all possible, the new antibiotic

should be unrelated chemically to the antibiotics already in use. Other attempts to prevent emergence of resistant mutants have been made, such as combined treatment.

The Use of Antibiotic Combinations

There has been an increasing tendency over the past few years to use combinations of antibiotics in the treatment of infectious processes. Many claims have been made for the superiority of mixtures of two or more antibiotics over any one of the drugs used alone, and the increasing number of commercially premixed combinations has added considerably to their use. However, the choice of various combinations of antibiotics and chemotherapeutic agents must follow certain principles in order to achieve the objectives, and all too frequently these principles are ignored.

The studies of Jawetz and Gunnison (1955) and other investigators have shown that combinations of antibiotics may produce various effects both in vitro and in vivo. In brief, these effects may be (1) synergism, where the combined antimicrobial effect is much greater than that of mere addition, (2) additive, where the antibacterial activity of the combination is greater than that of either drug alone but neither more nor less than that obtained by doubling the concentration of either, (3) indifference, where there is neither increase nor decrease in action, and (4) antagonism, an effect less than that achieved with the more effective of the two drugs acting alone.

The evidence from in vitro experiments and from animal experiments has been summarized by Jawetz and Gunnison, who have listed currently used antibiotics in two groups: (1) primarily bactericidal, containing penicillin, streptomycin, bacitracin, neomycin, and (2) primarily bacteriostatic, containing chlortetracycline, oxytetracycline, tetracycline, chloramphenicol, erythromycin, carbomycin, and spiramycin. Mixtures of two or more members of Group I antibiotics frequently show synergism, sometimes indifference, but never antagonism. Members of Group II are neither synergistic nor antagonistic to each other, but simple additive effects are frequently observed. When

an antibiotic from Group I is combined with an antibiotic from Group II, the effect may be synergism, indifference, or antagonism. It is impossible to predict whether synergism or antagonism will occur with a given drug combination, since the outcome depends upon such complex factors as the relative concentration of the two antibiotics and the susceptibility or resistance of the bacterium to each individual drug. The result of a specific drug combination acting on a particular strain of the organism can only be determined by actual experiment and may not always be the same *in vivo* as *in vitro*.

For clinical purposes, *synergism* means a therapeutic effect obtained by the use of two or more drugs which could not be obtained with either drug alone in full therapeutic doses. On this basis there are very few types of infection where true synergism has been demonstrated clinically. These are in the treatment of enterococcic endocarditis with a combination of penicillin and streptomycin, of brucellosis with streptomycin and one of the tetracyclines or chloramphenicol, of certain staphylococcal infections with a combination of penicillin and chlortetracycline or streptomycin and oxytetracycline, and finally in the treatment of tuberculosis with paired combinations of streptomycin, isonicotinic acid hydrazide, and para-aminosalicylic acid (Dowling, 1957).

True clinical *antagonism* between paired antibiotics has been observed less frequently. Very definite ratios of two antibiotics must be present for antagonism to occur; for this reason such an effect is not often encountered clinically since the doses used are much larger than minimal effective doses. The best clinical example of antagonism is that reported by Lepper and Dowling (1951) when penicillin and chlortetracycline were used together in pneumococcic meningitis. Antagonism, however, may occur when the concentration of one or both antibiotics is at or below the minimal effective concentration. This has been demonstrated by Strom (1955) who, in the treatment of patients with scarlet fever, used either penicillin or chlortetracycline alone or the two combined. In summary, clinical evidence favoring the use of combinations of

antibiotics for the express purpose of obtaining better results than those obtained by using the most effective antibiotic alone can be justified only in the instances quoted above and in certain mixed infections.

Another reason given for the use of combinations is to obtain a good therapeutic result with nontoxic doses of two antibiotics which would otherwise require a toxic dose of either alone. Such combinations have been studied, particularly in the use of streptomycin and dihydrostreptomycin. It would appear from the recent studies of Wier and his associates (1956) that streptomycin alone is less likely to cause deafness than the use of the combination.

Still another reason for the use of antibiotic combinations is said to be the prevention of the appearance of resistant strains of the infecting organism. This theory has been applied practically for some years in the treatment of tuberculosis. Such combinations have also been utilized particularly in the treatment of staphylococcal infections. However, the usefulness of such a procedure is of limited value since the organism must be sensitive to both antibiotics before treatment is started, and in order to prevent the appearance of resistant strains, the two drugs should always be administered together in a given hospital and preferably throughout the entire country. The latter concept is hardly practicable, and it would appear that the prime use of such a scheme to delay the appearance of resistant strains is in the treatment of tuberculosis.

Combinations of antibiotics have been used in an attempt to combat the mixed flora of the gastrointestinal tract, thereby preventing the peritonitis that follows soilage from the intestine, or to treat mixed infections of the respiratory tract, the skin, and the urinary tract. Any one of the tetracyclines or chloramphenicol will prove as effective in lowering the bacterial count of the intestine as any of the recommended combinations of antibiotics.

There is also good evidence that the simultaneous use of polymyxin B and neomycin is more effective than either antibiotic alone and perhaps more effective than the tetracyclines or chloramphenicol. This combination has the

added advantage that hypersensitivity is unlikely to develop since absorption from the gastrointestinal tract may be kept to the minimum by proper dosage. In the actual treatment of peritonitis, the tetracyclines have been shown to be equal and perhaps superior to the combination of penicillin and streptomycin. If the latter are to be used, however, they should be administered separately, thus allowing a greater flexibility of dosage and route of administration than mixtures already prepared and dispensed.

Best results are obtained in the treatment of chronic bronchitis and bronchiectasis with one of the tetracyclines or chloramphenicol, and there is no evidence to date that these results can be improved with combinations of antibiotics. Lung abscess can best be treated with penicillin alone or a tetracycline alone. The use of nystatin in conjunction with a tetracycline is advocated by many to reduce the numbers of *Candida albicans* in the presence of large numbers of *C. albicans* in the intestinal tract, however, does not necessarily result in symptoms. If one is successful in suppressing *C. albicans*, some other organism which is not affected by the antibiotic used, such as *Proteus vulgaris* or resistant staphylococci, may become the predominant microorganism. It is also possible that the extensive use of nystatin in a hospital will cause the appearance of nystatin-resistant strains of *Candida* so that the drug will be useless when it is needed for therapeutic purposes.

The use of combinations of antibiotics has been recommended for the treatment of seriously ill patients before a bacteriologic diagnosis can be made. This practice is permissible if such therapy be given to those patients in whom the clinical diagnosis of an infection is reasonably certain and if the antibiotics used are selected to combat that particular infection. However, in the majority of cases, treatment prior to diagnosis is not necessary and in fact very often delays proper diagnosis and makes it more difficult. Also, under these circumstances, hypersensitivity to an antibiotic may occur, and if the proper combinations are not used, resistant forms of the organism may appear. If antibiotic therapy must

be used, an accurate clinical diagnosis is imperative, and this should be supported as quickly as possible by a dependable bacteriologic diagnosis together with in vitro sensitivity tests on the causative organism. In the rare cases where combined treatment is essential, one should bear in mind the possibility of antagonism when an antibiotic from Group I is combined with an antibiotic from Group II. In all cases each antibiotic should be given in full therapeutic doses.

Steroids and Antibiotics

There is no experimental or clinical evidence to suggest that cortisone or related compounds have any antimicrobial action. However, when used judiciously with antibiotics, they may at times be helpful and even lifesaving. Steroids probably have no place in the treatment of mild or moderately severe infections of bacterial origin. But evidence at the moment would indicate that a combination of steroids and antibiotics may be of considerable value in the treatment of overwhelming infections such as meningococemia, in the treatment of selected cases of tuberculous meningitis, and in combating the toxemia and debility encountered in the early stages of severe typhoid fever and brucellosis. There is also good clinical evidence that the use of steroids together with antitoxin and penicillin is of real value in the treatment of patients suffering with severe tetanus. These, according to Herrell (1957), are the only bacterial diseases in which the steroids have proved of value, and their usefulness is not in any way connected with antibacterial activity. It should be mentioned that we know very little about the possible effect of cortisone and related compounds on phagocytosis and on antibody production. Experimental data indicate that while phagocytosis may not be interfered with, the ability of cells of cortisone-treated animals to destroy the ingested organisms is greatly depressed. Other experimental evidence suggests that cortisone interferes with antibody production.

Complications of Antibiotic Treatment

A number of complications are frequently encountered following the use of antibiotics

either alone or in combination. These are the appearance of secondary infections or superinfections due to organisms resistant to the antibiotic in use, the development of moniliasis, either local or general in nature, and the emergence of antibiotic-resistant strains of bacteria. One of the major problems resulting from the widespread use of antibiotics is the occurrence during or after treatment of secondary infections or superinfections caused either by bacteria resistant to the drug or by other microorganisms which, being opportunists, are able to take over due to the considerable reduction of the normal bacterial flora resulting from therapy. For example, massive doses of penicillin not only eliminate pneumococci in cases of pneumonia but also the majority of gram-positive bacteria from the upper respiratory tract. In the laboratory we can almost invariably tell when the patient has received penicillin by the predominance of coliform bacteria in the sputum. This disturbance in the balance of nature may sometimes result in definite symptoms when, for instance, the replacing organism is *Pseudomonas aeruginosa* or *Proteus vulgaris*.

One of the serious complications of antibiotic therapy is *staphylococcal enteritis*, which may occur following the use of any antibiotic but particularly after broad-spectrum antibiotics. In this case, staphylococci resistant to the antibiotic become the predominant intestinal organisms and, due to their toxins, are extremely irritating to the gastrointestinal mucosa. When the staphylococci are eliminated by the administration of an antibiotic to which they are sensitive, the symptoms of the enteritis disappear. Changes in bacterial flora of the respiratory or gastrointestinal tract which occur during antibiotic treatment do not always produce symptoms. If the replacing organism is a pathogen or potential pathogen, however, the result may be of great importance to the patient's well-being and also to the physician in determining his method of treatment.

One of the sequelae of antibiotic therapy is the outgrowth of the yeastlike fungus *Candida albicans*, normally a saprophytic organism present in small numbers in the upper respiratory tract or in the intestinal tract of some 15-20% of normal individuals. After the elimination of normal respiratory or in-

testinal flora, *C. albicans* becomes pathogenic and tends to invade the epithelium. In many cases rather annoying symptoms are produced which are characterized by moderate to severe diarrhea with perianal pruritus. In occasional cases, generalized and fatal systemic infections due to *C. albicans* have been described following antibiotic therapy. It is possible with the use of nystatin to eliminate *C. albicans* from the intestinal tract with almost complete relief of symptoms. However, all too frequently when the antibiotic is withdrawn *C. albicans* again appears in large numbers.



Fig 6—Pseudomembranous enterocolitis due to antibiotic-resistant *Staphylococcus pyogenes*

Most antibiotics in clinical use today have been so purified that the toxic side effects such as nausea, vomiting, and diarrhea have been reduced to an absolute minimum. These side effects can readily be arrested by withdrawing the antibiotic responsible for them. However, allergic or hypersensitivity reactions are commonly seen side effects of antibiotic therapy, due to the fact that practically all reactions to penicillin are of the allergic type and that

penicillin is the most frequently used antibiotic. Less serious hypersensitivity reactions are dermatitis, conjunctivitis, and stomatitis. These may occur in a sensitive individual from either local or systemic application. A wide variety of skin lesions may be seen following the use of antibiotics, chiefly penicillin and streptomycin. A serum sickness type of reaction consisting of fever and skin rash, with pain and swelling of multiple joints, is seen not uncommonly after administration of penicillin. Anaphylactic shock is the most serious of the penicillin reactions and may occasionally be fatal. Until recently the occurrence of this type of reaction was considered extremely rare. Lately, however, reports on fatal and nonfatal anaphylactic reactions to penicillin have been appearing with increasing frequency, no doubt because of the size of the population that is today sensitized to this antibiotic. Certain of the antibiotics, particularly neomycin and bacitracin, have a very low sensitizing index and as a general rule are used only for local applications. When administered systemically the possibility of a drug reaction on subsequent use is very low.

Indications for Antibiotic Therapy

There are at present 18 different antibiotics in common clinical use with others at the stage of experimental animal or clinical trial. If treatment is to be most effective, the appropriate antibiotic must be chosen quickly enough to prevent tissue damage or irreversible physiologic changes.

In the early days of the antibiotic era, the initiation of drug therapy without a bacteriologic diagnosis was perhaps permissible when only two or three antimicrobial agents were available. With the large number of drugs now available it is no longer feasible to follow this practice, particularly since some of the drugs though highly active are rather limited in scope. Ideally, of course, a bacteriologic diagnosis should be obtained before the initiation of antibiotic therapy. In the great majority of instances this policy is possible, but in extremely acute infections a good deal of clinical acumen is required in order to make shrewd bacteriologic inferences at the bedside. Therapy may be started but not until

adequate and proper specimens have been taken for bacteriologic diagnosis.

Antibiotics may be indicated for either the prevention or treatment of infections. The treatment of individual bacterial infections will be dealt with in a subsequent section. Prevention of streptococcal respiratory infections in persons who have had rheumatic fever or prevention of the bacteremias which may follow oral manipulations or surgery in patients with past or present rheumatic fever is considered desirable. Patients undergoing abdominal surgery involving possible perforation of the intestine should receive antibiotics preoperatively. Patients with diabetes, nephritis, or other complicating diseases which make them susceptible to infections should be prophylactically treated with antibiotics prior to surgery. Surgical procedures which must be carried out on infected or potentially infected sites should also be preceded by antibiotic prophylaxis. It should, however, be emphasized very strongly that all the modern "miracle" drugs do not permit the surgeon the slightest deviation from accepted standards of aseptic technique. They do make possible, in many instances, successful operations under conditions which formerly were followed by a high mortality rate. Skillful aseptic surgery remains of the utmost importance, and proper attention to nutrition, hydration, and all other supportive measures is most essential. The common practice of carrying out all surgical procedures under an "umbrella" of antibiotics is to be deplored.

A number of factors may influence the effectiveness of antibiotics *in vivo*, and these must be taken into consideration when determining the antibiotic to be used and the route of administration. One of the most important considerations is the concentration of antibiotic which can be expected in the environment of the organism. In the case of septicemia or of bacteremia, this is no problem. In general, the antibiotics available today are freely distributed through the body, but each has its own peculiarities which will be described. In fibrotic or necrotic lesions which are avascular or relatively so, it is very difficult for the antibiotic to diffuse to the center of the lesion in adequate concentrations. In such cases, surgical drainage combined with

local instillation of the antibiotic is the preferred treatment. For the treatment of systemic infections, the concentration of the chosen antibiotic should be maintained at least during successive recurring periods at 5-10 times the minimal inhibitory amount required *in vitro* for the particular organism

ANTIBIOTICS PRESENTLY USED

Penicillin diffuses after parenteral introduction into most tissues but does not penetrate freely into the bone marrow, the joints, body cavities, the intact central nervous tissue and not very readily into the spinal fluid, even in cases of meningitis, unless very high doses are used. In cases of empyema or other localized infections which are accessible to topical treatment, a combination of topical and systemic treatment is advisable. Antibiotics, in the form of aerosols either alone or in combination with intramuscular injections, may be the treatment of choice in certain types of upper respiratory infections. The large percentage of resistant strains of *Staphylococcus pyogenes* has put severe limitations on the usefulness of this antibiotic for the treatment of these infections.

Oral penicillin has proved to be effective against sensitive organisms, particularly for prophylaxis in patients with rheumatic fever. High dosage of oral penicillin has been necessitated by excessive destruction of the drug by acid in the gastrointestinal tract. Some time ago phenoxymethyl penicillin, an analogue of penicillin G, was described and was further investigated because of its unusual acid stability. This form of penicillin, which has come to be known as penicillin V, apparently remains insoluble in the acid pH of the stomach but is readily soluble and readily absorbed upon reaching the alkaline environment of the small intestine. Higher serum levels are obtained following oral administration with penicillin V than with an equal dosage of penicillin G. The absorption of benzathine penicillin V is much less affected by the simultaneous intake of food than is that of benzathine penicillin G. Hence penicillin V may be given with less regard to feeding and is in fact preferably given after meals. The administration of probenecid enhances serum levels of penicillin V in a


manner similar to its effect with penicillin G. It is now generally considered that penicillin V administered orally is more efficient because of its stability to gastric acids and because higher serum levels are obtained.

Streptomycin, like penicillin, is readily absorbed in the blood stream after intramuscular injection and is distributed to the various tissues. On the whole, so far as dosage and route of administration are concerned, very much the same rules apply as for penicillin. Streptomycin is active chiefly against gram-negative organisms, but some gram-positive species are also sensitive. The particular value of streptomycin is its activity against *Mycobacterium tuberculosis* and its synergistic effect with penicillin in the treatment of subacute bacterial endocarditis due to enterococci. If used over prolonged periods, damage to the auditory nerve may occur in a fairly large number of patients. Development of microbial resistance is a frequent phenomenon and may create treatment problems.

Chlortetracycline, oxytetracycline, and tetracycline are best discussed as a group as they show many common characteristics. All three have a broad antibacterial spectrum demonstrated by their activity against both gram-positive and gram-negative organisms and their activity against certain rickettsiae and large viruses. All three are rapidly absorbed when taken by mouth and are excreted in large quantities in the urine. All three are free from serious toxic properties, although nausea, vomiting, and diarrhea frequently interfere with the usage of the tetracyclines. Strains developing resistance to one agent usually show a marked cross-resistance to the other two analogues. The choice in any given infection will depend upon the sensitivity of the particular organism or organisms and upon possible sensitivities of the patient to one or the other of the antibiotics.

Chloromycetin (chloramphenicol) also belongs to the group of broad-spectrum antibiotics and, although chemically distinct, resembles in its activity and other properties the tetracycline derivatives. Gastrointestinal disturbances during Chloromycetin treatment have been reported but are not a major concern. Some consideration should be given to

TABLE I
THE PRACTICAL ANTIMICROBIAL SPECTRUM OF PRESENT ANTIBIOTICS

FUNGI	ACID-FAST BACILLI	GRAM- NEGATIVE BACTERIA	GRAM- POSITIVE BACTERIA	SPIRO- CHETES	RICKETTSIAE	VIRUSES		PROTOZOA
						LARGE	SMALL	
			Penicillin					
		Streptomycin						
		The Three Tetracyclines						
		Chloramphenicol						
		Neomycin						
		Polymyxin						
		Bacitracin						
		Tyrothricin						
Nystatin		Erythromycin						
		Carbomycin				Carbomycin		
	Cycloserine	Novobiocin						
Amphotericin B		Oleandomycin						
		Spiramycin						
		Kanamycin						

possible suppressive effects on the hematopoietic system by this drug if used over long periods.

Tyrothricin (combination of gramicidin and tyrocidine) is too toxic for systemic use but has certain advantages for topical application. It is effective mainly against gram-positive organisms.

Bacitracin has a spectrum similar to penicillin and has the advantage of not being inhibited by penicillinase, which may be an important point in the treatment of mixed infections due to gram-negative and gram-positive flora; it seems devoid of sensitizing properties. Its main value is in local application (surgical wound infections, furuncles, carbun-

cles, abscesses, and pyogenic dermatoses). The preparations now available for systemic treatment if given in the recommended dosage (20,000-25,000 units every 6-8 hours i.m.) and with a high fluid intake have no serious side effects and represent a valuable alternative to penicillin or other antibiotics, particularly in the treatment of resistant staphylococcal infections.

Polymyxin is active chiefly against gram-negative organisms. At the present time it is the most effective agent against *Pseudomonas aeruginosa*. Topical applications of polymyxin combined with bacitracin and neomycin are considered the treatment of choice in mixed bacterial infections of burns and wounds.

Parenteral use of polymyxin is limited to serious systemic pseudomonas infections, and proper attention must be given to its potential toxicity.

Neomycin has an antibacterial spectrum similar to that of polymyxin, although the latter is more effective against most pseudomonas strains and the former against proteus. Neomycin is restricted almost entirely to local treatment and is favored by many for pre-operative intestinal preparation, as little or no absorption takes place. Because of its marked toxicity on parenteral dosage, this form of treatment is used only under special circumstances.

Erythromycin has an activity against gram-positive organisms similar to that of penicillin, and its greatest use is in the treatment of infections due to staphylococci, streptococci, and pneumococci. Gram-positive organisms resistant to all other antibiotics still may be attacked by this drug, and patients who are sensitive to other available antibiotics may be treated successfully with erythromycin. While it is somewhat less active than the tetracyclines against gram-negative organisms, it is a useful antibiotic to hold in reserve for the treatment of infections due to gram-negative cocci, particularly the gonococcus. It has some degree of activity against the large viruses, the rickettsiae, and some of the protozoa, although tetracyclines are more useful in these particular types of infections. One of the advantages of using erythromycin is that, due to its relative inactivity against gram-negative rods, it is perhaps less likely to alter the intestinal flora and to lead to complicating superinfections with *Candida*.

No cross-resistance has been demonstrated between erythromycin and penicillin, streptomycin, the tetracyclines, or chloramphenicol. However, cross-resistance occurs between erythromycin, carbomycin, and spiramycin. In man, toxic symptoms are not observed as a rule in the recommended dose of 300-500 mg q.6.h. Higher doses will cause gastric distress. Following oral administration erythromycin is readily absorbed, with a peak blood concentration occurring 2-4 hours subsequent to the administration of a single dose. Like penicillin, erythromycin does not readily pass

through the blood-brain barrier in the absence of inflammation. The drug will diffuse through the placenta. It diffuses readily into ascitic and pleural fluids. In the presence of normal liver function it is concentrated in the liver and excreted in the bile in active form. It is excreted constantly in the urine. Since erythromycin unprotected is partially destroyed in the upper gastrointestinal tract, specially coated tablets are used which protect the drug against gastric acids.

Carbomycin is principally active against gram-positive bacteria but has little or no effect against gram-negative bacteria. In addition it possesses some activity against rickettsiae and the large viruses. Its activity against gram-positive organisms is somewhat less than that of penicillin. Since no cross-resistance develops between carbomycin and penicillin, streptomycin, the tetracyclines, and chloramphenicol, this drug may be used in the treatment of infections due to gram-positive cocci resistant to these other antibiotics. However, cross-resistance develops between carbomycin, erythromycin, and spiramycin. Favorable clinical response has been obtained with carbomycin in acute infections caused by gram-positive cocci, but the results were less striking than those which would have been expected from penicillin. It may be said then that carbomycin is a poor substitute for penicillin in the treatment of infections due to gram-positive organisms, except in those cases where the organism is resistant to a number of other antibiotics. It would appear under such circumstances that erythromycin will produce a better result than carbomycin.

Oleandomycin, although only recently released for use, is not a new antibiotic. It appears to belong to the erythromycin-carbomycin-spiramycin group of antibiotics. Oleandomycin inhibits gram-positive organisms in vitro in a concentration of three gammas/ml. or less. It is active mainly against the gram-positive organisms and members of the *Neisseria*, *Hemophilus*, and *Brucella* groups, the rickettsiae, and to a lesser extent against the larger viruses and protozoa. There does not appear to be any cross-resistance with the common antibiotics, although about 20% of

the strains which become resistant to this drug *in vivo* show cross-resistance to erythromycin and spiramycin.

It has a low degree of toxicity and is well tolerated by man. It is not bound by serum proteins, and serum levels of 2.5 gammas/ml are obtained on a dosage of 2 Gm/day. Its greatest usefulness appears to be in the case of staphylococcal infections, where the organism is resistant to many other antibiotics.

Nystatin was discovered by Brown and Hazen in 1955, but its utility as an antifungal agent has not been thoroughly investigated until the past 2-3 years. The reason for the renewed interest in antifungal antibiotics such as nystatin has been discussed earlier. To treat patients with oral moniliasis, vaginal infection, and cutaneous involvement, the drug is used in several different forms, including topical ointments, solutions, powders, troches, vaginal suppositories, and vaginal gels. In all reported cases the results were good to excellent. The effects of nystatin in cases of deep mycotic infections have been equivocal, although *in vitro* the organisms appear to be sensitive. Nystatin probably should be administered along with antibacterial drugs to prevent the overgrowth of *Candida* in certain types of patients who are more likely to develop moniliasis, i.e., the debilitated patient, diabetics, and premature infants.

Novobiocin is mainly active against gram-positive cocci, although some gram-negative rods are also sensitive. *In vitro* antibacterial studies with novobiocin show that most strains of *Staphylococcus pyogenes* are inhibited by concentrations of 0.098-1.56 gammas/ml. Novobiocin sensitivity appears to bear no relation to susceptibility to other available antibiotics since freshly isolated strains of staphylococci resistant to penicillin, streptomycin, and the tetracyclines all proved sensitive to novobiocin *in vitro*. A high proportion of strains of *Proteus vulgaris* is inhibited in vitro by 8 gammas/ml or less. Other gram-negative organisms are not inhibited by novobiocin in concentrations obtainable with the recommended clinical dosage. The sodium salt of novobiocin is well tolerated in man, although a yellow discoloration of the plasma may appear a few hours after the first dose, which does not react to tests for bile. It would

appear that this yellow pigment is a metabolite formed from novobiocin.

Serum levels of 2-80 gammas/ml. are obtained and maintained on a dose of 250 mg. q 6 h or 500 mg. every 12 hours. There is considerable evidence to indicate that novobiocin is reversibly bound to serum albumin, thereby providing a reservoir of utilizable antibiotic. The drug does not diffuse through the normal blood-brain barrier.

The majority of staphylococcal and streptococcal infections treated with novobiocin respond very well to the doses mentioned. There is a very marked tendency on the part of staphylococci to become resistant to novobiocin *in vitro*. This has not been a problem in the treatment of patients.

Novobiocin is an extremely effective antibiotic for the treatment of resistant staphylococcal infections, and its careful use will make it a most valuable addition to the antibiotic family.

Cycloserine differs in chemical structure from any previously identified antibiotic. It has a low molecular weight, is highly diffusible, and has a broad antibacterial spectrum. It is said to have a synergistic effect *in vitro* with many other antibiotics. Although cycloserine is less effective *in vitro* than some of the well-known and currently used antibiotics, its activity *in vivo* is much higher than might be anticipated from *in vitro* tests. This, however, was not so in the case of the tubercle bacillus. Despite a promising degree of *in vitro* activity, little effect was demonstrable in experimental tuberculosis in mice. Several groups, however, have shown it to be an effective agent in the treatment of pulmonary tuberculosis in man, where it is used very cautiously because of its renal toxicity.

It has been suggested that this drug may be of considerable importance in helping to explain the mechanism of antibiotics, since there is some indication that it may be interfering with a common enzyme system or blocking a common metabolic pathway of a wide variety of microorganisms.

Spiramycin has been in use in Europe for more than a year but has only recently been introduced to this continent. It belongs to the same group of antibiotics as erythromycin and

carbomycin and shows cross-resistance with these two drugs Spiramycin is well tolerated when administered orally, and no cases of staphylococcal enteritis or Candida infection have been reported following its use. It is particularly suitable for the treatment of infections due to gram-positive cocci which are resistant to penicillin and other commonly used antibiotics. It has been used successfully in treating staphylococcal enteritis resulting from the use of other antibiotics. The recommended oral dose of spiramycin is 3 Gm/day given in divided doses at 6- to 8-hour intervals. In severe cases this may be raised to 4-5 Gm/day. It has been suggested that spiramycin should be reserved for the treatment of patients who are sensitive to or cannot tolerate other antibiotics and those with infections due to organisms resistant to other antibiotics.

Amphotericin B is a relatively new antibiotic which has been shown to be very active against pathogenic fungi in vitro. No major toxicity has been observed on either oral or intravenous administration of this antibiotic. Therapeutic trials in cases of progressive histoplasmosis, cryptococcosis, and North American blastomycosis have given promising results, using intravenous administration, whereas the oral preparation has given rather doubtful results. This antibiotic appears worthy of more detailed clinical trials.

Kanamycin is one of the most recent antibiotics to be introduced for clinical use. It is active against a wide variety of gram-positive, gram-negative, and acid-fast organisms. There appears to be no cross-resistance, at least in the case of staphylococci, with penicillin, streptomycin, tetracycline, chloramphenicol, erythromycin, oleandomycin, and novobiocin. However, kanamycin and neomycin are almost completely cross-resistant, while kanamycin and streptomycin show incomplete cross-resistance. Staphylococci appear to develop resistance very slowly in vitro, while *Mycobacterium tuberculosis* does so more rapidly (Gourevitch et al., 1958).

Kanamycin is poorly absorbed after oral administration and at present only an intramuscular dosage form is available. Early clinical trials suggest occasional toxic manifesta-

tions, i.e., renal irritation, 8th nerve dysfunction, skin eruptions, and neutropenia. The recommended adult dose is 1-2 Gm/day in 2-4 equal amounts intramuscularly.

A number of antibiotics which are said to be active against certain types of tumor cells have been isolated in the past two years. None have reached the point at which they are ready for clinical trials, but they are being tested against experimental tumors in animals.

MICROORGANISMS OF IMPORTANCE IN SURGERY

In the following paragraphs the important pathogenic microorganisms in their relation to disease will be discussed. For the sake of uniformity the classification and terminology as given in Bergey's Manual (1948) will be followed, but long-established names in clinical use are given in brackets to avoid confusion, especially for those familiar with an accustomed terminology.

STAPHYLOCOCCUS PYOGENES

The term *Staphylococcus* for the species of medical interest has been in use for more than 60 years and will probably persist. Staphylococci are parasitic on the skin and mucous membranes and about 20% of people in some regions are nasal carriers. They are gram-positive, spherical cells and occur characteristically in clusters. *Staphylococcus* grows on all ordinary culture media. A useful provisional differentiation of the pathogenic from saprophytic strains is obtained by means of the "coagulase test." Pathogenic strains coagulate human or rabbit plasma (coagulase positive). Because of their wide distribution and their relative resistance to drying and heat, infections with staphylococcus are very frequent and will be encountered by the surgeons in one form or another almost daily.

Various clinical pictures are produced, ranging from mild superficial localized skin lesions to fulminating generalized infections. The ability of *Staphylococcus pyogenes* to cause disease is dependent on the toxins and enzymes produced by this organism, and the frequency and severity of the infections, as in most other diseases, depend on the relative susceptibility of the host and the defense

mechanism of the body. A soluble, filtrable, thermolabile exotoxin is produced by *Staph. pyogenes*. This exotoxin is antigenic and gives rise to an antitoxin. By treatment with formalin, an antigenic toxoid can be prepared and can be used to produce active antitoxin immunity. The exotoxin of *Staph. pyogenes* strains pathogenic for man contains a hemolysin for rabbit cells (alpha hemolysin), produces tissue necrosis, and is lethal for experimental animals.

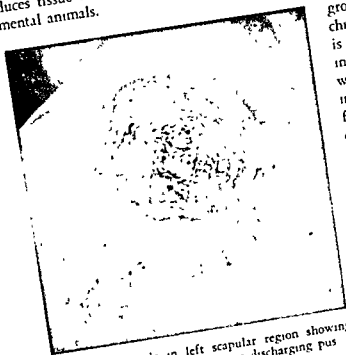


Fig. 7.—Carbuncle in left scapular region showing characteristic multiple openings discharging pus.

Whether the exotoxin is a single entity or whether these three reactions are caused by different fractions of the toxin is a question which still awaits an answer. Leukocidin, another toxic fraction of *Staph. pyogenes*, is of great importance for the establishment of infection because of its destructive action on leukocytes. This and coagulase are certainly partly responsible for the ease with which staphylococcus implantation occurs. Of the enzymes elaborated by *Staph. pyogenes*, mention has already been made of the coagulase which permits the differentiation of pathogenic from nonpathogenic strains. Fibrinolysin is another enzyme produced by staphylococcus for man to be confined to strains pathogenic for man. A mucolytic enzyme, hyaluronidase, is produced, which increases the permeability of connective tissue and assists the spread of the infection from the initial focus.

Staph. pyogenes is a good example of an organism which causes disease by active multiplication and toxic products. The clinical manifestations represent the sum total of both factors, and the clinical features of severe infections somewhat depend on which predominates, toxemia or bacteremia with pyemic abscesses. If the defense mechanism of the host plays its proper part after the initial invasion, the infection remains localized.

Staphylococcus infections occur in all age groups, but certain types are more common in children, e.g., osteomyelitis. *Staph. pyogenes* is probably the most common cause of wound infections, either alone or in combination with other pathogens. Primary staphylococcus infections range from the most superficial folliculitis through furuncles, carbuncles, and destructive osteomyelitis to fulminating septicopyemia. Many show a tendency to chronicity or persistence, particularly if the resistance mechanism is weakened by chronic diseases. Generalized spread may follow, either because of insufficient resistance or because the particular strain involved shows a high degree of invasiveness or toxin production. Thrombophlebitis and metastatic abscesses in practically any organ are a common sequence of generalized infection and occasion the severity of the clinical picture. The mortality rate of *Staph. pyogenes* septicemia in the pre-penicillin days was as high as 50-90%.

Staphylococcal infections are characterized by the formation of abundant pus. The pus is of a creamy consistency, slightly yellow in color, and consists mainly of polymorphonuclear leukocytes. Careful microscopic studies of stained films of the pus are often of help in assessing the state of resistance. Large numbers of engulfed cocci are a sign of good phagocytic response, whereas destroyed leukocytes with masses of cocci outside should be a warning that the infection may spread if effective measures are not taken. Sound surgical judgment has to decide the indication for operation and adequate drainage. Acute infections in well vascularized tissues will respond more readily to systemic treatment with appropriate antibiotics. Infections of the bone and joints, and older abscesses with fibrotic avascular surrounding tissue, require local as

well as systemic application after proper drainage in most instances Staphylococcal empyema and brain abscesses have been treated successfully with systemic and topical use of antibiotics after drainage

It has already been stated in the general discussion on chemotherapeutic agents that treatment of staphylococcal infections with antibiotics has become a major problem. Penicillin is still the drug of choice if the strain is sensitive, but only a small percentage of staphylococcal strains in hospital practice is susceptible to penicillin It is no longer a question of using a "drug of choice" but of finding an antibiotic which will inhibit the strain in practically obtainable concentrations Sensitivity testing of staphylococci is imperative if treatment with an antibiotic is indicated The clinical importance of staphylococcal resistance can hardly be overemphasized The vast number of reports on hospital staphylococcus infections is proof of the seriousness of this problem Infections vary from relatively mild superficial to severe and even fatal types Wound infections, urinary tract infections, and staphylococcal pneumonias are the most frequently encountered forms Staphylococcal enterocolitis as a possible complication of antibiotic therapy has already been discussed Various measures to combat these hospital infections, occasionally occurring in epidemics, have been suggested (Starkey, 1956) The difficulties of effective antibiotic treatment of these infections have focussed renewed attention on older forms of treatment In addition to any effective antibiotic, the use of antitoxin is indicated in severe fulminating generalized infections when toxemia dominates the clinical picture, and may be lifesaving in less severe cases if the strain is highly resistant Antitoxin should be administered intravenously as early as possible in such cases

In chronic and recurrent staphylococcal infections, as well as in early stages of boils, staphylococcus toxoid has proved its value

Antibiotics are often contraindicated in these cases unless the severity of a boil or carbuncle makes their use mandatory The very chronicity of these infections, requiring the repeated use of antibiotics, leads to the de-

velopment of drug-fast strains with all the discussed consequences The immunity produced by a course of toxoid is antitoxic and protects the tissues against the various destructive actions of the toxin No general rules can be given as to how many courses may be necessary Individuals vary considerably in their antitoxin response, and adequate titer has to be judged in every case by clinical improvement With care and patience most gratifying results can be obtained in recurrent furunculosis and chronic osteomyelitis In the rare condition of congenital aglobulinemia characterized by recurrent infections, the use of gamma globulin has proved of value in supporting the immune mechanisms

STREPTOCOCCUS

The genus *Streptococcus* is of great importance in surgical practice and causes a wide variety of infections Streptococci are gram-positive cocci which usually occur in chains of varying length and at times in pairs A variety of species has long been recognized and many attempts at classification have been made Differentiation according to changes produced on red blood cells has led to the terms hemolytic, viridans, and indifferant, which correspond respectively to *beta*, *alpha*, and *gamma* hemolysis This caused overemphasis of hemolysis and the use of "hemolyticus" as a species epithet for which there is no justification Actually there are five recognized species in the pyogenes group and two in the enterococcus group which are all hemolytic Other classifications are based on the action of streptococci on a number of carbohydrates and other biologic reactions, and on these Sherman (1937) divided the aerobic streptococci into four sections (1) the hemolytic pyogenic group, (2) the viridans group, (3) the lactic group, (4) the enterococcus group A subdivision of the hemolytic streptococci on the basis of antigenic differences has been developed by Lancefield (1928) and some of these subgroups differentiate a single species, while others comprise two or more distinct species Lancefield subgroup D comprises all the enterococci whether they are hemolytic or not No completely satisfactory classification has as yet been found for the viridans group

and the anaerobic species. The growth requirements of streptococci vary for the various species, but blood agar usually provides a reasonably satisfactory medium for the isolation and characteristic colony production.

The variety of clinical signs and symptoms produced by streptococcal infections has led to the intensive search for the factors responsible for the differences both in the bacterial cell and in the host. Streptococci are widely distributed among man, animals, and probably plants. Some species are harbored as commensals in the human mouth and throat. Most species may survive at least for several weeks in clothing, bedding, and food. Milk is a favorable medium, and milk-borne streptococcal epidemics are not uncommon. Crowding as in schools, barracks, hospital wards, etc., increases the incidence of streptococcal infections. The two dread diseases of the surgeon and obstetrician, erysipelas and puerperal fever, are fortunately more of historic interest today, but the efforts of men like Holmes, Semmelweis, Billroth, Lister, and many others should not be forgotten.

Extensive studies have led to the recognition of a number of components in the streptococcal cell which have contributed to our understanding of the disease processes. The serologic classification of many aerobic streptococci depends on differences of a carbohydrate "C" substance, and although it has no relationship to virulence, it provides the grouping of streptococci as already pointed out. The vast majority of human infections seem to be caused by Group A streptococci, although other groups occasionally cause disease. The groups can be further divided into types, and within Group A, for instance, at least 40 specific types have been identified. The type-specific antigen is a protein "M" substance, and antibodies against it confer type-specific protection. The amount of "M" substance produced by a given strain is partly responsible for virulence. Type-specific antigens in other groups are either proteins or polysaccharides. Two other fractions occur in the cells, "T" and "P" substances, which are not known to have any bearing on the virulence or pathogenicity of the strain. A number of other substances elaborated by streptococci have to be discussed briefly because they are essential for the un-

derstanding of the pathologic features of streptococcal infections. The erythrogenic toxin, produced by streptococci, particularly of Group A, some of Groups C and G, is held responsible for the rash in scarlet fever. Immunity after scarlet fever is antitoxic, and only the erythrogenic toxin is neutralized, but no protective effect against the bacterial cell is provided. At least five immunologically distinct toxins are known which may explain the occurrence of an occasional second or even third attack of scarlet fever.

Most Group A streptococci produce two different kinds of hemolysins, O and S, and the latter is pathogenic for laboratory animals. Antibodies against streptolysin O can be demonstrated following infections with streptococci producing streptolysin O and may persist for years. The part this O lysin plays in the pathogenicity of the strain is not known, but streptolysin O antibodies have been investigated for epidemiologic studies.

Two enzymes, or pro-enzymes, produced by many strains of streptococci of Groups A, C, and G, namely, streptokinase (fibrinolysin) and proteinase, are both antigenic, but their relation to the lesions produced in infection is still obscure. Some strains elaborate hyaluronidase, an enzyme which is capable of breaking down hyaluronic acid, which forms the intercellular ground substance of connective tissue and cartilage. This enzyme was thought to be responsible for the invasiveness of streptococci, but many highly pathogenic strains do not produce it; furthermore, many highly virulent streptococci are encapsulated and the capsule is formed mainly by hyaluronic acid.

Summarizing, we can only state at present that pathogenicity is associated with the "M" substance and the erythrogenic toxin. How much the other discussed components contribute to the virulence of a given strain remains to be proved.

It is of interest to realize that some of the substances elaborated by streptococcus are now used therapeutically by the surgeon—*streptokinase* and *streptodornase*.

Hardly any other bacterial species is capable of causing so many diverse pathologic and clinical pictures as streptococci. Streptococci

commonly enter through accidental wounds which may be quite small and hardly more than scratches. The response to this invasion depends on the strain and the susceptibility of the host. We have to differentiate between infections caused by the various groups of aerobic streptococci and the microaerophilic and the anaerobic streptococci.

The following clinical manifestations are caused in the vast majority of cases by *Streptococcus pyogenes* Lancefield Group A, more rarely by Group C, and very occasionally by Group G.

1 *Erysipelas* The clinical picture is characterized by a *brawny-red* induration of the skin. The margin of the erythema is raised and irregular. Occasionally small blisters are formed on the surface, from which *Str. pyogenes* can be isolated. If unchecked by treatment and in a patient without resistance, erysipelas can spread rapidly. Under such conditions, signs and symptoms of toxemia develop. Histologically the main feature of the lesion is intense inflammation of the superficial lymphatics which are filled with polymorphonuclear leukocytes, fibrin, and bacteria.

2 *Lymphangitis, lymphadenitis, cellulitis, and septicemia* From a small puncture wound a red streak along the lymph vessel rapidly develops, the regional lymph nodes become tender and enlarged, and softening and supuration may follow. If spread is not checked bacteremia and septicemia may follow, usually originating from a thrombophlebitis or directly from the lymphatics. Chills and high fever are the clinical signs of blood stream invasion, and other signs and symptoms may develop, depending on the site or sites where infected emboli may be lodged. These may give rise to abscesses in the spleen, kidney, or brain or to purulent arthritis, pleuritis, pericarditis, peritonitis, or meningitis. This sequence of events may occur irrespective of the primary portal of entry. The upper respiratory tract is a frequent primary site of invasion and of major interest to the otolaryngologist. Pharyngitis, follicular tonsillitis, peritonsillar abscesses (quinsy), retropharyngeal abscesses, and the now rare condition of Ludwig's angina are all caused by *Str. pyogenes*. From the nasopharynx the streptococci may pass through the Eustachian tube to the middle

ear and mastoid and to the large cerebral sinuses. Any of these may in turn lead to generalized infection. The involvement of the regional lymph nodes is very characteristic of streptococcal infection.

Recognition of the cause and proper counteraction have almost eliminated puerperal fever, a once very prevalent type of streptococcal infection, and if infection post partum does occur immediate treatment will in most instances prevent serious sequelae.

Infections with *Str. pyogenes* have fortunately lost much of their serious implication for the patient. *Surgical drainage, wound débridement, and chemotherapy*, either in the form of sulfonamides or in more serious cases in combination with penicillin or other antibiotics, will in most cases prevent spread and induce recovery.

Dosage and mode of application have to be adjusted to the individual case and should follow the principles as outlined in the general discussion. For superficial wound infections or infections of burns where local treatment may be a more rational form, topical application of bacitracin may be preferable to penicillin because of lack of sensitization.

The *viridans* group of streptococci is of much less concern in surgical practice. They probably play a role in infections of the gall bladder and apical abscesses of teeth and are the cause of a certain number of urinary tract infections. Pathogenicity of the lactic group for man has never been established. In the treatment of any infection due to a member of the viridans group of streptococci, it should be kept in mind that these organisms are less sensitive to penicillin and a higher dosage is required.

Subacute bacterial endocarditis is considered a medical problem, with the exception of those cases where the vegetations are situated in a patent ductus arteriosus. Ligature or division and suture of the ductus may be indicated. Intensive antibiotic treatment before and after operation will aid this procedure.

The last group of the aerobic streptococci comprises four species, commonly called *enterococci* or *fecal streptococci*, and all four species belong serologically to Lancefield Group D. Enterococci, as the name implies,

form part of the flora of the intestines, particularly the colon. Their main importance is in infection of the peritoneum and the urinary tract and in subacute bacterial endocarditis. Either alone, or more often with the colon bacillus, they are found in appendiceal abscesses, localized or diffuse peritonitis, and following inflammatory processes or injuries to the terminal ileum, appendix, or colon. In the urinary tract they cause cystitis, pyelitis, and pyelonephritis. Invasion of the blood stream is rare but does occur, particularly following surgery on the infected kidney. Combined penicillin-streptomycin is the accepted treatment in subacute bacterial endocarditis.

Several species of *anaerobic streptococci* are of *clinical importance*. They can be isolated from the mouth, intestines, and vagina of apparently healthy persons; relatively little is known about their antigenic structure, and their pathogenicity for laboratory animals seems to vary widely. Their presence is often suggested by a foul odor and gas formation. Anaerobic streptococci are a frequent source of *post-partum infection*, either of a localized nature, as endometritis, or giving rise to generalized infections in which they can be isolated from the blood stream. Anaerobic streptococci are also often found in *lung abscesses*, usually in combination with other organisms,



FIG. 8 Typical undermining ulceration (Meleney) due to a microaerophilic hemolytic streptococcus

due to enterococci. Sulfadiazine and other sulfa drugs are used successfully in the treatment of urinary tract infections.

Meleney (1935) described a peculiar spreading, burrowing, undermining ulceration of the skin caused by a microaerophilic hemolytic streptococcus. Treatment with zinc peroxide has been highly effective if the method as outlined by Meleney is carefully followed. "Progressive postoperative bacterial synergistic gangrene" is due to a nonhemolytic microaerophilic streptococcus (*Str. erolutus*) in synergism with a *Staph. pyogenes* according to Meleney (1919).

more rarely in mastoiditis and brain abscesses. Traumatic wounds with crushed tissues favor the growth of this group. Abscesses adjacent to the intestinal tract (*perirectal*, *pilonidal cysts*) are sometimes caused by anaerobic species, which can also be found in mixed cultures in *appendiceal abscesses* and *peritonitis*. The pus and discharge are often, but not always, foul smelling. Most anaerobic strains of streptococci are sensitive to penicillin, but as they often occur in mixed infections, combined treatment with penicillin and streptomycin or one of the broad spectrum antibiotics may be a more adequate form of

therapy. Drainage of any accessible accumulation of pus and removal of necrotic or gangrenous tissue are essential and, depending on the severity of the infection and the tissues or organs involved, local and systemic treatment with the chosen chemotherapeutic agents may be indicated

DIPLOCOCCUS PNEUMONIAE (PNEUMOCOCCUS)

The pneumococcus is a gram-positive, ovoid coccus with an easily demonstrable capsule. The pneumococci are arranged in pairs and short chains. They can be grown fairly readily, and on blood agar the colonies in aerobic culture produce a zone of greening. Bile or bile salts bring about rapid autolysis of the pneumococci, and turbid suspensions are cleared in a short time on incubation at 37° C. This simple test can be used to differentiate pneumococci from the viridans group of streptococci, the only organisms with which they could be confused. Pneumococci occur in the upper respiratory tract and cause infections mainly of the lungs, the accessory sinuses, middle ear, and meninges. Rarer but of greater importance to the general surgeon are pneumococcal peritonitis, metastatic purulent arthritis, and empyema. As mentioned above, a characteristic of the pneumococcus is the capsule, and on the basis of differences in immunochemical structure of the capsular polysaccharide, pneumococci can be divided into at least 75 types. These capsular polysaccharides are antigenic, and type-specific antibodies are protective. The capsule is an essential attribute for the pathogenicity or virulence of pneumococci by increasing the resistance of the bacterial cell to phagocytosis (Avery, 1932) (Wood, et al, 1946). No toxins in the accepted sense have been demonstrated. The physical appearance of pneumococcal pus differs from other pus by its high content of fibrin and its extremely tenacious character which is of practical importance because it hinders proper drainage and penetration of therapeutic agents. Streptodornase is used to liquefy pneumococcal pus in order to facilitate the access of drugs to the organisms.

The sulfa drugs and antibiotics have considerably lowered the mortality rate of pneumococcal infections. This is of great impor-

tance to the surgeon, as postoperative pneumonia was one of the serious consequences of many surgical interventions. Empyema, too, has become a relatively rare disease, and if it occurs can usually be well managed. The pus should be aspirated and replaced by local instillation of an antibiotic (penicillin) supplemented by systemic treatment if necessary. The same approach is successful in the management of pneumococcal arthritis.

Pneumococcal meningitis remains a serious problem; primary foci, usually in the sinuses or mastoid, must be eliminated. Development of resistance of pneumococci to penicillin is a rare occurrence, but the possibility must be kept in mind, and if clinical improvement does not follow an ordinarily sufficient dosage of penicillin, the sensitivity of the strain should be tested. Significant spinal fluid concentrations are obtained following standard dosage of tetracyclines and these antibiotics are used in meningeal infections. As pneumococci are carried by many people in the nasopharynx and cause a large number of sinus infections, the surgeon must be alert to the danger of pneumococcal meningitis in all cases of skull fractures.

ENTEROBACTERIACEAE

This family contains a number of species; some live in the intestinal tract as commensals and others are obligatory parasites. All species are gram-negative rods and many show active motility by means of flagellae. All members of the family can be grown on ordinary culture media and ferment a variety of carbohydrates with the production of acid or acid and gas which, with other metabolic characters, are used to differentiate the various species. The lactose-fermenting genera are *Escherichia*, *Aerobacter*, and *Klebsiella*, and the first two are often referred to as the colon-aerogenes group. *Escherichia* can be divided into three species, *E. coli*, *E. freundii*, and *E. intermedium*, but for the sake of simplicity we shall use the collective term *E. coli* for the three species. *E. coli* is an inhabitant of the intestinal tract of man and animals and as such is not only harmless but, as already mentioned, is also useful in synthesizing certain vitamins (Johannson and Sarles, 1949). This has certain practical implications for the surgeon. The preparation of

the intestinal tract with sulfonamides and antibiotics before major surgical procedures on the colon has been adopted as a routine procedure and the decrease in bacterial flora, if protracted, may lead to vitamin deficiencies. This fact has been recognized and joint administration of vitamins and antibiotics is recommended.

The infections caused by *E. coli* are usually of a localized nature involving organs anatomically related to the intestines. *E. coli* is the most common cause of cystitis, pyelitis, and pyelonephritis. Of greater interest to the general surgeon are the infections in the abdominal cavity, peritonitis, and cholecystitis. In appendiceal abscess, *E. coli* can be found in combination with a variety of other organisms; it certainly is not the primary cause of appendicitis. In peritonitis following perforation of some part of the intestine, *E. coli* is often the only or the predominating organism. In infants invasion of the blood stream followed by metastatic abscesses, particularly in the brain, is not a rarity. *E. coli* also plays a part, often in combination with other organisms (staphylococcus and streptococcus), in burns or in postoperative infections of abdominal wounds. The purulent discharge from *E. coli* infections often has a foul odor.

Removal of the primary focus of infection (appendix, gall bladder) and effective drainage of abscesses are as always the important steps. In the choice of an antibiotic, consideration must be given to the sensitivity of the strain. In the treatment of urinary tract infections, attention should be paid to any anatomic malformations or other occlusions which prevent free drainage and penetration, otherwise even the most effective antibiotic will only produce temporary relief. The *Aerobacter* of different species produce, on the whole, the same type of infections as *E. coli*.

Klebsiella pneumoniae is a gram-negative, nonmotile rod. It grows well on ordinary nutrient media with large mucoid colonies. A striking morphologic character is the capsule containing a type-specific polysaccharide. And Julianelle (1926) described three antigenic types, A, B, and C, and a heterogeneous Group X. Types A and B are much more frequently found than type C and seem to be

more highly pathogenic. Friedländer's bacillus appears to live in the upper respiratory tract of man and has also been isolated from feces in healthy persons. Surgical infections caused by Friedländer's bacillus are empyema, bronchiectasis, sinusitis, occasionally appendiceal abscess, cystitis, and pyelonephritis. Brain abscesses and generalized septicemia with metastatic abscesses are rare complications. The exudates in Friedländer infections, unless frankly purulent, are highly viscous, due to the large amount of capsular material. Sulfadiazine and streptomycin have sometimes shown good results in the treatment of Klebsiella infections and the broad-spectrum antibiotics have been used successfully in small groups of cases. In an individual case the choice of drug is best made on the basis of sensitivity tests.

Salmonella typhi (typhoid bacillus) is a gram-negative motile bacillus, the causative agent of typhoid fever. It concerns the surgeon both during the active disease and in connection with lesions occurring years after the initial infection. The typhoid bacillus grows readily. It can be isolated from the blood of the patient during the acute stage (first week and beginning of second week) and from the urine and stool from the end of the first week on. Antibodies against various fractions of the bacillus appear in the blood, usually in the third week, and may persist for several years after recovery, conferring a certain degree of immunity. The Widal reaction is based on the demonstration of these antibodies by agglutination of bacterial suspensions. The widespread artificial immunization against typhoid fever has somewhat impaired the diagnostic significance of the Widal test and the evaluation of positive results has to be based on at least two successive tests at a few days' interval. Endotoxins which are liberated by autolysis of the bacilli are highly toxic and considered responsible for the clinical findings which are chills, fever, malaise, and headache.

The surgical lesions during the active stage of typhoid fever are orchitis, epididymitis, perichondritis, and perforation of the intestinal ulcers with local or generalized peritonitis. Meningitis and empyema may also occur during the acute stage. It must be kept in mind that typhoid fever is a systemic disease and

bacteremia is the main feature. Localization of organisms in practically any organ may result, although the gall bladder, kidneys, and bones are most commonly affected.

Chronic cholecystitis with or without acute exacerbations, often with formation of stones, is frequently due to the typhoid bacillus. Suppurative periostitis of the tibia, ribs, or vertebrae may be a late sequel. The diagnosis can be established only by the isolation of *Sal. typhi*. A few other members of the large Salmonella group may give rise to a similar clinical picture. Chloromycetin is the treatment of choice for typhoid fever. The results in a large series of cases have been very gratifying; the hopes, however, that the carrier state may be prevented or eradicated have not materialized.

Only one other group of lactose-negative organisms of the Enterobacteriaceae family is of interest to the surgeon: *Proteus*. The members of this group are gram negative, motile bacilli, which are widely distributed in nature, sewage, and manure, and are frequently found in small numbers in the feces. Members of the proteus group are frequently the cause of urinary tract infections, which can be extremely severe and lead to invasion of the blood stream, particularly following renal surgery. As one of the intestinal inhabitants, it is sometimes found in combination with other organisms in any infection of the abdominal cavity. The chief importance of the proteus group is as secondary invaders in wounds, middle ear diseases, ulcerations, and burns, particularly of the lower extremities (fecal contamination). Some members of the proteus group are highly proteolytic, and any attempt at skin grafting while the area is infected by these microbes is futile. Wound exudates of proteus infections have a characteristic unpleasant odor. These infections, although as a rule not serious on the skin, are very stubborn. Because of pronounced variations in susceptibility to antibiotics from strain to strain, sensitivity testing is essential for rational treatment.

Although not belonging to the family of Enterobacteriaceae but to the Pseudomonadaceae, the only member of clinical importance, namely, *Pseudomonas aeruginosa* (*B. pyocyaneus*), is discussed in connection with proteus,

because they quite often occur together in mixed infections and have other features in common. *Pr. aeruginosa* is a gram-negative, usually motile bacillus, which produces a highly diffusible greenish or greenish-blue pigment, and this organism has been long known to the surgeon as "blue pus" former. Like proteus, it is widely distributed in nature and occurs occasionally in the intestine. Similar to proteus it causes infections of the urinary tract, the middle ear, and particularly infections of burns and wounds. Meningitis, arthritis, and eye infections have been reported. The bluish-green color and a sweetish smell of the exudate is very characteristic. So far as chemotherapy is concerned the same rules apply as for proteus infections.

NEISSERIA GONORRHOEAE (GONOCOCCUS)

The gonococcus appears in exudates and cultures as a gram-negative diplococcus, with the adjacent sides flattened (coffee-bean shaped). The cultivation of the gonococcus presents certain difficulties, as it is fastidious and easily killed by drying or sunlight. Great care has to be taken in securing exudates, and best results with culture methods are obtained if the appropriate media are inoculated at the bedside. In acute disease of the male the finding of large numbers of morphologically typical intracellular or extracellular organisms on stained films may seem sufficient corroboration of the clinical diagnosis.

The surgeon is, as a rule, confronted with complications or sequelae of the primary gonococcal infection. Infection of the urogenital tract in the female is a frequent complication of gonorrheal cervicitis; urethritis, infection of Skene's or Bartholin's glands, salpingitis, and peritonitis are the lesions caused by lymphatic spread or direct progression. A creamy mucopurulent discharge is characteristic of gonorrheal infection. Fibrotic occlusion of the Fallopian tubes may necessitate surgical measures. In the male, prostatitis, urethral strictures, and epididymitis are the common complications of urethritis. Invasion of the blood stream by the gonococcus may take place and produce arthritis, fibrositis, iritis, endocarditis, pericarditis, and occasionally meningitis. In the

acute state, penicillin, 200,000-300,000 units, will cure the majority of cases. Localized complications, such as arthritis or Bartholin abscesses, may require surgical drainage and local instillation of penicillin as well as systemic treatment

PARVOBACTERIACEAE

The family consists of several genera and includes a large number of species, all of which are primarily of interest to the physician. A few species cause abscesses and other lesions which require surgical attention

Pasteurella.—*Pasteurella multocida* and *Pasteurella tularensis* appear to be the two species which need to be considered. Both are gram-negative, short, nonmotile, oval bacilli, the latter may show great pleomorphism. *P. multocida* grows readily but *P. tularensis* needs special media for primary isolation and will be missed unless there is some indication given to the laboratory of the suspected type of infection. *P. multocida* occurs as saprophytes and parasites in many animals and can be found frequently in the upper respiratory tract of normal domestic animals. Human infections in the form of abscesses, frequently complicated by osteomyelitis, are generally due to animal bites, particularly cat, dog, and rabbit. Generalized infections and cases of meningitis and empyema have been reported. All the strains of *P. multocida* which have been tested were sensitive to penicillin (Schupper, 1947).

P. tularensis is the causative agent of tularemia. The main sources of infection for man are (1) direct contact with infected animals, (2) the bites of ticks and flies (deer flies), (3) ingestion of contaminated meat or water, (4) laboratory infections. Some occupations show a higher incidence of infections. Hunters, farmers, and laboratory workers. If the organisms penetrate the skin, a primary lesion in the form of an ulcer develops in a certain percentage of cases. Spread occurs along the deep and superficial lymphatics, leading to lymphangitis and lymphadenitis with suppuration. This is the type of tularemia infection which the surgeon encounters. Generalized signs of fever and elevated sedimentation rate are always present, the white count may or may not be elevated. A transitory bacteremia

is usual during the first week but disappears as soon as antibodies are formed, but spread of the infection by the lymphatic route may take place with renewed, usually fatal, invasion of the blood stream, unless checked by treatment. The diagnosis can be made by the isolation of *P. tularensis* from the primary lesions or the pus from lymph nodes in the early stages. If isolation of the organism fails, the clinical diagnosis can be confirmed in the later stages of the disease by serologic methods. Agglutinating and complement fixing antibodies appear in the blood from the second week on and show a rising titer during the third and up to the fifth week. These antibodies may persist for many years or even through life. A positive test, therefore, does not necessarily indicate present infection with *P. tularensis*, but the rise in titer between the second and third week of a disease suspected to be tularemia is of a great diagnostic significance. An intradermal test with specially prepared antigen (Foshay, 1940) is of diagnostic value. It shows the tuberculin type of reaction 48 hours after inoculation and is usually positive earlier than the agglutination test. The altered skin reactivity may persist for the remainder of life. Tularemia had been a highly fatal disease until streptomycin became available, this antibiotic has an almost immediate effect and a short course of six days has been found effective, even in the pneumonic form of tularemia, if treatment is started early in the course of the disease. The tetracyclines have been used in a number of cases with very satisfactory results.

Brucellaceae.—*Brucella abortus*, *Brucella melitensis* are the cause of acute and chronic brucellosis, a relatively common disease on the North American continent. Although primarily a systemic disease with which the physician has to deal, brucella infection may lead to localized lesions in bones and joints and as such becomes the concern of the surgeon. Because of the great difficulties in diagnosis, it appears important to draw the attention of the surgeon to this type of infection and make him conscious of the fact that brucellosis gives rise to a variety of lesions.

Brucella is a gram negative, small, non-motile rod, which causes disease in ungulates

transmissible to man. The difficulties of isolation, particularly in chronic cases, have been stressed by all workers, and *Brucella abortus* requires increased CO₂ tension for isolation. The main source of infection is unpasteurized milk, milk products, and infected meat. The localized lesions are mainly granulomatous in character, and proliferation of the cells of the reticuloendothelial system forms the basis of the lesions. The clinical picture has so many variations that the multiplicity of symptoms as such is often suspicious. The only exact diagnosis can be made by the isolation and identification of the organism which, as already mentioned, is beset with difficulties. Skin reaction to *Brucella* vaccine or brucellergin and the agglutination test and the opsonophagocytic index are diagnostic adjuvants, but only of relative value. As immunity reactions may persist for many years, a positive test is the only evidence that exposure to *Brucella* occurred at some time but does not necessarily indicate an active infection. Negative results, on the other hand, do not exclude active brucellosis (Huddleson, 1943). Bone and joint complications of brucellosis are probably not very frequent, but the small number of reported cases may be misleading because of the rarity with which the diagnosis can be established with certainty. Arthralgia in multiple joints seems to be a common symptom, but suppurative arthritis, osteomyelitis, and spondylitis have only more recently been given proper attention. Every attempt should be made to verify the clinical diagnosis by the isolation of the microbe in order to institute therapy. Streptomycin in combination with a tetracycline is the accepted form of treatment.

Bacteroidae.—*Bacteroides* are small, anaerobic, gram-negative, pleomorphic, non-spore-forming bacilli; their normal habitat is on the mucous membranes of the body. They cause putrid infections of wounds either alone or in combination with other bacteria. Post-partum infections, tonsillar abscesses, and subphrenic abscesses are the main lesions caused by this genus. Blood stream invasion from any of these foci may occur, with metastatic abscess formation and a high fatality rate in the reported cases. Anaerobiosis is essential for isolation, and it seems probable that such in-

fections are missed unless anaerobic cultures are routine in the laboratory. McVay and co-workers (1949) reported two cases successfully treated with chlortetracycline. In vitro test showed the organisms (*B. funduliformis*) resistant to penicillin, sulfonamides, and streptomycin.

BACILLACEAE

Only one species of the aerobic genus *Bacillus* is pathogenic, causing the infection known as anthrax. *Bacillus anthracis* is a facultative, aerobic, gram-positive, spore-forming, capsulated, nonmotile bacillus, occurring often in long chains. Growth occurs readily on conventional laboratory media. The spores are very resistant and may survive for years in earth or other materials. Anthrax is primarily a disease of domestic animals; human cases are relatively rare and occur mainly in men occupied in certain industries dealing with hides or skins, or in agricultural workers. Shaving brushes and furs may be rare sources of infection. Cutaneous anthrax (malignant pustule) starts as a small red spot which shows a central blister and some edema. In the blister fluid the anthrax bacillus can be readily found. During this stage the nature of the lesion may not be recognized unless the occupation of the patient rouses suspicion. In later stages the necrotic center of the lesion with the black crust "eschar" and the usually marked edema are characteristic of anthrax. Enlargement and tenderness of the regional lymph nodes are frequently present, and depending on the severity of the infection, systemic signs and symptoms of fever and prostration occur. The bacteriologic diagnosis is made by direct culture or inoculation of exudate into mice or guinea pigs and subsequent culture. In severe unchecked infections bacteremia occurs, and the mortality rate of such untreated patients may be as high as 20%. Surgical incision of the primary lesion is contraindicated, and early recognition of the true nature of this infection will prevent dangerous interference which may lead to bacteremia. Penicillin is the drug of choice, and treatment should be maintained at least until bacteria disappear from the lesion. Good results have been obtained with antiserum.

CLOSTRIDIUM

Clostridium is of much greater practical importance to the surgeon. This genus contains a large number of species of which only some are pathogenic for man, among them the organisms connected with gas gangrene and tetanus. The clostridia are gram-positive, anaerobic, rod-shaped organisms producing endospores; many species are motile. They occur naturally in soil and the intestinal tract of man and animals. Under anaerobic conditions most species grow on a variety of media and can be differentiated on the basis of biochemical reactions. The clostridial species most commonly present in gas gangrene elicit their effect in the tissues, and the systemic signs and symptoms are caused by toxins and enzymes produced by the bacterial cells. In most infections more than one pathogenic species is present and various nontoxin-producing saprophytic species occur in the same wounds. The following species are regarded as primary cause of gas gangrene (Reed, 1949): *Cl. perfringens* (Welch's bacillus), *Cl. novyi*, *Cl. septicum*, *Cl. bisfermentans* (*Cl. sordellii*), and rarer *Cl. histolyticum*. Other organisms (gram-positive cocci and gram-negative rods) are frequently present in the wounds with clostridia. As all clostridia require more or less strict anaerobic conditions, special features of the wounds are essential for their implantation and multiplication. Crushing injuries of the tissues, with foreign bodies, bone splinters, clothing, and soil particles, provide an ideal basis, and the virulence of the particular species introduced determines the severity of the infection. Local defense mechanisms of the host seem to be interfered with by the necrotizing and leukocidal toxins. The isolation of pathogenic clostridia from the wound does not necessarily imply clinical gas gangrene as there are wide variations in toxigenicity of different strains of the same species. The five mentioned species of *Clostridium* elaborate soluble exotoxins. *Cl. perfringens* has been divided into four types, A, B, C, and D, on the basis of seven different toxins, but only type A has so far been isolated from human infections. Type A produces so-called alpha toxin which has been identified as lecithinase and forms the main lethal component of culture filtrates. Other

enzymes produced by *Cl. perfringens* are hyaluronidase and kappa toxin, a collagen-splitting enzyme, and a variety of additional enzymes have been isolated and studied. All these enzymes appear to be of importance for the growth and multiplication of *Cl. perfringens* in the tissues and the majority of the histologic changes in the muscles can be produced with culture filtrates. Other pathogenic species of clostridia have also been studied as to their toxin and enzyme systems (DeSpain Smith, 1949). All elaborate soluble toxins which are neutralized by specific antitoxins. The toxins have lethal, necrotizing, and hemolytic actions either due to a single or to multiple substances.

The clinical picture of gas gangrene infection is characterized by edema of the subcutaneous tissues; the muscle fibers are friable and discolored, gas bubbles may appear between muscle fibers and the surrounding tissues and account for "crepitation." A blood-stained, often foul-smelling, thin exudate is discharged, containing gas bubbles. If highly proteolytic species take part in the infections, rapid and widespread tissue destruction takes place. Systemic signs and symptoms of severe toxemia with leukopenia, fever, and high pulse rate may be marked. Invasion of the blood stream by one or more species can occur. The clinical picture is not characteristic for any specific type of clostridial infection but the local infection by a single species is often characteristic and very different from that of mixed infections.

In clinically established gas gangrene, time is of the utmost importance, and treatment with antitoxin must be started at once. Polyvalent antitoxin (*Cl. perfringens*, *Cl. septicum*, and *Cl. novyi*) standardized in international units should be given either intravenously or intramuscularly, depending on the gravity of the infection. The antitoxins are prepared by immunization of horses, and the necessary precautions have to be taken to prevent serious incidents due to hypersensitivity. The minimal recommended dose in established infections is three times the prophylactic dose (9,000 units *Cl. perfringens*, 4,500 units *Cl. septicum*, and 3,000 units *Cl. novyi*). This dose should be repeated every four to six hours according to the response of the patient. Chemotherapy

must be considered an adjuvant only to surgery and antitoxin. Local applications of bacitracin and penicillin as well as systemic treatment is advisable, because relatively little will penetrate into the ischemic parts from the circulation.

Clostridium tetani belongs to the same genus as the organisms causing gas gangrene, but provokes an entirely different clinical picture. *Cl. tetani* is a slender gram-positive bacillus with terminal spores and requires very strict anaerobic conditions for growth. It is widely distributed in soil and occurs occasionally in the feces of man and horses. Due to the almost universal use of toxoid for active immunization, tetanus has become a very rare disease, and many a surgeon may not encounter a case during his active life, but on the other hand the very fact of its rarity may cause diagnostic difficulties in the critical early stages of tetanus with possible fatal consequences for the patient.

Tetanus—Tetanus is a typical example of a pure toxemia; all clinical signs and symptoms are due to the action of the toxin and not to the multiplication of the bacteria. Tetanus may follow certain penetrating types of wounds, infections of burns and severe frostbites, or, fortunately, rarely surgical procedures, particularly in abdominal surgery and extractions of teeth. An interesting case has been reported by Murray and Denton (1948) in which clinical tetanus was almost certainly contracted from plaster of Paris. Certain conditions of the wound favor, and are almost a prerequisite for, the germination of *Cl. tetani*. Deep penetrating wounds with associated infection or foreign bodies provide the anaerobic environment which is necessary, but the wound may be very small and even apparently healed by the time clinical tetanus develops. It has been shown experimentally that if washed spores are introduced into healthy tissues, they are taken up by phagocytes and may be eliminated, but if the tissues are damaged and necrotic, the spores germinate and toxin is elaborated. It should be remembered that spores may persist in the tissues for a considerable time. If and when the toxin reaches the anterior horn cells of the nervous system, clinical symptoms of tetanus become manifest.

The pathway of the toxin is still a controversial subject, two main hypotheses exist: (1) toxin is absorbed by the motor nerve endings and reaches the anterior horn cells through the axis cylinders; (2) toxin is carried by the arterial blood to the central nervous system. The latter theory is receiving most support at present. The incubation period in tetanus varies considerably and may be as short as 3-4 days or as long as several weeks, with an average of 10 days. The prophylactic use of tetanus antitoxin after injuries will either prevent tetanus completely or prolong the incubation period, and the clinical disease will be milder.

Clinically, stiffness of the neck, trismus, and difficulties in swallowing are often the first symptoms. Tonic contractions of muscles and very painful clonic spasms elicited by very trivial external stimuli follow. Contractions of the facial muscles produce the characteristic "risus sardonicus." In later stages all the muscles are affected and death is due to respiratory failure. The temperature may be slightly elevated, the pulse is rapid, and the patient remains conscious until the end.

The **clinical diagnosis** should not present any difficulties even in the initial stages if the surgeon is alert to the possibility. The bacteriologic proof is often very difficult or even impossible without adequate specimens. As previously mentioned, the initial lesion may be completely healed, but even if a wound is present, relatively large amounts of tissues and debris from the depth of the wound must be available for cultures to provide some chance for the isolation of *Cl. tetani*.

Treatment—Once clinical signs and symptoms of tetanus are pronounced, the chances of recovery are slim, but good results have been obtained with large doses of antitoxin by various routes, provided complete removal of the focus is achieved. Constant sedation (paraldehyde, chloral, etc.) and muscle relaxants (curare) are essential to prevent exhaustion. In a large series of cases the continuous intravenous administration of mephenesin in addition to established schemes of treatment resulted in a considerably reduced mortality rate (Veronesi, 1956). Supplementary oxygen therapy is important. Antitoxin should be administered intrathecally

(10-20,000 units), intravenously (20-50,000 units), and intramuscularly (50-80,000 units) in the first 24 hours. The intravenous and intramuscular doses must be repeated and carried on, depending on the progress of the patient. Even more heroic doses will not save every patient, and the chief effort of the physician must be the prevention of tetanus by the prophylactic use of antitoxin (1,500 units) as soon as possible after injuries occur. Penicillin should be used to prevent secondary infections (pneumonia), and as *Cl. tetani* is sensitive to penicillin it may check multiplication in the focus, but it has no effect on the toxin absorption.

CORYNEBACTERIACEAE

Corynebacterium diphtheriae is a gram-positive, nonmotile, nonspore-forming bacillus of ten club shaped and usually arranged in palisades. Growth can be obtained under aerobic conditions on the usual laboratory media, but tellurite-containing media are generally employed to suppress the growth of other organisms and to permit differentiation of the three recognized types of *C. diphtheriae* (gravis, mitis, and intermedius). It produces a powerful toxin which is responsible for the disease.

The only interest for the surgeon is that occasionally wounds may become infected with *C. diphtheriae*, and because of the rarity of such infections in temperate climates, they frequently remain undiagnosed until the appearance of late paralysis causes suspicion. Every wound with a grayish slough or membrane and no tendency of healing should be suspected and cultured to arrive at the correct diagnosis. Penicillin is used as an adjuvant with antitoxin.

ERYSIPLOTHRIX

Erysipelothrix rhusiopathiae causes erysipelas, a usually harmless cutaneous infection. The causative organism is a gram-positive, nonmotile, nonspore-forming bacillus. Growth is obtained under reduced oxygen tension on simple media or better on enriched media. Butchers and persons handling poultry and fish are prone to this infection. A purple erythema, with some edema and a slightly ele-

vated margin, is characteristic of this lesion, which occurs chiefly on the hands. The infection remains localized as a rule, although peripheral extension with central clearing is common. *E. rhusiopathiae* is sensitive to penicillin, and the use of it probably shortens the course, although the infection is self-limiting and treatment is not a necessity.

MYCOBACTERIACEAE

Mycobacterium tuberculosis of either human or bovine type is responsible for the clinical disease of tuberculosis. With the discovery of this bacillus in 1882 by Robert Koch, a common cause was found for an extraordinarily wide variety of lesions and clinical symptoms. Surgeons and physicians are equally concerned in the diagnosis and treatment of tuberculosis. *M. tuberculosis* is a slender bacillus, nonmotile and nonencapsulated. Its most characteristic property is the resistance to decolorization by acids, "acid fastness." Special media are used for the isolation of the tubercle bacillus from exudates or tissues, and growth is characteristically slow. Recently media and methods have been developed (Dubos and Middlebrook, 1947; Reed and Morganti, 1956) which permit more rapid growth.

The pathogenesis of tuberculosis is a very intricate subject and cannot be discussed even superficially in this chapter. The reader should refer to general textbooks of pathology or to special monographs (Rich, 1944).

Tuberculous lesions are either productive or exudative in nature, but both types may be present at the same time in the same organ. Microscopically and histologically the productive type of lesion is represented by "tubercle" formation. The exudative type resembles in its very early stages the inflammatory reaction caused by pyogenic organisms, and exudation of fluid and polymorphonuclear cell infiltration are its chief characteristics. This latter type of cell is soon replaced by mononuclear forms. The surgeon is primarily concerned with tuberculosis of certain tissues. These include the lymph nodes, bones and joints, kidneys, epididymes, prostate and seminal vesicles, the pelvic organs in the female, the intestinal tract, the serous membranes (peritonitis, pleuritis, pericarditis), the central nervous system (men-

ingitis and tuberculoma), the larynx and trachea. The clinical features, the differential diagnosis, and the surgical procedures are dealt with in the special chapters.

The *diagnosis* rests ultimately on the demonstration of virulent tubercle bacilli in the exudates or tissues. The finding of acid-fast bacilli on smears is presumptive but not conclusive evidence of tuberculous infection. Saprophytic mycobacteria and acid-fast strains of the genus *Nocardia* may resemble tubercle bacilli so closely that they cannot be differentiated on stained films. Relatively large numbers of organisms have to be present in a specimen to be detected on a smear, and cultivation and animal inoculation must be resorted to if early lesions are to be diagnosed. The histologic examination will often be of great help, but if tissue is being removed, part should be used for animal inoculation which will prove conclusively the presence or absence of virulent tubercle bacilli. X-ray examinations and tuberculin tests have only a limited significance compared with the cultural methods and pathogenicity tests. However, even one or two negative cultures do not rule out tuberculosis.

Treatment.—With the discovery of streptomycin by Waksman and Schatz (1944) active treatment of tuberculosis has become a reality. No longer need the physician and surgeon trust only to the defense mechanism of the body to overcome the infection with the help of such supportive means as rest and nourishment.

Streptomycin, in combination with para-aminosalicylic acid or isoniazid, has proved to be the most satisfactory method of treatment. The addition of PAS or INH to streptomycin delays the development of strains resistant to the latter by 2-3 months and also permits the use of lower doses of streptomycin with a lower incidence of toxic side effects. Neomycin and viomycin are active against *M. tuberculosis* but due to their toxicity must be used with great caution. Cycloserine is still undergoing clinical trials, as is streptovaricin. It is important to check the sensitivity of the patient's strain to these tuberculostatic drugs at regular intervals so that therapy may be altered upon the appearance of the slightest increase in resistance. Treatment based on

inaccurate sensitivity tests produces disappointing results (Pierce, et al, 1957).

The use of tuberculostatic drugs alone is not sufficient for tuberculosis but should be considered a very valuable adjunct to other established forms of treatment. In military tuberculosis and tuberculous meningitis it is the only promising treatment available. It is of great value to the surgeon as preparatory treatment before chest surgery and in the treatment of tuberculous sinuses and tuberculosis of the bones and joints. Indeed, most forms of tuberculosis are benefited by these drugs improving the general condition of the patient, thus making surgery possible.

ACTINOMYCES

Actinomyces bovis and/or *israeli* is the cause of actinomycosis in man. *A. bovis* is a gram-positive, branching, filamentous, nonmotile organism. No spores are formed. Pus from actinomycotic lesions frequently contains "sulfur granules," whitish or yellowish firm granules which, when slightly crushed and viewed under the microscope, show entangled filaments with swollen clublike ends. This appearance, which is also characteristic in histologic sections, is responsible for the name "ray fungus." *A. bovis* can be grown on usual laboratory media under anaerobic conditions. Growth is slow, taking generally 4-6 days at 37° C. *A. bovis* seems to be a strict parasite and has not been found outside the human or animal body. It can be found in scrapings from the teeth, gums, and tonsillar crypts in apparently healthy persons, and the disease is due to an endogenous infection. Although the exact pathogenesis is little understood, trauma, e.g., tooth extractions, fractures of the jaw, or other injuries to the mucous membranes of the mouth, appears to favor the invasion of *A. bovis*. The relative rarity of the disease suggests that as yet unknown factors must play an important role.

Actinomycosis is a subacute or chronic, slowly progressive disease, with three main localizations: the cervicofacial, thoracic, and abdominal regions. The first form is the most common and affects the soft tissues of face and neck. The lesions show marked induration and a peculiar hard consistency is felt.

The skin over the lesions is swollen and brownish red in color. If central softening and break through the skin occur, chronic fistulas are formed. The abdominal type starts usually in the appendiceal region and is commonly mistaken for a simple appendicitis. After appendectomy the poor healing of the wound and fistula formation and later the formation of a tumorlike mass should arouse suspicion. Thoracic actinomycosis affects the lungs, and, if undiagnosed and untreated, will form abscesses and cavities. All three spread slowly but relentlessly, even eroding bone

because, as pointed out, the mouth is the normal habitat of *A. bovis*.

Penicillin is considered the treatment of choice. Rather high doses should be given, one to five million units per day for a minimum of from 6-8 weeks. Surgical excision or drainage may be indicated in some cases.

NOCARDIA

Some species of the genus *Nocardia* are pathogenic for men, causing chronic suppurative or granulomatous lesions. Nocard

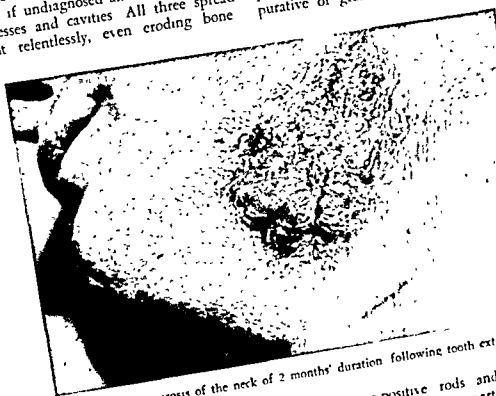


FIG. 9—Actinomycosis of the neck of 2 months' duration following tooth extraction

Hematogenous spread with metastases in many organs has been observed. The clinical diagnosis of actinomycosis can be verified only by the cultivation and isolation of *A. bovis*. The microscopic demonstration of gram-positive filaments or of typical "ray fungus" in histologic sections is acceptable as provisional diagnosis, but cultural isolation should be at least attempted. This presents usually no difficulty in material from primarily closed lesions, provided the laboratory receives some indication of the presumptive diagnosis from the surgeon and cultures are kept long enough. The isolation from heavily contaminated material is more difficult, but if proper methods are used it is generally successful. The interpretation of positive findings from sputum requires caution

are gram-positive rods and branching filaments, some species are partially acid fast and these may be mistaken for tubercle bacilli in stained films. In pus or exudates from lesions, granules similar to the "sulfur granules" in actinomycosis can be found. *Nocardia* can be grown on simple media under aerobic conditions but growth is slow.

Nocardiae occur freely in nature, and infection is either air borne through inhalation or by introduction into the subcutaneous tissue through trauma. Pulmonary nocardiosis resembles tuberculosis both by x-ray and in clinical symptomatology. Hematogenous spread leads to metastatic lesions throughout the body. Brain lesions and meningitis may be caused by *Nocardia* either as primary lesions

or as metastatic lesions from the lungs. In the majority of reported cases the diagnosis was made only after death. The lesions of the subcutaneous tissues and bones are easily mistaken for actinomycosis or fungus infections. Multiple draining sinuses are characteristic of these lesions, which result in the clinical picture of Madura foot. Generalized granulomatous disease due to a *Nocardia* species has been reported recently by Cuttino and McCabe (1949). The frequency of *Nocardia* infections is difficult to estimate, but it is probably higher than the reported cases would indicate. The pulmonary type has had a very high mortality rate. The correct diagnosis can be made only by the isolation and identification of the organism. Sulfadiazine is the drug of choice, but the addition of streptomycin or one of the tetracyclines may be advisable. Treatment should be continued even after clinical manifestations have subsided (Peabody and Seabury, 1957).

SYSTEMIC MYCOTIC INFECTIONS

Most of the fungi responsible for systemic mycotic infections in man belong to the *Fungi imperfecti*. As the isolation media and methods for these organisms differ from those customarily used for bacteria, the fungus etiology of a lesion or disease must be suspected in order to make the proper provisions and arrive at the correct diagnosis. Contrary to earlier beliefs, fungus infections are now known to be widely distributed and even to occur occasionally in epidemic form. Some are world-wide in distribution, and others are restricted to certain areas. Most of the systemic infections are not transmitted from man to man but are acquired from exogenous sources, infected soil, and dust. The clinical manifestations are protean in nature and may resemble those of other chronic diseases such as tuberculosis, syphilis, and tumors. Early and accurate diagnosis is essential, and surgical excision and drainage may be indicated and of definite value. The treatment up to very recently has been mainly supportive, but newly discovered chemotherapeutic agents appear very promising.

Sporotrichosis—This infection is caused by *Sporotrichum schenckii*. This fungus can be

grown on the common laboratory media and forms characteristic colonies on Sabouraud's agar. The isolation of the fungus is the only exact method of diagnosis. The fungus causes subacute or chronic granulomatous lesions usually on the exposed parts of the body. The primary lesion appears in the form of a rather uncharacteristic-looking abscess or ulcer which fails to heal. From this initial lesion the fungus invades the lymphatics, and a very typical clinical picture develops. Chronic lymphangitis with multiple subcutaneous abscesses along the lymph vessels. The abscesses rupture and chronic ulcers without tendency to healing appear. These ulcers are usually secondarily infected with staphylococci or other organisms, and if only the superficial exudate is taken for cultures, the lesions are frequently diagnosed wrongly. Pus or exudate from unbroken lesions should be cultured. A skin test with heat-killed suspension of the fungus shows a positive reaction of the tuberculin type after the first week of disease.

The infection responds to treatment with iodides (Lugol's) orally, but such treatment has to be continued over a period of months.

Blastomycosis.—This is a granulomatous disease of the skin or internal organs caused by *Blastomyces dermatitidis*. This fungus grows on the usual laboratory media and shows two types of growth, a yeastlike colony when grown at 37° C and a moldlike form at room temperature. *B. dermatitidis* frequently infects the lungs and can be mistaken for tuberculosis. Spread via the blood vessels occurs and metastatic abscesses appear in the skin, the bones, liver, spleen, and central nervous system. Primary infection of the skin consists of ulcers with a raised, irregular, papilliform border showing milium abscesses. The differential diagnostic considerations in both the cutaneous and pulmonary (systemic) forms comprise a number of other fungus infections, infectious and noninfectious granulomas, and tumors. The isolation and identification of the fungus from the exudate provide the only accurate means of diagnosis. For the cutaneous forms a combination of iodides, x-ray therapy, surgical procedures, and desensitization with vaccines has given the best results. The treatment of the systemic form has become more promising since the introduction

of the aromatic diamidines, stilbamidine, and 2-hydroxystilbamidine. In localized pulmonary blastomycosis, lung resection may be a valuable adjunct to treatment with stilbamidines (Curtis and Bocobo, 1957). The newly discovered fungistatic, amphotericin B, may revolutionize the treatment of this infection



Fig 10—Multiple cutaneous lesions caused by *Blastomycet dermatitis* showing raised irregular papilliform borders with milium abscesses

Coccidioidomycosis (Coccidioides immitis).—This is caused by *Coccidioides immitis*. The occurrence of this organism appears to be confined to certain endemic areas (San Joaquin Valley in California, parts of Texas, and Arizona, etc.), where it has been found in the soil and is transmitted by dust. In the majority of cases infection of the lung takes place, primary infection of the skin through wounds is rare. The upper respiratory infection of the acute type manifests itself with the signs and symptoms of a bronchopneumonia with fever and chills and is known as San Joaquin fever, or Valley fever, which is generally a nonfatal, self limited infection. Dissemination may occur and gives rise to the generalized form, known as coccidioidomycosis

loma, which resembles tuberculosis very closely, both on clinical and x-ray findings. Lesions may appear anywhere in the body, and the mortality rate is very high (50-60%). The diagnosis can be made by the cultivation of *C. immitis* from exudates or by the inoculation of mice and guinea pigs with pleural fluid, sputum, etc. However, the risk of infection to nonimmune laboratory personnel is so great that cultural isolation should be attempted only under special circumstances. A positive skin reaction of the tuberculin type to coccidioidin can be elicited by the third week following infection. In patients with disseminated disease the skin test may be negative. Complement fixation and precipitation tests have been developed and are used as diagnostic aids. In disseminated coccidioidomycosis all available forms of treatment must be tried: stilbamidines, amphotericin, and surgery. Winn (1957) discusses the present concept of the surgical management

Histoplasmosis.—This is essentially a disease of the reticuloendothelial system caused by *Histoplasma capsulatum*. On cultures this organism grows in two morphologic forms depending on the medium used and the temperature of incubation. On blood agar at 37° C and in tissues, *H. capsulatum* appears as small oval budding yeast cells which in tissue sections are found inside the cells. The natural reservoir of *H. capsulatum* is the soil, and all available evidence indicates that histoplasmosis is an air-borne infection. The majority of infections is not clinically apparent. This fact has been recognized only since the histoplasmin skin test has been extensively employed as an epidemiologic tool. The clinical manifestations of the active infection with the fungus are manifold, and in the differential diagnosis many other acute and chronic infections, tumors, and granulomas must be considered. Of special interest to the surgeon are the "coin" lesions and multiple calcifications due to healed histoplasmic infections. The diagnosis depends on the isolation of *H. capsulatum* from the peripheral blood, the bone marrow, lymph nodes, or skin ulcers, or on the demonstration of the organism in histologic sections. Skin sensitivity to histoplasmin develops during the third or fourth week after

infection A positive reaction has the same significance as a positive tuberculin test Complement fixing and precipitating antibodies can be demonstrated in the serum at about the same time Spontaneous recovery appears to be the rule and fortunately only the rare disseminated case ends fatally The experiences with amphotericin are too recent and few to permit any predictions of its effectiveness in the disseminated form, although promising results have been reported (Lehan, et al, 1957)

Cryptococcosis (Torulosis).—This fungus infection due to *Cryptococcus neoformans* shows a decided preference for the central nervous system *C. neoformans* is a budding yeastlike organism, showing both in cultures and tissues or exudates a large capsule *C. neoformans* can be easily cultured on ordinary laboratory media The central nervous system is the most frequently affected organ either primarily or secondarily from lung or skin lesions Clinically the infections resemble tuberculous meningitis, brain abscess, brain tumor, or syphilis The cutaneous lesions appear in the form of subcutaneous abscesses or tumors which break through the skin and form ulcers Generalized spread from the skin lesions almost invariably involves the central nervous system as well as the lungs, liver, spleen, etc The diagnosis is made by the demonstration of the fungus in exudates, by cultural isolation, or animal inoculation (mice). A drop of spinal fluid or pus or other exudates, suspected of containing *C. neoformans*, should be mixed with a drop of India ink on a slide under a cover slip When viewed under the microscope the fungus appears as a round, budding cell surrounded by a large capsule No generally effective treatment of cryptococcosis is known The central nervous form is almost invariably fatal Improvement in some cases has been reported after the use of iodides, sulfa drugs, and x-ray, but it is by no means consistent The new antifungal agent, amphotericin B, appears to offer some promise.

Other fungi may occasionally cause lesions which have to be differentiated from other infections For detailed information on fungus infections of medical importance, the student should consult special textbooks (Conant, et al, 1954)

MIXED INFECTIONS

This discussion has been concerned with the more common bacterial and fungal agents that cause infections of interest to the surgeon. Although the frequent involvement of more than one microbe in these infections has been mentioned in various connections, the great practical importance of mixed infections deserves emphasis A few examples might illustrate this point

Sputum or bronchial aspirations from bronchiectasis or lung abscesses yield as a rule a highly mixed flora, containing many aerobic as well as anaerobic species Even under optimal conditions not all species will be isolated, and those isolated cannot always be completely identified within a reasonable time. Spirochetes and fusiforms commonly present in such specimens can only be demonstrated on smears It is often difficult or impossible to determine the initial cause of the infection or to assess the exact role of the various species present The choice of a suitable chemotherapeutic agent can be based only on the predominating flora

The situation is similar in cases of peritonitis following rupture of the gut. Here, too, the great variety of organisms cultured makes exact identification and sensitivity testing of each species impractical If the infection remains localized, topical treatment with a mixture of bacitracin-neomycin will often be effective However, in severe infections systemic antibiotic treatment is indicated

Infected burns also present special features Large areas of the body are exposed to bacteria, and in addition to accepted pathogens, a number of "opportunists" may complicate the picture One of the principal surgical objectives is to avoid infection in burns of a limited area (e.g., of the hand by early excision of the burned area, about the third or fourth day, with immediate skin grafting) and to minimize and control the infective process in the more extensive burns A word of caution is in order here on the use of corticosteroid hormones in these cases The general state of the patient may make their use imperative, but in such cases the surgeon must be on guard against possible spread of the infection and must administer systemic as well as local treatment with a suitable, eff

CONCLUDING REMARKS

In the diagnosis of infections the proper taking of specimens is of the utmost importance. Even the best bacteriologic laboratory can only isolate the flora in the specimen received. Certain principles have to be kept in mind to make a quick and accurate diagnosis possible, and the physician and surgeon have to contribute their parts.

1. The introduction of contaminating organisms must be prevented, instruments used for taking the specimens and the containers must be sterile.

2. Specimens should be taken from the freshest lesions, and the site of progressive infection, e.g., from the edges of ulcers, from unopened abscesses, rather than broken ones, whenever possible.

3. Sufficient material should be collected, so far as feasible, if various examinations are required or when the chances of finding the causative organism increase (up to a point) with the amount of material available (e.g., 24-hour specimens of urine for the demonstration of tubercle bacilli).

4. Information on the clinical condition of the patient and on any treatment received has to be given to the laboratory to aid in the selection of suitable media and techniques; whenever possible specimens should be collected before treatment is started. In problem cases the collaboration of the surgeon and laboratory chief is essential for results and their evaluation.

5. Last but not least, specimens should reach the laboratory as soon as possible after they have been obtained, to prevent drying or other injuries to fastidious organisms.

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- vascular system
- psychologic stimuli, e.g., pain,
- anesthetic agents
- procedures, e.g., thoracic operations
- women, e.g., perforated peptic ulcer
- dry
- lesions, e.g., mitral and aortic
- lesions, e.g., tamponade
- lesions, e.g., infarct
- ias, e.g., paroxysmal tachycardia

Physiopathology

Due to fluid loss there is a decrease in output resulting from inadequate contraction of the ventricles because of a decrease in venous return. There is no significant change in hemoglobin or hematocrit levels during hemorrhage, except hours later. However, the reduction in blood volume and plasma loss or dehydration, there is a reduction early.

Metabolic changes in shock are characterized by anoxia resulting from deficient tissue perfusion. There is an acidosis with a fall in pH. Impaired renal function is manifested by a rise in plasma NPN and a demineralization of urine. Acute renal failure, or lower nephron nephrosis, may also occur as discussed later. Impairment of tissue perfusion may occur with serious consequences in terms of its role in protection against sepsis as mentioned elsewhere.

Experimentally it has been shown that the liver also contributes to the state of shock by the elaboration of humoral factors. In profound shock the kidney produces a constrictor principle called VEM* and also a vasodilator material called VDM. As shock approaches the stage of irreversible shock, VDM appears in the blood in large amounts. In animals made resistant to shock by previous graded exposures, there is a decrease in the blood VDM.

The brain is very susceptible to anoxia. If the anoxia is prolonged or severe, there may be a period of apparent recovery, coma, convulsions, and death.

There are also compensatory changes consisting of vasoconstriction and hemodilution.

EM = vasoconstrictor material.

The effect of the former is to reduce the size of the vascular bed by selective vasoconstriction in the skin and kidneys. It has been estimated that approximately 500 ml of blood normally contained in the vessels of the skin are thereby made available for vital organs such as the brain. Vasoconstriction in the vessels of the kidneys may have a damaging effect as mentioned above. Vasoconstriction is also important in that, by maintaining blood pressure at or near normal levels, it may mask severe hemorrhage and impending shock. In severe or untreated cases of shock, the mechanism ultimately fails.

A less effective compensatory mechanism is hemodilution. By this means the reduced blood volume is partially restored by passage of water and electrolytes from tissue spaces into the blood stream. Hemodilution may not occur in dehydration or in severe anemia. Vasoconstriction and hemodilution cannot be relied upon, therefore, to maintain an adequate blood volume in cases in which hemorrhage has been severe or prolonged.

Pathologic Anatomy

Except for the characteristic findings of the complications and consequences of shock, i.e., acute renal failure, myocardial infarction, etc., to be discussed later, there are no abnormal changes. The organs may be pale or plethoric, depending on the amount of blood administered.

Diagnosis

The clinical features of shock—the signs and symptoms such as restlessness, subnormal temperature, rapid pulse, pallor, sweating, cold extremities, and low blood pressure, and the metabolic and other changes already described—are those of the fully developed state. It is the early recognition of the causes of reduction in blood volume that should be the aim of diagnosis in order to prevent shock and its grave effects.

The most common cause of reduction in blood volume is hemorrhage. The hemorrhage may be either external or internal. It may be traumatic; it may occur during or following surgery; or it may be spontaneous, e.g., peptic ulcer, ruptured ectopic pregnancy, or purpura.

Shock and Blood Transfusion

Paul G. Weil, MD.

SHOCK

No subject in surgery is of greater importance than shock. Despite a better understanding of its causes and treatment gained from the many investigations of the problem and by the development of blood banks and transfusion services during recent years, shock still remains a dreaded complication of surgical operations and trauma.

Shock, also known as *collapse* or *peripheral circulatory failure*, may be defined as the syndrome characterized by subnormal temperature, pallor, cyanosis, sweating, cold extremities, thirst, low urine output, and low blood pressure due to a reduction in blood volume usually the result of hemorrhage.

Until a few years ago, shock perhaps even excelled eclampsia as the "disease of theories." There was general agreement that the main factor in shock was an acute reduction in blood volume, but there was, however, a difference of opinion on the cause of the decrease in blood volume.

Theories

The principal theories and those on which present day management of shock depends are as follows:

1. *Neurogenic*. Nervous impulses originating from tissue damaged by trauma or at operation cause a dilatation of the vascular system, leading to a reduction in effective blood volume.

2. *Toxic*. Noxious substances liberated by damaged tissue increase the permeability of capillaries, leading to loss of plasma from the circulation, reduction in blood volume, and hemoconcentration, i.e., a relative increase in the cellular elements of the blood.

3. *Hemorrhagic*. The reduction in blood volume is due to hemorrhage resulting from trauma or operation or occurring spontaneously.

These conflicting theories have recently been resolved, and the following concepts are those now generally held. *Primary*, or *neurogenic*, shock is due to a dilatation of the vascular system. *Secondary*, or *oligemic*, shock, also known as *traumatic* and *surgical shock*, is due to a reduction in blood volume resulting from fluid loss.

The shock syndrome may also result from failure of the heart itself, so called *cardio-circulatory shock*. Various classifications of shock have been used. The one that follows is based on the mechanisms of its development.

1. Reduced blood volume
 - A. Loss of blood
 - Internal e.g., ruptured spleen
 - External e.g., traumatic amputation
 - B. Loss of plasma
 - Internal e.g., peritonitis
 - External e.g., burns, crushing injuries
 - C. Loss of water and electrolytes (dehydration) e.g., gastroenteritis

II. Dilatation of vascular system

- A. Physical and psychologic stimuli, e.g., pain, anxiety
- B. Drugs, e.g., anesthetic agents
- C. Surgical procedures, e.g., thoracic operations
- D. Acute abdomen, e.g., perforated peptic ulcer
- E. Infections

III. Cardiocirculatory

- A. Endocardial lesions, e.g., mitral and aortic stenosis
- B. Pericardial lesions, e.g., tamponade
- C. Myocardial lesions, e.g., infarct
- D. Arrhythmias, e.g., paroxysmal tachycardia

Physiopathology

In shock due to fluid loss there is a decreased cardiac output resulting from inadequate filling of the ventricles because of a diminished venous return. There is no significant change in hemoglobin or hematocrit levels in shock from hemorrhage, except hours later. When, however, the reduction in blood volume is due to plasma loss or dehydration, there is hemoconcentration early.

The metabolic changes in shock are consequent to the anoxia resulting from deficient circulation. There is an acidosis with a fall in CO_2 . An impaired renal function is manifested by a rise in plasma NPN and a decreased formation of urine. Acute renal failure, the so-called lower nephron nephrosis, may supervene as discussed later. Impairment of liver function may occur with serious consequences in terms of its role in protection against sepsis as mentioned elsewhere.

Experimentally it has been shown that the kidney and liver also contribute to the state of shock by the elaboration of humoral factors. In profound shock the kidney produces a vasoconstrictor principle called VEM* and the liver, a vasodilator material called VDM. As the animal approaches the stage of irreversible shock, VDM appears in the blood in increasing amounts. In animals made resistant to damage by previous graded exposures, there is a decrease in the blood VDM.

The brain is very susceptible to anoxia. If shock is prolonged or severe, there may occur after a period of apparent recovery, *coma*, *convulsions*, and *death*.

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The effect of the former is to reduce the size of the vascular bed by selective vasoconstriction in the skin and kidneys. It has been estimated that approximately 500 ml. of blood normally contained in the vessels of the skin are thereby made available for vital organs such as the brain. Vasoconstriction in the vessels of the kidneys may have a damaging effect as mentioned above. Vasoconstriction is also important in that, by maintaining blood pressure at or near normal levels, it may mask severe hemorrhage and impending shock. In severe or untreated cases of shock, the mechanism ultimately fails.

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Pathologic Anatomy

Except for the characteristic findings of the complications and consequences of shock, i.e., acute renal failure, myocardial infarction, etc., to be discussed later, there are no abnormal changes. The organs may be pale or plethoric, depending on the amount of blood administered.

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The clinical features of shock—the signs and symptoms such as restlessness, subnormal temperature, rapid pulse, pallor, sweating, cold extremities, and low blood pressure, and the metabolic and other changes already described—are those of the fully developed state. It is the *early recognition* of the causes of reduction in blood volume that should be the aim of diagnosis in order to prevent shock and its grave effects.

The most common cause of reduction in blood volume is *hemorrhage*. The hemorrhage may be either external or internal. It may be due to trauma; it may occur during or following operation; or it may be spontaneous, e.g., from a peptic ulcer, ruptured ectopic pregnancy, thrombocytopenic purpura.

*VEM = vasoeffecter material

The large amounts of blood that may be lost in traumatic injuries or at operation have been shown in numerous studies. It can be anticipated that wounds which are caused by violent force and which result in extensive injury will be accompanied by shock.

Loss of blood into the soft tissues, body cavities, or gastrointestinal tract is difficult to evaluate. The swelling of soft tissues or the presence of dullness on percussion over body cavities is useful as an indication of the extent of hemorrhage.

Once shock has developed, the only reliable clinical guide is the level of the blood pressure (In individuals with hypertensive disease, the blood pressure may not fall below the normal range.) The pulse rate is not always increased. Blood studies such as hemoglobin or hematocrit determinations are of no use inasmuch as they do not reflect the changes in blood volume in hemorrhage. Blood volume estimations may be valuable in complicated cases, especially if a preoperative determination has been made.

Reduction of blood volume from *loss of plasma* most commonly occurs in cases of burns. Plasma is lost from damaged capillaries in and adjacent to the burned areas. The loss of plasma leads to hemoconcentration. In intestinal obstruction, especially when accompanied by local circulatory stasis as in mesenteric thrombosis, there may be a diffusion of plasma into the peritoneal cavity and bowel lumen with a resultant reduction in blood volume. The crush syndrome follows compression injuries to the extremities. When the compression is released, plasma is lost into the damaged tissues, and shock develops.

In conditions leading to *dehydration*, such as excessive vomiting, diarrhea, gastrointestinal fistulas, there is a reduction in blood volume due to loss of water and electrolytes from the blood.

Although of no use in hemorrhagic shock, the determination of the degree of hemoconcentration by hemoglobin or hematocrit studies is of great value in estimating the degree of reduction of blood volume from loss of plasma (or in dehydration) and in determining the amount of plasma (or saline) to be infused.

Shock resulting from a disturbance of the nervous regulation of vascular tone is next in importance to that due to hemorrhage. Various stimuli, psychic and physical, drugs, especially anesthetic agents, surgical procedures, and infections may cause peripheral vascular dilatation.

Cardiocirculatory or cardiogenic collapse is due to failure of the heart itself. Cardiac tamponade resulting from acute pericardial effusion or hemorrhage causes a characteristic syndrome of shock, venous engorgement, and dyspnea. Myocardial infarction is often accompanied by shock. The arrhythmias, paroxysmal tachycardia, and heart block may cause peripheral circulatory failure.

In cases of severe mitral or aortic stenosis sudden strain or the stress of major surgery may cause collapse due to the inability of the damaged heart to put out more blood on demand. The diagnosis of shock due to these causes depends on the history and physical and electrocardiographic findings.

Prophylaxis

Patients in shock withstand operative procedures poorly. As a rule, surgical measures should not be undertaken until shock has been relieved. When immediate operation is imperative to control hemorrhage or for other urgent reasons, transfusion should be started at once and continued throughout the operation.

In many patients with chronic disease there is often an associated anemia due to various causes, including malignancy, infection, and malnutrition. In such patients there is usually a reduction in total blood volume because the reduction in the red cell mass may be only partially compensated for by an increased plasma volume. The decrease in both the volume and particularly the oxygen-carrying capacity of the blood must be restored by transfusions of whole blood before major operations are performed on such patients. The usual practice is to give 500 to 1,000 ml. of blood, the amount depending on the degree of anemia, on each of several successive days immediately prior to the day of operation. Red cell suspensions as described later may also be used.

The detection of hemorrhagic tendencies, especially in diseases of the liver and gall bladder, by coagulation tests, e.g., prothrombin determination, and their correction will be discussed later.

The choice of an *anesthetic* demands great care when shock is either present or anticipated. Oxygen should be given continuously during anesthesia in all cases of shock to avoid anoxia. It is the responsibility of the anesthesiologist to watch for the early signs of shock in all operations, looking particularly for falling blood pressure. Blood pressure readings should be made repeatedly during the opera-

Shock may manifest itself not only during or at the end of operation but also at any time after the operation—even hours later. When shock occurs several hours after the end of an operation, it may be due to hemorrhage from a loose ligature. In such cases, transfusions will have only a temporary effect in restoring the blood pressure. Serious consideration must be given, therefore, to immediate *reoperation* for control of hemorrhage in all patients who develop shock following operation and in whom the blood pressure cannot be restored or maintained by transfusions.

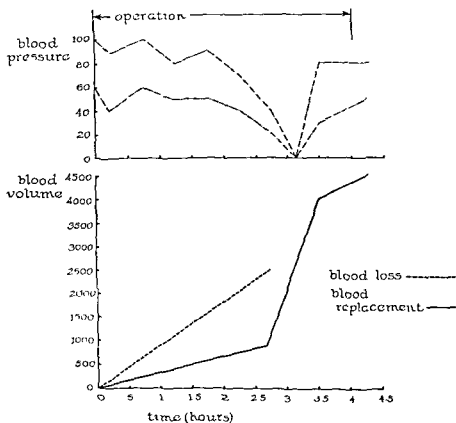


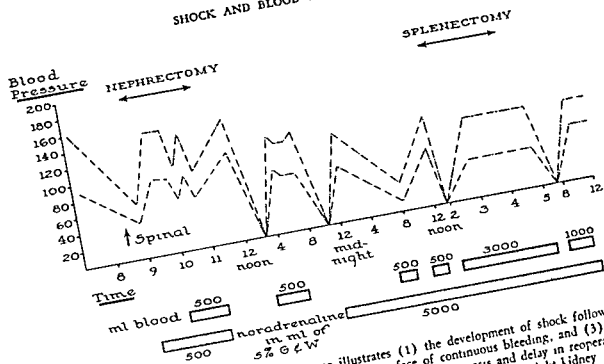
Fig 11—Illustrates (1) the large amount of blood that may be lost at operation, in this case a pneumonectomy for pulmonary tuberculosis; (2) the development of shock during operation because of failure to maintain blood replacement *pari passu* with blood loss which was measured by the Wangenstein method, and (3) the proper treatment of shock by immediate, rapid, and adequate replacement of blood

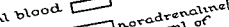
tion and at frequent intervals during the immediate postoperative period

Careful *surgical technique* both in the gentle handling of tissue and in the use of hemostasis is important in minimizing hemorrhage.

Treatment

The treatment of shock consists principally in the replacement of the fluid lost and, to be discussed later, the use of vasoconstrictor drugs and hormones in cases in which vaso-



Time 





ml blood  noradrenaline 
 in ml of 
 5% G & W

Fig. 12—Postoperative shock. This case illustrates (1) the development of shock following operation, (2) the transient effect of transfusion in the face of continuous bleeding, and (3) the necessity for reoperation to control hemorrhage. The difficulty in diagnosis and delay in reoperation were due to the fact that the splenic vessels were torn during operation on the right kidney.

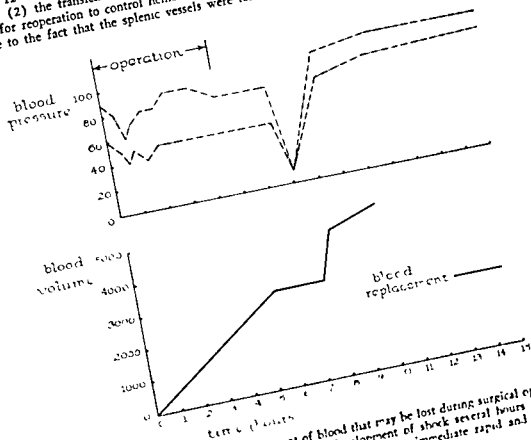


Fig. 15.—Illustrates (1) the large amount of blood that may be lost during surgical operation, in this case removal of mediastinal tumor, (2) the development of shock several hours after the end of the operation, and (3) the proper treatment of shock by immediate rapid and adequate blood transfusion

dilatation is a factor. In *hemorrhagic shock* there is no substitute for *whole blood*. Plasma transfusion, although it restores blood volume in this condition, does not improve the reduced oxygen-carrying capacity of the blood. The use of plasma or plasma substitutes in shock from hemorrhage should be confined to the emergency measure of restoring or maintaining blood volume during the period of delay necessary to the starting of a whole blood transfusion or in supplementing blood transfusion if supplies of blood are limited

Whole Blood Transfusion.—There are several devices frequently used for the rapid restoration and, with continuing blood loss, the maintenance of normal blood volume. Blood may be transfused into *several veins simultaneously*. It may be *forced in* by attaching the pump of a blood pressure apparatus to the air inlet tube of the transfusion bottle and applying pressure, care being taken to prevent the entrance of air into the flow of blood. In the severely shocked patient, with cold extremities, the peripheral veins are usu-

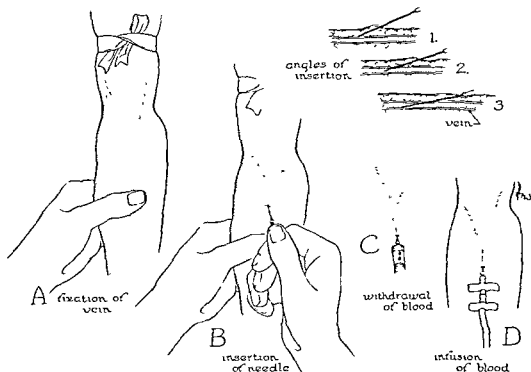


Fig 14—Technique of venipuncture.

Just as important as knowing *what to give* is knowing *how to give it*. In shock from hemorrhage, blood must be given *immediately*, and it must be given *rapidly* and often in *large quantities*, of the order of 5 to 10 liters. These three aspects of the treatment of shock cannot be overemphasized. They are determined not by one's impression of the amount of blood lost (which is usually an underestimation), or by the large amount which may have already been administered, but only by the response of the patient to transfusion as indicated by the level of the arterial blood pressure

ally contracted. Dilatation of the veins may be induced by the *application of heat* to the extremity to be used for transfusion. Since, however, time is of the essence in the treatment of shock, at the first indication that the collapsed state of the veins may delay a successful venipuncture, a "cut-down" should be started at once.

The technique of the "cut-down" is as follows. The site usually chosen is the ankle, although any of the larger veins in the arm may be used. If the ankle is chosen, the vein may be found lying immediately above and in front of the medial malleolus. Under local

SHOCK AND BLOOD TRANSFUSION

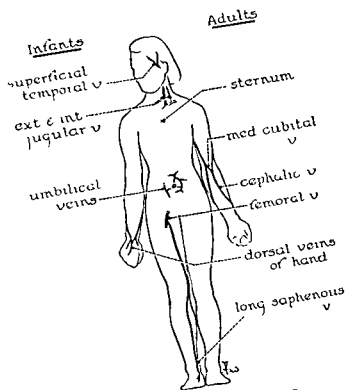
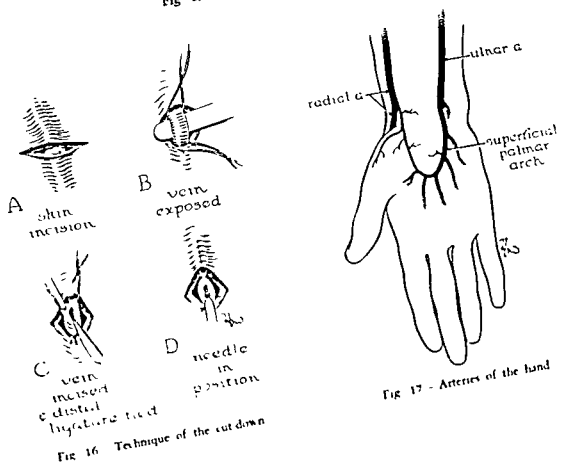


Fig 15—Routes for transfusion



anesthesia the vein is exposed with blunt dissection. It is separated from the surrounding tissues by placing the handle of a scalpel or other flat instrument between the undersurface of the vein and the skin. Two ligatures are then drawn between the vein and the skin; one is placed distally and tied around the vein; the proximal ligature is left untied. A small incision is made in the anterior wall of the vein between the two ligatures with a pair of scissors or other sharp-pointed instrument. A Lindemann needle or cannula is then inserted proximally for some distance into the vein and tied securely in place by means of the second ligature. The needle or cannula may be immediately connected to a transfusion set or kept patent by the attachment of a slow-drip intravenous solution of saline.

Intra-arterial transfusion has recently been introduced as a method to be used in profuse hemorrhage or profound shock when restoration of the blood volume by the intravenous route is ineffective. The transfusion is usually administered via one of the arteries of the hand,* most often the radial. By means of pressure, blood may be forced retrograde into the arterial system so as to fill rapidly the renal, coronary, and cerebral vessels simultaneously with the restoration of blood volume. Recent studies indicate, however, that the important factor is the rate rather than the route of transfusion.

Plasma Transfusion.—The treatment of shock due to a reduction in blood volume from loss of plasma as in burns, intestinal obstruction, or peritonitis is the infusion of plasma, albumin, whole blood, or plasma expanders. Dehydration due to vomiting in such cases must be treated with saline infusion. When present, infection, which may cause vasodilatation, must be treated with antibiotic and vasopressor drugs.

The circulatory collapse that may supervene with *dehydration*, in patients with the low-salt syndrome, in diabetic coma, the crisis of adrenocortical insufficiency, or acute gastroenteritis, should be treated with saline infusions, blood transfusion, and the specific hormone and antibiotic indicated. Vasopressor drugs may also be useful.

*In a right-handed patient, intra-arterial transfusion should be given in the left hand.

Vasoconstrictors.—The decline in blood pressure that may result from spinal anesthesia is prevented by using ephedrine before anesthesia, and it is treated by using Neo-Synephrine or other vasoconstrictor agents. Norepinephrine is used rather than epinephrine because of the danger of the latter drug causing cardiac arrhythmia in the presence of anesthesia and because of its constriction of the coronary vessels. Vasoconstrictor drugs are also used more and more frequently to maintain normal blood pressure postoperatively and to restore blood pressure in cases of shock due to blood loss.

Collapse may be severe in fulminating infections, in contaminated wounds of the abdominal cavity or perineal region, and following transfusion of contaminated blood. The intravenous and intramuscular injection of wide-spectrum antibiotics early and in large dosage is imperative. Replacement of blood, plasma, and electrolytes and the use of vasoconstrictors, as in the treatment of shock due to other causes, are also required.

Transfusions are usually contraindicated in the treatment of cardiogenic shock. Cardiac tamponade is treated by pericardial paracentesis or thoracotomy. The shock accompanying myocardial infarction is treated with norepinephrine. Therapy of the arrhythmias that may result in shock is described fully in other texts.

Adjuvant Measures.—Although transfusion is the main measure in the therapy of secondary shock, there are other adjuvant measures which have a valuable place in its treatment.

1. The control of hemorrhage is obviously of utmost importance.

2. **Vasoconstrictor drugs** are valuable agents in counteracting vasodilatation and in restoring the reduction in effective blood volume in cases of neurogenic shock. Their use in cases of secondary shock is less satisfactory. Since, however, there may be an element of neurogenic shock in cases of hemorrhage, it is often desirable to use them in certain circumstances, i.e., trauma, or infection accompanying hemorrhage. The use of adrenergic blocking agents such as chlorpromazine, which are beneficial in the prevention of shock in the experimental animal, has its rationale in their vasodilating effect on the vessels of kidney and other vital

SHOCK AND BLOOD TRANSFUSION

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organs whose blood supply in prolonged shock may be greatly impaired by extreme vasoconstriction. Their clinical use, however, is still *sub judice*.

3. Immobilization of fractures is an important measure, preventing further trauma at the fracture site.

4. The administration of oxygen is beneficial in cases complicated by cardiopulmonary disease or injury, and during anesthesia.

5. Apprehension, pain, and fatigue must be allayed because they may induce vasodilatation. Morphine, judiciously used, is the drug of choice.

that the amount of blood shifted from the lower parts of the body to its center improves the vasomotor status. The shock position may be inadvisable in patients with cerebral or cardiopulmonary complications.

8 The early use of wide-spectrum antibiotics in large amounts in the presence of infection or contaminated wounds is of paramount importance.

9. ACTH and cortisone have special value in the treatment of shock in the following circumstances:

- a Operations involving removal or interference with secretion of pituitary and adrenal glands, e.g., Cushing's disease
- b Operations on patients who have been under treatment with cortisone for a long time
- c Addison's disease

In one type of case the sudden withdrawal of adrenocortical secretion may precipitate collapse. In the other type, patients whose adrenocortical activity has been suppressed by long, continuous treatment demand of the sudden stress of operation. Peripheral circulatory collapse and hypoglycemia result. In both cases, treatment consists of giving cortisone in adequate doses 100-200 mg. daily before and after operation, gradually reducing the dosage in the postoperative period. During operation it may be necessary to treat with intravenous cortisone. Blood transfusions and vasoconstrictors are also indicated.

The occurrence of collapse in patients with adrenocortical insufficiency when infection or trauma supervenes is treated with cortisone intramuscularly and intravenously. As already mentioned above, shock may also occur in patients under long-term treatment with cortisone who develop infection or receive an injury or undergo an operation. Such an eventuality must be avoided in patients with arthritis, asthma, or dermatitis or in others receiving cortisone for long periods. Evidence of its use must be specifically sought in taking the history of such patients prior to operation. Under such circumstances, as well as during the course of any intervening illness, the cortisone should be increased. In all such cases when shock is either present or may be anticipated, it is well to support the circulation by using norepinephrine or other vasoconstrictor.

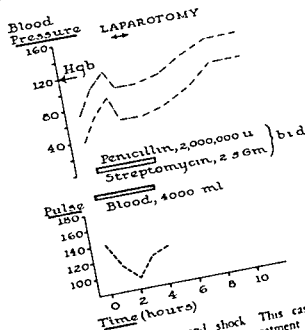


Fig. 18—Infection and shock. This case illustrates the importance of adequate treatment of infection as well as shock which was due to peritonitis following perforation of a duodenal ulcer one week previously. At operation 1 liter of pus were found within the abdomen, which accounted for the hemoglobin concentration (Hgb 120%)

6 Body temperature should be maintained but the excessive application of heat, an effective vasodilator, by relaxing compensatory vasoconstriction may precipitate shock.

7 Because of vasomotor instability, the patient who is bleeding or already in shock should be moved as little as possible. Flexion of the foot of the bed in patients with a moderate reduction in blood volume causes a definite increase in blood pressure, indicating

and to give blood transfusions during the critical phases.

Chronically ill patients may not respond well to an added stress because of a relative adrenocortical insufficiency, and an operation may precipitate collapse. In such cases ACTH and cortisone should be used on an empiric basis along with transfusions and vasoconstrictors to restore the blood pressure.

Complications and Consequences

The consequences of *severe or untreated shock* are several. Some of the more serious of them are acute renal failure, myocardial infarction, cerebral thrombosis, and irreversible or delayed shock. Myocardial infarction and cerebral thrombosis are more likely to occur in the elderly and in individuals with coronary or cerebral arteriosclerosis.

Delayed shock is a term used to describe the fatal form in which, after restoration of normal blood pressure by transfusions following an initial period of severe or prolonged hypotension, the blood pressure steadily declines, and the patient dies even though the blood volume may be normal. The cause is unknown, but severe infection and massive hemorrhage inadequately treated either separately or in combination are important contributory factors.

The term *irreversible shock* has crept into clinical usage where it has no place. It belongs in the experimentalist's vocabulary where it means that shock has been produced in such a manner that precludes the animal's survival from hemorrhage. The term has often been used to describe therapeutic failure in patients who have been treated for shock. Its clinical usage should be discouraged, not only for the reasons just mentioned, but because it is dangerous since it may lead to the neglect of opportunity for effective therapy.

In conclusion, it is worth while to repeat that blood must be given as soon as possible after hemorrhage, and it must be given rapidly and in adequate amounts. The view that blood transfusion by raising the blood pressure may increase or cause a resumption of bleeding is entirely unfounded, and if transfusion is for this reason withheld, the results may be fatal. In cases of massive hemorrhage requiring large

amounts of blood to restore blood pressure to normal, inadequate amounts may be given for fear of overloading the circulation. Such fears are likewise unfounded if the level of the blood pressure is used as the guide for determining the amount of blood to be used. Only after the blood volume and blood pressure have been restored to normal is it likely that further transfusion will overload the circulation. When the normal blood volume as reflected by a normal blood pressure has been achieved and maintained, the need for rapid transfusion no longer exists. The transfusion may then be continued at a slower rate. Signs of increasing the blood volume beyond normal may be found by inspecting the neck veins for fullness and by auscultating the chest for râles at the bases. More deaths result from transfusion given "too little and too late" than from circulatory overloading.

The treatment of shock because of its emergency nature has been compared with the treatment of diabetic coma in that it is not enough to start treatment, write orders, and return later to see how the patient is progressing. There is probably no other condition that demands such resourcefulness, close supervision, and sense of urgency during the entire period of treatment. The effects of anoxia are so serious that in profound shock the period during which transfusion may be effective is often measured only in minutes, almost never in hours.

BLOOD TRANSFUSION

The recent advances in the recognition and treatment of shock and the development of blood banks and transfusion services have contributed greatly to the progress of surgery. They have become important factors both in the performance of operations and in the recovery from injuries in which the amount of blood lost may be considerable. Shock has been discussed in the preceding section.

Blood transfusion may be considered under five main headings:

1. Biochemical aspects
2. Immunologic aspects
3. Therapeutic aspects
4. Reactions
5. The blood bank and transfusion services

Biochemical Aspects

The constituents of whole blood which are of chief importance in transfusion therapy are the erythrocytes and the plasma. The solutions used for the preservation of blood have been designed for the best preservation of the erythrocytes without too great dilution of the whole blood mixture. The solution includes

Under the ordinary storage conditions of the blood bank, blood may be used up to three weeks following donation. Maximum benefit is obtained by the transfusion of fresh blood. The rate of loss of viability of erythrocytes in preserved blood is approximately 1% of the cells per day, but after three weeks they deteriorate more rapidly. Blood which has not been used by the end of this period is set aside

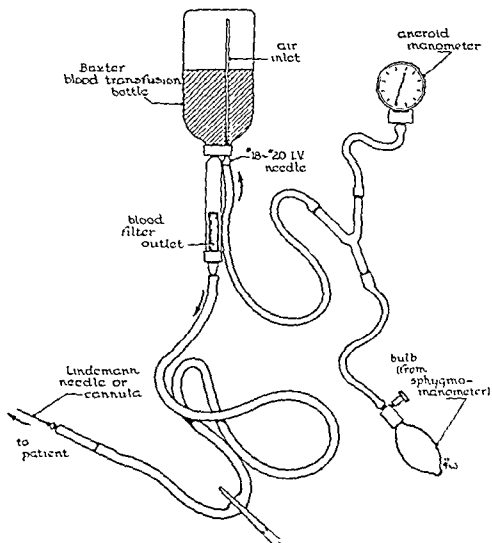


Fig 19—Blood transfusion apparatus with pressure connection for rapid intravenous or intra-arterial transfusion

sodium citrate as anticoagulant and dextrose as preservative. The proportion of whole blood to solution of anticoagulant and preservative in the average bottle of blood is approximately three parts whole blood to one part diluent. As an additional preservative measure, blood must be stored at a temperature of 4° C. Freezing and thawing destroy the erythrocytes

to permit the sedimentation of the cellular elements. The supernatant plasma is then drawn off and stored. The cells are discarded.

Plasma can now be separated at the various commercial and government-operated laboratories into several components, chiefly albumin, fibrinogen, and gamma globulin. The use of plasma as a blood substitute is diminishing be-

cause of (1) the development of plasma expanders, (2) certain dangers inherent in plasma infusion,* and (3) the waste of gamma globulin, fibrinogen, etc., when whole plasma is used for the treatment of shock.

Albumin is the most important constituent of plasma from the standpoint of colloid osmotic properties. Its administration provides a rapid method of treating shock because it quickly shifts extravascular fluid into the circulation. It must be used cautiously in patients suspected of suffering from dehydration as well as shock.

The other important plasma fraction used in rare cases of shock is fibrinogen. More and more cases of bleeding due to a coagulation defect resulting from hypofibrinogenemia are being reported. Fibrinogen administration is often the only means of restoring the fibrinogen level to normal and of correcting the coagulation defect in cases of hypofibrinogenemia. The diagnosis of hypofibrinogenemia must be considered in the presence of hemorrhage occurring in cases of metastatic carcinoma of the prostate and pancreas, severe liver disease, and toxemia. It is also to be suspected in severe hemorrhage occurring during major lung surgery. The chief conditions, however, in which it may occur are missed abortion, e.g., Rh isosensitization with a dead fetus, and premature separation of the placenta at term, e.g., abruptio placentae.

Clinically the condition should be suspected in obstetric patients, of the type just described, who continue to bleed from the vagina despite repeated transfusions of even freshly drawn blood and other measures usually employed to stop hemorrhage. The patient's blood can be tested for fibrinogenopenia by the following procedure:

- (1) Withdraw 3 ml of blood
- (2) Allow to stand for 5 minutes.
- (3) If no clot appears, the fibrinogen level is at a critically low level

*Blood may contain the virus of hepatitis. Pooled plasma prepared from several donations therefore increases the risk of transmission of the virus. Although plasma stored at room temperature for six months is said to be safe in this respect, there is the possibility that it may contain bacterial contaminants which produce a potent toxin in lethal amounts over such a long period.

Fibrinogen is now commercially available. Every maternity hospital should maintain an adequate stock for emergency use. Bleeding can usually be controlled by the infusion of 4 Gm. Since fibrinogen prepared from plasma, which may contain the hepatitis virus, carries the virus with it in an active state, it should not be used unless absolutely indicated.*

Platelets (and leukocytes) disintegrate rapidly in stored blood unless special measures are taken to preserve them intact. The anaphilic and labile factors also disappear rapidly in stored blood or plasma. Prothrombin is fairly well maintained in bank blood during the first week of storage.

The treatment of shock due to hemorrhage resulting from deficient platelets has recently been improved by the introduction of methods for the preservation of platelets for transfusion. Even fresh blood drawn in ordinary bottles is said not to contain sufficient thrombocytes. Hemorrhage from thrombocytopenia is often uncontrollable by ordinary transfusions. If blood is drawn into plastic bags or glass containers coated with silicone, the platelets may be preserved long enough to effect cessation of bleeding in such cases if the blood is immediately transfused. A simple procedure used at the Royal Victoria Hospital is as follows:

- (1) The patient's blood is grouped
- (2) A blood sample is obtained from a donor of the same group as the patient
- (3) The donor's and patient's blood is cross-matched
- (4) If compatible, the donor is bled into a Fenwal plastic bag or a Baxter bottle coated with silicone
- (5) The patient is transfused immediately after the donation has been completed

The coagulation process and the hemorrhagic disorders and their treatment are summarized in Table 2.

The following cases illustrate the facts summarized in Table 2.

CASE 1. THROMBOCYTOPENIC PURPURA.—A 32-year-old woman was transferred to the Royal Victoria Hospital from another hospital for treatment of

*The prophylactic use of gamma globulin in cases where fibrinogen has been administered is recommended.

SHOCK AND BLOOD TRANSFUSION

TABLE 2

THE COAGULATION PROCESS		HEMORRHAGIC DISORDERS	TRANSFUSION TREATMENT
1 Platelets + AHG, PTA, PTC	Thromboplastin	Thrombocytopenic purpura	Silicone blood
Foreign surface		Hemophilias	Fresh plasma
2 Thromboplastin + Prothrombin	Thrombin	Hypoprothrombinemias	Fresh blood
Calcium + LF + SF		Hypofibrinogenemias	Fresh plasma
3 Thrombin + Fibrinogen	Fibrin		Fibrinogen
			Whole blood

AHG = Antihemophilic globulin PTA = Plasma thromboplastic antecedent. PTC = Plasma thrombotic component LF = Labile factor SF = Stable factor

acute renal failure on April 13, 1957. On admission she was critically ill, febrile (102° F), dehydrated, with generalized tremors and purpura. The history revealed that on March 30 she had sustained a perforation of the uterus when an attempt was made to empty the uterus of a 4-month pregnancy. The following day laparotomy was performed, and the abdominal cavity was found to contain a dead fetus and about 4,000 ml of bloody fluid, which, together with the uterus, were removed. The laboratory findings on admission included NPN 221 mg %, platelets scanty, WBC, 45,000, urine specific gravity 1.020, albumin 1+, numerous RBC and occasional WBC, rare granular cast, volume 2,000 ml per 24 hours. The serum Na, K, Cl, and CO₂ were within normal limits. For the next few days the patient remained in a critical condition, with evidence of bleeding into the gastrointestinal tract. Treatment consisted of Chloromycetin, 500 mg b.i.d., for the peritonitis, maintenance of normal fluid and electrolyte balance, and the transfusion of platelet-rich blood and platelet concentrates. She gradually improved, and by May 1 the NPN had declined to 38 mg % and the platelets had become abundant. During this time she had received 7 bottles of platelet-preserved blood (500 ml each) and platelet concentrates prepared from 9 bottles of blood.

CASE 2 HEMOPHILIA.—A 10-year-old boy, a known hemophilic, was transferred to the Montreal Neurological Institute January 18, 1957, from the Montreal Children's Hospital where he had been admitted January 16 following a fall the previous day. He was transfused with several units of plasma and transferred to the Montreal Neurological Institute because of developing paresis. At operation compression of the cord due to an epidural hematoma was found. The coagulation time was markedly prolonged on admission. He was transfused with 400 ml of fresh plasma and 3,000 ml of blood during the operation. Following operation he received daily infusion of fresh blood because of continuous oozing from the wound, hematuria, and epistaxis. Coagulation time throughout his stay in hospital remained prolonged and he developed a circulating anticoagulant.

At the time of discharge the wound had healed and there were no further hemorrhagic manifestations despite the prolonged coagulation time. During his hospitalization of 10 weeks he received 30 transfusions of fresh blood (500 ml each) plus 36 transfusions of fresh plasma (150 ml each).

CASE 3 HYPOPROTHROMBINEMIA.—A 71-year-old man was admitted on January 20, 1957, to the Royal Victoria Hospital with a history of jaundice of one month's duration. After investigation of liver function and coagulation studies, he was prepared for laparotomy. As his prothrombic activity was markedly impaired (25% of normal), he was treated with vitamin K and transfusion (1,000 ml) of fresh blood preoperatively. A stone in the common duct and a pathologic gall bladder were removed. Postoperatively he developed shock due to profuse hemorrhage and required 8 bottles of blood (4,000 ml). During the next few weeks his prothrombic activity averaged 40% of normal, and the deficiency was found to reside in the proconvertin fraction (stable factor). He was treated with 8 more transfusions of fresh blood before his discharge from hospital.

CASE 4 HYPOFIBRINOGENEMIA.—A 41-year-old woman, group O Rh negative, nonisensitized para IV, was admitted to the Royal Victoria Montreal Maternity Hospital bleeding from a placenta previa. After losing a small amount of blood during the afternoon she was delivered at 5:15 p.m. of a normal infant per vaginam with low forceps, there followed at 5:55 p.m. At delivery there was a loss of approximately 600 ml of blood from the placental site controlled by manual pressure. She was transfused with 1,000 ml of blood. Ergo metrine was administered, bleeding continued, low fibrinogenemia was diagnosed, and preparations for an immediate hysterectomy were made, during which time she was transfused with 2 Gm of fibrinogen and 1,000 ml of blood. An additional 1 Gm of fibrinogen and 2,000 ml of blood were transfused.

to control bleeding and maintain normal blood pressure. The following day blood studies revealed a hemoglobin of 58% and a reduced plasma volume as well. She was then transfused with 500 ml. of whole blood and 500 ml. of packed cells, an additional 1,000 ml. of whole blood being transfused several days later to restore the hemoglobin and blood volume to normal.

The use of *red cell suspensions*, or "*packed cells*" as they are commonly called, has proved valuable in the treatment of chronic anemia in patients with cardiac or renal disease in whom the volume of fluid administered must be restricted. This procedure also conserves plasma for, except in cases of shock or hypoproteinemia, whole blood is not usually needed in the treatment of chronic anemia. The method of preparation consists in allowing the red cells to sediment spontaneously or with the aid of centrifuging, drawing off the supernatant plasma, and transferring the cells to a separate container. If the blood has been collected in one of the dextrose preservative solutions, the cells may be stored in the sedimented state as long as whole blood. Their administration is the same as for whole blood after appropriate grouping and cross-matching.

The preparation of plasma and red cell suspensions demands the most careful technique and sterile precautions since these materials, which are excellent bacterial culture media, may be stored for a considerable period before being used. It is important to note that some pathogenic organisms whose toxins are lethal may continue to multiply in contaminated plasma even when stored at 4° C. If a transfusion for any reason has to be stopped, the same bottle of whole blood, plasma, red cell suspension, or any other intravenous solution should not be used again if more than an hour has elapsed between the first attempt and the next because of the danger of contamination.

Whole blood, plasma, or red cell suspension must always be administered with an intravenous set containing a *fine-mesh filter* to remove particulate matter or clots that may have formed.

Immunologic Aspects

The immunologic factors of clinical importance in blood transfusion are as follows:

1. The agglutinogens A and B
2. The Rh and related factors
3. Other rare factors

Of equal concern are the corresponding antibodies, the agglutinins anti-A, anti-B, anti-Rh, anti-Kell, anti-Duffy, etc.

The Landsteiner (or ABO) Blood Groups.—All individuals may be divided into four groups, depending on whether their erythrocytes contain either of the agglutinogens A or B, both, or neither of them. Agglutinins are present in the serum of an individual corresponding to the agglutinin which is absent from the erythrocytes of that individual as illustrated in Table 3.

TABLE 3
INTERNATIONAL (LANDSTEINER) NOMENCLATURE

NAME OF BLOOD GROUP	AGGLUTINOGEN IN ERYTHROCYTES	AGGLUTININ IN SERUM
AB	AB	Neither
A	A	Anti-B (beta)
B	B	Anti-A (alpha)
O	Neither	Anti-A and anti-B

In every transfusion it is the donor's erythrocytes that must be compatible with the recipient's, i.e., not agglutinated (clumped), by the agglutinins in the recipient's serum. As indicated in Table 4 Group O blood may be given to individuals of any group because O blood contains neither agglutinin A nor B. Persons belonging to Group AB may receive blood of any group because their serum does not contain either agglutinin anti-A or anti-B. With these two exceptions donor and recipient blood must belong to the same group to avoid agglutination, hemolysis, and the serious consequences of an incompatible transfusion reaction.

TABLE 4
INTERACTION OF THE 4 BLOOD GROUPS

	AB SERUM (NO AGGLUTININS)	A SERUM (ANTI-B AGGLUTININS)	B SERUM (ANTI-A AGGLUTININS)	O SERUM (ANTI-A AND B AGGLUTININS)
CELLS				
AB	-	+	+	+
A	-	-	+	+
B	-	+	-	+
O	-	-	-	-

+ = agglutination

- = no agglutination

TABLE 3

NOMENCLATURE		PER CENT PRESENT	PER CENT ABSENT
WIENER	FISHER-RACE		
Anti-Rh ₀	Anti-D	85	15
Anti-Hr ₀	Anti-d	63	37
Anti-rh ₀	Anti-C	70	30
Anti-hr ₀	Anti-c	60	20
Anti-rh ₁	Anti-E	30	70
Anti-hr ₁	Anti-e	97	3

Laboratory Technique.—Students and interns should be familiar with the technique of grouping and cross-matching blood because of the frequent urgent need for transfusion in the prevention and treatment of shock. The test for determining to which of the four blood groups a patient or donor belongs is performed as follows:

Two drops of citrated blood are added to approximately 5 ml. of normal saline A drop of the saline cell suspension is added to each of two serologic tubes. To one tube add a drop of anti-A grouping serum; to the other tube add a drop of anti-B grouping serum. Centrifuge at 1,000 rpm for two minutes. Examine for agglutination with the aid of a microscope. Reference to Table 4 will show how any unknown blood can be typed by noting the reaction of the erythrocytes with the anti-A and anti-B serums.

The technique of cross-matching is as follows. To a serologic tube marked "major" add a drop of a saline suspension of donor's cells and a drop of recipient's serum or plasma. To another tube marked "minor" add a drop of a saline suspension of recipient's cells and a drop of donor's plasma. Centrifuge and examine for agglutination.

Clumping of the cells in the *major cross-match* indicates that the bloods are incompatible and that the donor blood must not be given.

Agglutination in the *minor cross-match* may occur if (1) Group O donor blood is being tested against the blood of A, B, or AB recipient; (2) Group AB recipient blood is being tested against the blood of an A, B, or O donor. Only in these two instances may agglutination in the *minor cross-match* be disregarded because otherwise it indicates that the donor and recipient blood are incompatible.

The RH Blood Groups.—There is a group of six related factors which has a special importance in obstetrics. This group, known as the Rh Hr or CDE-cde system, and its incidence in the white race are shown in Table 5.

The original Rh factor is the most antigenic. The terms Rh positive and Rh negative therefore refer to the presence or absence of Rh. (D).

Although the anti-Rh agglutinins are not normally present in the serum, they may be acquired during pregnancy or after transfusion of blood of a different Rh type from that of the recipient. Isoimmunization to D is frequent, to c and E much less frequent, and to d, C, and e very rare. D is involved in approximately 93% of cases of hemolytic disease of the newborn. Although rare, immunization of Rh-positive individuals may occur; in such instances it usually involves c.

The anti-Rh agglutinins (Rh antibodies combine with the Rh agglutinogens of the fetal erythrocytes and cause hemolysis, a condition known as *hemolytic disease of the newborn* or *erythroblastosis fetalis*. Nothing happens to the mother because there are no Rh agglutinogens in her erythrocytes to combine with the antibodies in her serum. If, however, she receives a transfusion of Rh-positive blood during pregnancy or in the puerperium, the transfused erythrocytes combine with the Rh antibodies in her circulation to cause a transfusion reaction which may be fatal.

Transfusions of Rh-positive blood into Rh-negative individuals may result also in isoimmunization, and a severe reaction during a subsequent transfusion of Rh-positive blood, or as in the example of a mating cited above, they may initiate the process of isoimmunization even before the woman becomes pregnant for the first time. It is important, therefore, to test for the Rh in addition to the ABO factors and to use only blood of a suitable type. This is especially important in pregnancy and the puerperium because of the possibility of the presence of circulating Rh antibodies, and also in all female patients from infancy to the end of the childbearing period to prevent Rh isoimmunization by transfusion.

Laboratory Technique.—The simplest and quickest test for the determination of the Rh type is the open slide method. Place on a glass

source of error in cross-matching. Such an error may be avoided by the prompt examination of the cross-match.

Therapeutic Aspects

The treatment of shock with whole blood and plasma, as well as the use of plasma in various other conditions, and the use of red cell suspensions in chronic anemia have already been described in detail in this and the preceding section.

The use of *blood substitutes*, however, deserves special mention. It is now customary to refer to the substances which increase blood volume as *plasma expanders*, *extenders*, or *augmenters*. There is no substitute for blood

massive or prolonged hemorrhage, e.g., gastrointestinal bleeding, in which condition blood in large amounts is required.

In many cases a substitute alone will prevent or counteract shock. Most blood transfusions are given for operations requiring from 500 to 1,000 ml. of blood replacement. It is in this type of case that the substitute have an important role as blood-sparing agent.

Because the expanders have certain disadvantages, the search for an ideal one continues. It is centered on a fraction of human plasma containing albumin and the alpha and beta globulins and is free of hepatitis virus. Gelatin gels at room temperature. Polyvinyl pyrrolidone (PVP) is not metabolized and

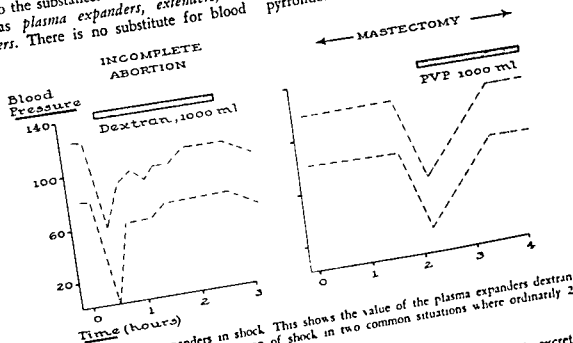


Fig 21—Plasma expanders in shock. This shows the value of the plasma expanders dextran and PVP in the treatment and prevention of shock in two common situations where ordinarily 2 or 3 bottles of blood are used

when hemorrhage is massive. In cases, however, where amounts ranging from 500 to 1,500 ml of blood are ordinarily used, plasma expanders may be substituted for blood. In emergency situations their use is often a life-saving measure during the time necessary to start a transfusion. Their use in these circumstances justifies their being called blood substitutes.

In most hospitals blood is chronically in short supply. If the extenders were used in cases where ordinarily one or two bottles of blood are administered during operation, more blood would be available for the treatment of

consequently that which is not excreted stored in the reticuloendothelial system. Dextran interferes with the coagulation mechanism and may cause bleeding from the operative site.

Since dextran in amounts of more than 1,000 ml has been shown to cause a bleeding tendency, it should not be used in patients with coagulation defects (thrombocytopenia, hypoprothrombinemia, etc.) or in those receiving anticoagulant therapy. Despite these limitations, however, they are valuable therapeutic agents when used in the recommended amounts, in properly selected cases, and with their disadvantages in mind.

Transfusion Reactions

The incidence of transfusion reactions and their occurrence despite the utmost care in the grouping, cross-matching, and transfusion of blood afford a constant reminder that transfusion itself may be considered a major surgical procedure and should be undertaken only when definite indications for such therapy exist. The least serious of all is the so-called allergic reaction, although usually no allergic basis can be found in either the donor or recipient involved. The usual manifestation is urticaria, and the treatment is symptomatic.

Pyrogenic reactions are the most frequent of all transfusion complications. They are caused by pyrogens which are the polysaccharide fraction of endogenous substances of bacterial lysis present in transfusion equipment which has not been properly cleaned and autoclaved. The use of disposable donor and transfusion sets, guaranteed pyrogen-free by the dispenser, tends to reduce the incidence of such reactions. The symptoms are chill, increased temperature and pulse, restlessness, dyspnea, and pains in the chest and lumbar region. In a severe pyrogenic reaction there may be circulatory collapse and even death. The treatment consists of immediately stopping the transfusion and the application of supportive measures. Since the symptoms just described are also those of an incompatible transfusion, all reactions following the transfusion of whole blood or red cell suspensions should be regarded as being due to incompatibility until proved otherwise.

The causes of an incompatible or hemolytic reaction usually are attributable to technical or clerical errors in the laboratory or to carelessness on the part of those responsible for administering the blood. Because of the possibility of error in grouping or labelling blood, every transfusion of whole blood or red cell suspension should be cross-matched with the patient's blood before being given. This procedure detects such errors and is an additional safeguard in the prevention of reactions. Blood transfusion in many instances is lifesaving, but it can also be fatal. Constant care must be exercised in order to avoid such accidents.

The symptoms of an incompatible transfusion are those already described, plus the

signs of hemoglobinemia and hemoglobinuria, followed later by jaundice, oliguria, and, in fatal cases, death in uremia. Since the earliest manifestations of a hemolytic transfusion are usually those complained of by the patient, such as a feeling of chilliness, pains in the back, etc., extra care must be taken in giving transfusions to those under general anesthesia or otherwise unconscious. Excluding sudden fatality at the onset of the reaction, renal involvement is the cause of serious consequences.

The pathogenesis of the renal damage resulting in the condition known as *lower nephron nephrosis* is not entirely understood. It is doubtful whether hemolysis alone causes damage. Hemoglobinemias and hemoglobinurias are frequently observed in such conditions as the paroxysmal hemoglobinurias without causing renal insufficiency. The various factors involved in the renal damage may be summarized as lowered blood pressure, vasoconstriction of renal vessels, reduced renal blood flow, and the secretion of large amounts of hemoglobin or its derivatives in an acid urine. Morphologically the main alterations in the kidney involve the nephron where degeneration and necrosis of the tubular epithelium occur with heme casts in the distal segments and collecting tubules.

The treatment may be divided into two phases. In the first or immediate phase, on the first indication of a reaction as shown by the appearance of any of the above-mentioned symptoms, proceed immediately as follows:

1. Stop the transfusion.
2. Obtain a sample of the donor's blood and send it to the laboratory with a specimen of the patient's blood for regrouping and cross-matching and for examination for hemolysis.
3. Give the patient sodium lactate intravenously to render the urine alkaline as soon as possible.
4. Give glucose intravenously to provide fluid for diuresis.
5. Record the 24-hour fluid intake and output, examining the first specimen for hemoglobinuria.
6. Apply heat to the costolumbar regions for reflex relief of renal vasoconstriction.

During the second phase, treatment is directed toward maintaining a balance between fluid intake and output and toward preserving the acid-base equilibrium until renal function returns to normal. Until such time it is important not to force fluids, since pulmonary edema may develop. The use of the artificial kidney or other dialyzing mechanism should be considered in cases in which there is a gradually rising blood NPN and a decreasing urine output and specific gravity. The products of uremia are thereby removed during the period in which reparative processes may be proceeding in the damaged kidneys.

Other Complications.—

Jaundice—

Pretransfusion hemolysis. The least important in its clinical implications is that due to hemolysis of the donor cells either before or immediately after transfusion. The hemolysis is due to the early death of the cells because of improper storage or handling of blood, i.e., their life span has been so shortened that the cells are moribund when they are transfused. Blood is taken out of the bank to the wards or operating theater. It may be left standing at room temperature for several hours and then returned unused. The same blood may again be cross-matched and taken out for another patient. If this sort of thing happens two or three times, and the blood has not been properly cared for, the red cells reach senility earlier than the date of obsolescence stamped on the bottle. When such blood is transfused, massive hemolysis and jaundice may occur. There are usually no other manifestations such as precede the appearance of jaundice in the next type to be discussed. The prevention of this complication is the responsibility of everyone who orders a blood transfusion or handles a bottle of blood.

Hemolysis after transfusion. By far the most important of the causes of jaundice following transfusion is known as the hemolytic or incompatible transfusion reaction which has already been discussed.

Homologous serum jaundice. Finally, the third cause of jaundice is not a hemolytic process but a hepatitis which is caused by a virus transmitted in the blood or plasma of

the donor. Homologous serum jaundice may be differentiated from the preceding types by its late appearance at an average of 60 days following transfusion and by the various tests used to distinguish between hemolytic and nonhemolytic jaundice. Although the morbidity is extremely low, the mortality is high.

Embolism.—Embolism caused by the introduction of air in the transfusion should never occur. The use of specially prepared transfusion sets containing fine-mesh filters to remove particulate matter which might be the cause of embolism has already been emphasized.

Citrate Intoxication.—There is no evidence that, even in massive transfusions of 5-10 liters given over a short period of time, the citrate in the transfused blood or plasma has any toxic effect or causes any change in the coagulation time of the recipient's blood. The citrate radical is rapidly removed from the circulation, oxidized, and excreted.

Although citrate is nontoxic, by reason of its calcium-binding capacity which renders it an anticoagulant in vitro as shown in Table 2, it may become harmful in certain situations. Exchange transfusion in the treatment of hemolytic disease is supplemented by the intramuscular injection of calcium gluconate if the blood is collected into citrate. There is an increasing tendency, however, to collect blood into heparin for exchange transfusion.

In operations on the heart especially, but also in others such as artificial renal dialysis in which extracorporeal circulation is necessary, heparinized blood is required because of the danger of citrate reducing the level of available calcium, thus leading to increased cardiac irritability, ventricular fibrillation, and standstill.

Heparinized blood may itself be hazardous. Although it keeps the blood fluid, it is a poor anticoagulant, allowing clotting to proceed in vitro as shown in a few hours by a decreased content of the various coagulation factors (platelets, prothrombin, fibrinogen, etc.) in bank blood. Its use in large amounts may be followed by generalized postoperative hemorrhage.

Pulmonary Edema.—The sodium radical may not be excreted rapidly. Its water retaining properties, especially when large amounts of

blood are transfused, may contribute to the development of edema. In such transfusions, furthermore, the amount of sodium citrate may assume significant proportions in patients with cardiac or renal disease. For example, since the ratio of blood to diluent is of the order of 3:1, in a transfusion of 4,000 ml there will be 1,000 ml of diluent, containing 17 Gm sodium citrate. It is important, therefore, to use sparingly other intravenous solutions, especially saline (unless specific indications for their use are present), so as to avoid circulatory overloading and pulmonary edema in patients with chronic anemia complicated by cardiovascular or renal disease. In the treatment of pulmonary edema, the performance of a phlebotomy is expedited by the use of blood donor equipment if the donor bottle is of the type that contains a vacuum.

The Blood Bank and Transfusion Services

The blood bank has become established as an integral part of the hospital. There are, however, other transfusion services organized on a community or regional basis, and the various Red Cross societies have developed plans for national blood transfusion services which are in operation in Canada, Great Britain, and the United States. The purpose of both types of services is the same, namely, the provision of adequate amounts of whole blood and plasma properly prepared for immediate transfusion.

Fresh blood contains the maximum of all components of donor blood and is as effective as in a direct transfusion. *Stored blood*, i.e., blood collected in sodium citrate solution only and refrigerated, may be used up to five days following donation, since degenerative changes develop rapidly in the erythrocytes if no preservative is added. When dextrose is added to the blood-citrate mixture to impede the rate of destruction of the erythrocytes, the resultant product is known as *preserved blood*.

Blood is obtained from healthy donors, male and female, between the ages of 18 and 65.

Approximately 350 ml of blood, under sterile precautions, are withdrawn into a bottle containing a volume of 125 ml. of a solution of sodium citrate and dextrose. At the same

time enough additional blood is taken for filling two tubes, one for serologic testing for syphilis, the other to be used for grouping, typing, and cross-matching. The blood is immediately refrigerated. When it has been grouped for the ABO factors and Rh typed, and the test for syphilis is negative, the blood is properly labelled and ready for use. Upon receipt of a requisition for blood transfusion, the accompanying sample of patient's blood is grouped and typed. Donor blood corresponding to the recipient's in respect of the factors ABO and Rh is cross-matched with the patient's and, if found compatible, is marked with the patient's full name, age, sex, ward, and service for proper identification to avoid its being given to the wrong patient. It is then delivered to the ward or operating room or is kept in a reserve compartment of the refrigerator until needed.

All of the above procedures are carried out by the blood bank staff whose grave responsibility in their proper performance is almost unique in medicine. The results of most laboratory tests are used to corroborate a clinical diagnosis, and any discrepancy between the clinical and laboratory findings may be checked by repeating the tests before the treatment is instituted. An error in technique, interpretation, or labelling by the blood bank may result in a serious or even fatal transfusion reaction.

Transfusion has entered a new era. In the beginning, as it became clear that surgical shock is due to hemorrhage, blood banks were established for its prevention and treatment. The next decade was characterized by advances in immunohematology and their application to the prevention of transfusion reactions and the management of hemolytic diseases of the newborn.

At the present time the responsibilities of the blood bank have been augmented by advances in our understanding of the coagulation disorders, the availability of blood and plasma fractions and the recognition of the specific indications for their use, and the ever-increasing number of operations requiring massive, fresh or heparinized blood transfusion.

The responsibility for the direction of a blood bank may be assigned to an anesthetist,

a bacteriologist, pathologist, physician, or surgeon. Blood transfusion, because of its many aspects and their rapid development over the past few years, has itself become a specialty. The functions of the director of a transfusion service should include not only the establishment and maintenance of the proper technical and administrative procedures but, also of equal importance, certain other activities. These include the provision of consultative services in all aspects of blood transfusion therapy and instruction of students and interns in the early recognition of shock, its treatment, and research in the problems related to shock and blood transfusion.

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Film References

Running Time
17 min

Sound or Silent
Sound

Procurable From

Director, Armed Forces
Institute of Pathology
Washington 25, D. C.,
Attention: Chief Medical
Illustration Service

Sound
Color

Canadian Film Institute
142 Sparks St.
Ottawa, Ontario

Title

Clinical Shock (An analysis of the physiopathology of traumatic shock with special reference to diagnosis and treatment) (1952)
(Directed by John D. Stewart, M.D., Buffalo)

Blood Transfusion (An instructional training film demonstrating technique, indications, and hazards of transfusion)

25 min

Chapter 5

Preoperative and Postoperative Care

James F. Hopkirk, MD, and Richard C Long, MD

PREOPERATIVE MANAGEMENT

Assessment of the Surgical Patient as an Operative Risk

For the achievement of successful surgical treatment, the surgeon must make not only an exact diagnosis but also carefully plan and execute the operative procedure. This procedure must be carried out without untoward incidents which might lead to the death of the patient or to an unnecessary number of complications. The goal of each hospital admission and surgical procedure must be the early return of maximal possible function of the patient. This end can be gained only by a very careful preoperative appraisal of the patient, not so much in regard to the operative risk, but as to how much the entire phase of hospital treatment will either favorably or adversely affect him.

One of the most important phases of the preoperative and postoperative care is the development of an accurate medical record which must include a painstaking case history and physical examination, a preliminary working diagnosis, a graphic description of the operative procedure, detailed notes of the patient's progress in hospital, medications, complications, the success of the therapeutic procedure, and a well-considered final diagnosis.

Patients may be placed into two large categories in respect to their ability to withstand operative procedures

1. Good risk
2. Poor risk

The good-risk patient is one who comes for the treatment of some surgical condition which has caused little or no systemic effect and has no accompanying disease or group of diseases, e.g., uncomplicated hernia.

The poor-risk patient is one who presents himself with disordered bodily functions which have been caused by the surgical condition for which he seeks relief, or by other concomitant disease or diseases, e.g., hernia with strangulation where more extensive surgery may be necessary.

It is difficult to categorize each patient. A superficial appearance of reasonably good health is often misleading, and more complete physical or laboratory examination may place this patient in the poor-risk category.

Many factors affect the assessment of the patient as an operative risk.

1. **The Adjustment of the Individual.**—An important consideration is the individual's adjustment to his disease, to his attending physician or surgeon, and to the hospital wards and personnel.

2. **Age.**—Infants and young children constitute individual physiologic and metabolic problems. They are very prone to develop infection and require special operative techniques. Developmental anomalies which are usually first noticed in this age group will frequently affect the planning and execution of an operative procedure. The elderly patient also constitutes a special problem. He reacts

less favorably to the trauma of operation, his nutrition is frequently poor, and he may have accompanying cardiovascular, renal, or respiratory disease which necessitates a careful choice of anesthesia and sedation.

3. **Psychologic Status.**—It is important that the patient be carefully prepared as to the outcome of any operative procedure. An intimate patient-surgeon relationship is most important in all types of surgery. This is most essential in the handling of a patient with, for example, ulcerative colitis, for whom a permanent ileostomy will be required, or one with carcinoma of the rectum who will have a permanent abdominal colostomy.

4. **Degree of Obesity.**—The obese patient is always a poor surgical risk. Technical procedures are much more difficult in such cases, wound healing tends to be delayed, and the wounds are more prone to become infected.

5. **Nutritional Status.**—The recognition, prevention, and treatment of nutritional deficiencies are important because many surgical conditions may be improved by careful nutritional therapy, and many postoperative complications may be prevented by an adequate preoperative diet.

The assessment of the nutritional status of the surgical patient is made by a careful examination of the patient's dietary history, his weight history, the condition of his tissues, his response to adequate dietary therapy, and by such laboratory aids as estimation of the serum protein levels and the albumin-globulin ratio.

6. **Cardiovascular Status.**—The status of the cardiovascular system must be carefully assessed and particular attention paid to the history of recent cardiac accident, decreased exercise tolerance, presence of cardiac failure, or severe congenital heart disease. Hypertension necessitates that special steps be taken in the anesthetic management to prevent a hypotensive episode.

7. **Status of the Liver.**—The liver is frequently adversely affected by nutritional disorders and dietary deficiencies. All surgical patients must have a sufficient protein and carbohydrate intake because adequate storage of liver protein and glycogen is required to prevent untoward accidents, such as the so-

called liver failure which occurs sometimes following surgical procedures.

An attempt can be made to evaluate functional status of the liver by a careful study of diet and weight and by the response to adequate nutritional measure. For example, the disappearance of edema or following high protein intake. In addition, use of various liver function tests can give valuable information. (See Chapter 19, and Portal Hypertension.)

8. **Status of the Urinary Tract.**—The function of the kidney must at all times be fully assessed, as impaired renal function markedly increases the incidence of postoperative complications and possibly may even lead to the death of the patient. An adequate outflow of satisfactory specific gravity is one of the best indications of good renal function.

An estimate of the urinary function may be made by an inquiry into the following factors:

A. The urinary history, e.g., nephritis, prostatic disease.

B. Renal function tests which include:

- (1) urinalysis
- (2) urine concentration tests, Mosenthal
- (3) renal excretion tests, e.g., phenolphthalein excretion
- (4) blood serum estimations, e.g., serum nonprotein nitrogen

9. **Hematologic Status.**—The anemic patient does not withstand operation well. He is ill equipped to tolerate operative blood loss and is frequently in a state of malnutrition. It is therefore essential that the hemoglobin and red blood cells be within reasonably normal range prior to surgery, whether the surgical procedure is of an emergency or of elective nature. A knowledge of the level of the white blood cell count is important as occasion agranulocytosis or leukemia may be present.

10. **The Operation Itself.**—The magnitude and type of operation must be considered when assessing the risk to the individual patient. A poor-risk patient, for example, might withstand a minor procedure, whereas a major operation could be fatal.

The Attending Surgeon's Visit—It is the duty of the attending surgeon to take the time

to explain to the patient in simple language the stages in preoperative management which have been ordered, to describe in some manner the operative procedure and the anesthetic to be used, and to indicate to the best of his ability what the patient may expect in the immediate postoperative period.

Nutrition

The recognition, prevention, and treatment of nutritional deficiencies are important because many surgical conditions may be improved by adequate nutritional therapy, and many postoperative complications may be prevented by careful attention to diet.

The normal individual requires 2,500-3,000 calories per day, and the diet must include sufficient protein, carbohydrate, fat, vitamins, and minerals. Nutritional deficiencies in surgical patients are relatively common and arise as a result of

1. Inadequate intake (starvation, ulcer diet, parenteral feedings)
2. Increased requirements (hyperthyroidism, pregnancy, increased muscular activity)
3. Impaired absorption or excess loss (pyloric stenosis, esophageal obstruction, vomiting, diarrhea, intestinal fistula)
4. Defective utilization (diabetes)

Such deficiencies must be corrected before any major surgical procedure is contemplated.

Caloric Requirements.—An adequate caloric intake is necessary to prevent actual or partial starvation. An inadequate caloric intake results in starvation of the tissues with resultant tissue breakdown and an increase in nitrogen excretion.

The administration of a high caloric diet in a patient with a good appetite can readily be accomplished by an oral intake of high caloric foods supplemented if necessary by high caloric, high protein milk shakes. If the patient's condition is such that oral feeding is impossible or restricted, the problem becomes increasingly difficult. An attempt to supply the full caloric requirements with isotonic intravenous glucose alone is unsatisfactory, as such a large volume of fluids must be given. Intravenous fat if available, however, offers a

means of providing such a high caloric intake without the difficulties which arise when glucose solutions are used.

Tube feeding with carbohydrate, protein, and fat in the correct proportions may also be used at times to ensure the necessary number of calories.

Protein.—The body proteins are compounds of amino acids and form a proportion of all living tissues. Ingested proteins are hydrolyzed into their constituent amino acids, which are then absorbed from the small intestine.

The plasma proteins consist of three fractions.

1. *Albumin*, which is chiefly responsible for maintaining osmotic pressure
2. *Globulin*, which consists of immune substances, antibodies, and agglutinins
3. *Fibrinogen*, which is essential for the clotting of blood

The normal level of the plasma proteins is 7 Gm/100 ml. with an albumin/globulin ratio of 1.5/1. The serum protein, while useful as a guide, is not a true indication of the total body protein, for depleted reserve stores of protein in the tissues may be present with a normal level in the circulating blood. Gross edema usually occurs when the serum albumin is less than 3.0 Gm/100 ml. However, the degree of the edema may be modified by the presence of anemia and by disturbances in electrolyte balance.

There are five main causes of protein deficiency:

1. Insufficient intake of protein to meet the demands of the body; e.g., anorexia, obstruction of the upper alimentary tract, and vomiting
2. Inadequate digestion or absorption of protein; e.g., in chronic gastrointestinal disease, gastrojejunocolic fistula, abnormalities in secretion of the stomach or pancreas
3. Impaired protein synthesis; e.g., in severe liver disease
4. Excessive protein loss; e.g., in kidney disease, burns, ascites, ulcerative colitis, or empyema
5. Increased protein catabolism; e.g., in association with fever, hyperthyroidism, opera

tive procedures, severe trauma, malignant diseases, or extensive burns

The effects of such protein deficiency may cause serious complications in the surgical patient. Every operation increases nitrogen catabolism with a resultant demand on protein reserves and usually results in a temporary negative nitrogen balance. Protein depletion is increased by hemorrhage, serum loss, and suppuration. Inadequate protein intake is associated with delay in wound healing, wound disruption, and chronic indolent ulcers. Hypoproteinemia aggravates the tendency to edema following gastrointestinal operations with resultant obstruction of the anastomotic stoma. The liver, if deficient in protein and especially if low in methionine and cystine, is very susceptible to toxic agents and to the effects of operation

Correction of Protein Deficiency—The best way to supply the caloric and protein requirements of a patient is to provide an adequate oral intake. Whole foods, rather than protein hydrolysates, should be used. A diet containing 1 Gm of protein per kilogram of body weight is adequate for the average patient, although for short periods this amount may be reduced if sufficient carbohydrate is added. If there has been extensive trauma or severe infection, much larger quantities of protein, up to 300 Gm/day, may be required. There is evidence that a high protein diet given for a period prior to operation may increase the protein reserves of the body and so compensate for nitrogen loss in the postoperative period

Protein deficiency in the presence of anemia presents a special problem. If the body is in need of both hemoglobin and plasma protein, the formation of hemoglobin is favored. It is therefore most important that existing anemia should be corrected by adequate transfusions of whole blood in protein-deficient patients

It is essential that the patient be placed in positive nitrogen balance and that the plasma protein be restored to normal levels before a major surgical procedure is undertaken.

When normal oral feedings are contraindicated, or when only small amounts can be given by this route, other methods must be

utilized, e.g., tube feedings with protein hydrolysates or other protein fluids, jejunostomy or gastrostomy feeding, or intravenous feeding

Intravenous administration of whole blood, plasma, serum albumin, and protein hydrolysates can be used in tiding over a patient for a short time when oral intake is impossible or inadequate. Whole blood and blood derivatives are impractical for long-term use. They are expensive, the supply is limited, and the increased blood volume which follows their use may lead to cardiac embarrassment. Protein hydrolysates, reinforced with glucose, can maintain a positive nitrogen balance if given in sufficient amounts. In the usual protein hydrolysate solution, there are somewhat less than 50 Gm of protein per 1,000 ml, and there is considerable evidence to show that such hydrolysates are only partially utilized by the body. With the preparations available there is a high incidence of reactions: nausea, vomiting, fever, and headache. Solutions of pure amino acids are of greater value and are capable of providing adequate protein requirements with minimal or no side effects. However, in the presence of marked protein deficiency, it is impossible to supply sufficient protein by this method

Vitamins and Minerals—The administration of additional vitamins and minerals is unnecessary if the patient is taking a full and well-balanced diet. In debilitated patients and in those for whom parenteral feedings are required, vitamin supplements must be provided. Combustion of carbohydrate greatly increases the requirement for the vitamin B complex, and this should be given parenterally to all patients who are receiving intravenous glucose feedings. Vitamin C must also be given as its lack may lead to delay in wound healing. In the jaundiced patient or in one with advanced liver disease, parenteral vitamin K must be used

Body Fluid and Electrolytes

Fluid and electrolyte derangements are extremely common in the surgical patient. Even minor operations which require some degree of immobilization, or restriction of the intake of food and water, have a slight, although perhaps hardly perceptible, effect on the body

However, operations which require general anesthesia can, through toxic effects, vomiting, and the development of the alarm reaction, cause a metabolic disturbance which can be profound. This disturbance is due both to a deficit in water and electrolytes and a defect in the normal pattern of these substances. This defect may not be great and, indeed, is frequently unnoticed. However, the surgeon must anticipate the possibility of any disturbance in the fluid-electrolyte pattern and treat it in such a manner as to avoid a major physiologic breakdown.

Severe fluid and electrolyte derangements also occur as a result of an acute disease process. These derangements must receive early and intelligent restorative therapy in order that the underlying disease may be successfully treated.

In order to formulate a plan of successful fluid and electrolyte therapy, the type of imbalance leading to the dehydration must first be established, and the magnitude of the defect must be estimated, following which the correct treatment may be planned and then executed.

The body fluids make up, on the average, 60% of the total body weight. This figure varies with the sex, age, and bodily habitus of the patient. In the newborn infant, 77% of the body is water. This percentage gradually falls so that by the age of 9 years, it stabilizes at the average for the adult. In the adult, there is a distinct sexual difference, the male having 17% more body water than does the female.

The body water is contained in two great compartments. The intracellular compartment contains about two thirds and the extracellular compartment about one third of the total body water. The extracellular fluid is divided into the *interstitial* and *intravascular* fluids which comprise 15% and 5%, respectively, of the total body fluid—three fourths (10.5 L.) and one fourth (3.5 L.) of the extracellular fluid.

The dominant intracellular ions are the cation potassium (K^+) and the anion phosphorus expressed as phosphate (PO_4^-). The dominant extracellular ions are the cation sodium (Na^+) and the anion chloride (Cl^-).

However, both the intracellular and extracellular compartments contain appreciable amounts of all ions.

1. The Extracellular Compartment

Cations

Sodium.—The concentration of sodium in the extracellular fluid is 142 mEq/L. There are, on the average, 65 Gm. of sodium in the body, of which 38 Gm. are in the extracellular compartments, 6 Gm. within the cells, and 21 Gm. in the bone; this exists as sodium chloride. This body sodium is divided into two portions: (1) the *exchangeable sodium* which is physiologically active, and (2) the *inert sodium*, contained in the bones, which is physiologically inactive. Under certain circumstances, e.g., in severe dehydration, a portion of this inert sodium may be made available for bodily reactions.

The sodium ion makes up over 90% of the total base and is the principal factor controlling osmotic pressure in the extracellular fluid. The other cations (Ca, Mg, and K) have little effect on fluid shifts, although they have great physiologic importance.

The concentration of extracellular sodium is chiefly maintained by (1) the sodium intake of the individual, and (2) the selective secretory action of the kidney. In times of sodium deficiency, the kidney tubules conserve body sodium so efficiently that virtually none is lost in the urine. In times of sodium abundance, excess sodium is freely secreted by the kidney. Also a decrease in serum sodium concentration will indicate a loss of fluid in order to re-establish the normal serum levels. An increase in sodium concentration stimulates water retention, thus diluting the sodium ion back to its normal serum level.

The average intake of sodium is 6 Gm. per day, varying slightly with the individual's taste and habits. Sodium is lost from the body in urine and sweat. The urinary sodium averages about 110 mEq/day. Insensible perspiration is essentially electrolyte-free, but if perceptible sweating occurs there is electrolyte loss.

There is a continuous movement of sodium in and out of the cells in health and disease. The concentration of the intracellular sodium

varies directly with the pH of the extracellular fluid. In *alkalosis* (pH over 7.45) the intracellular sodium is elevated, and the intracellular potassium is decreased. The reverse is true in *acidosis*, whatever the etiology may be. The plasma concentration of the sodium is in equilibrium with that of the interstitial fluid so that routine analysis of the interstitial fluid as well as of the plasma sodium reflects the sodium picture of the interstitial fluid as well. In cases of potassium deficiency, sodium enters the cells in large quantities in order to preserve the cells' isotonicity. However, the newly shifted intracellular sodium does not function with the same biologic efficiency as does the normally occurring intracellular ion potassium.

Potassium.—Normally the concentration of extracellular potassium varies between 3.5-5.5 mEq/L., comprising about 2% of the total body potassium. Although the daily requirements of potassium are not known, the normal diet contains more than a sufficient amount for the body needs. It is normally excreted almost entirely in the urine at the rate of 30-50 mEq/day (1.45-1.95 Gm). In contradistinction to its control over sodium, the kidney has no power to conserve potassium in times of potassium deficiency, so that potassium continues to be lost at a constant rate providing the urinary output is maintained. The concentration of potassium in the extracellular fluid does not necessarily reflect the potassium concentration within the cell although, as with sodium, it indicates the level in the interstitial fluid.

Calcium.—The normal serum calcium is 4.5-5.8 mEq/L. (9-11.5 mg/100 ml.) This level is slightly, if at all, affected by the daily calcium intake. An excess is excreted by the kidneys, and any defect is made up by a shift of calcium from the bones to the extracellular fluid. The serum calcium level is largely regulated by the parathyroid hormone. Occasionally calcium deficiency will accompany other electrolyte imbalance, e.g., respiratory alkalosis.

Magnesium.—The normal serum magnesium is 1.4-2.4 mEq/L., and the total body content of magnesium is approximately 19 Gm. It is excreted in the feces (60%) and urine (40%). Most of the magnesium is contained in the intracellular water and in the bones.

Magnesium is an essential coenzyme in carbohydrate and protein metabolism and has an effect on neuromuscular irritability.

Anions

Chloride.—The normal serum chloride concentration is 103 mEq/L., and the total body content of chloride is approximately 8.5 Gm. The average intake of chloride, ingested chiefly as sodium chloride, varies from 69-260 mEq/day (2.4-9.2 Gm). The concentration of extracellular chloride varies inversely with the bicarbonate level. A loss of chloride from the body is met by an increase in bicarbonate so that there is no accompanying loss of water. This is in contradistinction to sodium loss, where no compensating factor exists and where water loss occurs. A deficit of the chloride ion leads to a loss of potassium. This condition is produced by an increase in bicarbonate and the development of a metabolic alkalosis. Potassium then leaves the cells to be replaced by sodium, and the potassium is excreted in the urine.

Bicarbonate.—The normal bicarbonate concentration in the plasma is 25-29 mEq/L. Bicarbonate is an end product of metabolism and hence is being continually produced. The concentration of bicarbonate in the extracellular fluid is controlled by the total quantity of base which is available to combine with it. This available base is the difference between the total cations (Na, K, and Mg) in the extracellular fluid and the anions chloride, phosphate, and plasma proteins. This difference is equal to the quantity of carbonic acid which must combine with a base, i.e., Na, to form bicarbonate (NaHCO_3) so that the fluid remains neutral. Bicarbonate ion concentrations depend chiefly upon kidney function, and as long as this is normal, derangements of pH seldom occur.

Normal Fluid Shifts

There is a continuous movement of water and electrolytes, chiefly sodium, between the vascular and the interstitial compartments. This movement is produced by the following:

- 1 The plasma protein concentration, which pulls fluid into the vascular compartment by osmotic action.

2. The intravascular hydrostatic pressure, which forces fluid out of the vascular compartment

3. The shifting electrolyte osmotic pressure of both the interstitial and intravascular areas

For practical purposes the plasma-electrolyte concentration may be taken as being the same as that found in the interstitial fluid, so that a biochemical examination of a sample of blood plasma will reflect the state of the interstitial fluid as a whole (in actual fact, the plasma-electrolyte concentration is slightly higher than that of the interstitial fluid).

Water also moves between the intracellular and extracellular compartments, and this movement is produced by changes in either water intake or output or by abnormal electrolyte losses. If the extracellular sodium level becomes lowered, causing a hypotonicity of this space, water is shifted into the cells in an attempt to bring about normal concentration and isotonicity. On the other hand, if water alone is deficient in the extracellular space, leading to a concentration of sodium, water then shifts out of the cells into the intracellular compartment, tending again to bring about isotonicity.

Disturbances of Acid-Base Balance

Acidosis.—Acidosis is due either to an increase in fixed acid or to a reduction in total available base, either of which will produce a reduction of bicarbonate concentration. This condition may be either respiratory or metabolic in origin.

Metabolic acidosis (acidemia) occurs most frequently in renal insufficiency where there is retention of acid, in uncontrolled diabetes from the production of keto-acids, and from the loss of alkaline secretions containing the sodium ion in severe diarrhea and in intestinal, pancreatic, or biliary fistulas.

Respiratory acidosis is far less common than the metabolic form and occurs where there is an interference with gaseous exchange within the lungs so that carbon dioxide is not eliminated.

Alkalosis.—Alkalosis is a decrease in hydrogen ion concentration and may be due to either metabolic or respiratory derangement.

Metabolic alkalosis (alkalemia) is due to (1) an increased loss of anions (chiefly chloride) in body secretions, such as occurs in the vomiting of pyloric stenosis or accompanies prolonged gastric suction, (2) excess bicarbonate administration, or (3) a potassium deficit.

Respiratory alkalosis occurs because of a decrease in extracellular carbonic acid concentration, as a result of hyperventilation.

Sodium Derangement

The sodium ion is the most important single factor in controlling the distribution of body water because of its high concentration in the extracellular fluid and the slowness of its passage across the cell membrane. Any drop in the concentration of sodium leads to a loss of water; an increase in sodium causes water retention.

Sodium deficiencies are of two types occurring in the following situations. (1) where the total body sodium is normal, but the concentration in the extracellular fluid is low (sodium concentration deficit), and (2) where the total body sodium is reduced (sodium depletion). This is an important consideration as the concentration of sodium in the extracellular fluid does not necessarily reflect the total body sodium; e.g., if the extracellular fluid has been diluted by the addition of large amounts of water, the extracellular concentration is low, while the total body sodium is normal or even increased. Except in edema, sodium concentration deficits rarely occur with sodium depletion. It is this latter syndrome which commonly occurs in the surgical patient.

The symptoms of sodium depletion vary with the magnitude of the loss. In the mild case the early symptoms are anorexia, apathy, lassitude, and weakness. Thirst is usually not present. As more sodium is lost, these symptoms become more marked, nausea and vomiting may occur, and symptoms associated with a decrease in the blood volume manifest themselves (dizziness, fainting, postural hypotension, and severe headache). The skin and mucous membranes show evidence of dehydration. In severe sodium depletion the patient may be stuporous or comatose with a

faint pulse, low blood pressure, atonic muscles, and gross evidence of dehydration

Treatment.—Before any treatment is instituted for the care of a depleted patient a careful evaluation should be made of (1) the case history, with particular reference to duration of illness, amount of vomiting, sweating, fever, and urinary output, (2) a physical examination, particularly noting the state of the mucous membranes, the skin turgor, blood pressure, and the presence or absence of sweating, and (3) laboratory studies of the blood, urine, and body secretions which should include estimation of serum Na, K, Cl, HCO_3 , NPN, hematocrit, and blood volume

Mild sodium depletion is readily treated by the administration of isotonic saline. Severe forms require the use of a hypertonic solution either 3% or 5% NaCl

2. The Intracellular Compartment

Essentially the same ions, although in different concentrations, are present in the intracellular as well as in the extracellular compartment. The dominant cation is potassium, and the dominant anions are phosphate and proteins. There is a continuous but slow movement of fluid and electrolytes between the cells and the extracellular fluid; changes in one compartment cause a compensatory shift of water and electrolytes from the other

Potassium

Ninety-eight per cent of the body potassium is within the cells. Although it is the predominant cation of the cell fluid, it accounts for only two thirds of the available intracellular base, the other one third being magnesium.

The concentration of potassium is 30 times higher in the intracellular compartment than it is in the interstitial fluid. Part of this potassium is in free ionic form, and part is bound to protein. Potassium is an essential part of the cell structure. This fact is evident in starvation, where the increased cellular breakdown which is reflected in nitrogen loss is accompanied by an increased loss of potassium. The ionic potassium renders the intracellular fluid isotonic with the extracellular fluid. Potassium is necessary for adequate cell function, and

any increase or deficiency of this ion is injurious to cell life. The concentration of the intracellular ion is 150 mEq/L.

In addition to the dominant potassium, phosphorus, and magnesium ions, the cells also contain sodium and chloride. The permeability of the cell membrane is under constant metabolic control, and any decrease in the potassium level is countered by a shift of the extracellular sodium into the cells in order to preserve the intracellular electrolyte concentration and provide physiologic isotonicity. If the level of extracellular sodium falls, potassium tends to shift from the cells to the extracellular compartment. However, this shift acts as a very temporary expedient, as the extracellular potassium is rapidly lost in the urine. Potassium is continuously lost via the kidney, which has no power to conserve this ion in times of hypokalemic duress. Only during oliguria or anuria does the urinary potassium appreciably diminish or cease.

Potassium Derangement

Excessive extracellular potassium is a highly toxic substance and if increased above physiologic level, will cause myocardial damage. This damage will give typical electrocardiographic changes. If the potassium level rises high enough, the heart will stop in diastole.

Deficiency in potassium also has a profound effect on the organism and occurs in the body according to the following sequence of events:

1. Lack of fluid and electrolyte intake
2. Shift of water and potassium from the cells to the extracellular fluid
3. The displaced or shifted potassium is then lost in the urine

Potassium deficiency occurs in the following conditions:

1. Lack of fluid intake
2. Increased abnormal potassium losses such as in vomiting and diarrhea
3. Diabetic acidosis
4. During improper fluid therapy (excessive administration of isotonic saline)
5. In adrenocortical tumors
6. During the administration of adrenocortical hormones

7. A relative deficiency in surgical shock or in thermal burns, as a result of the pooling of noneffective concentrations in, e.g., the area of the burn

8. Certain instances of chronic renal insufficiency

Symptoms.—In *hypopotassemia*, the patient is dull, apathetic, and may be mentally confused. Muscle tone is poor, causing bowel distention and respiratory disturbances. In the extreme case there is a flaccid paralysis of the extremities. The heart sounds are poor, the pulse is slow, and the blood pressure falls.

Hyperpotassemia is rare. Much of the symptom complex is confused with the underlying clinical cause which is usually renal shutdown. It can be caused by overvigorous therapy with parenteral potassium, particularly in the absence of a good urinary output. However, it can occur in patients with normal kidney function if excessive parenteral potassium is administered. Excess oral administration will not cause toxic levels if the kidney function is adequate.

Diagnosis.—The diagnosis of potassium derangement is made by a consideration of the following:

1. The serum electrolyte levels

2. Electrocardiographic changes

3. History of the patient with reference to duration of illness, amount of fluid intake, type of fluid loss, and volume of urine output

Therapy.—In every case of potassium deficiency there is an associated disturbance of the entire body fluid and electrolyte pattern. The administration of potassium must therefore be accompanied by adequate amounts of water, sodium and chloride, optimum caloric intake and, at times, adjustment of the acid-base ratio by the administration of acid or base solutions.

Potassium is best given by mouth, 3 Gm of potassium chloride in 20 ml. of water may be given q 6 h. If intravenous therapy is necessary, 60-80 mEq. may be given daily in the intravenous fluid. Occasionally in cases of extreme deficiency this dose may be increased,

but it is much safer to keep to these levels and be prepared to allow several days for the patient to regain a normal potassium balance. Because of the dangers of hyperpotassemia, an adequate urinary output is a prime essential before intravenous potassium therapy is started.

Dehydration

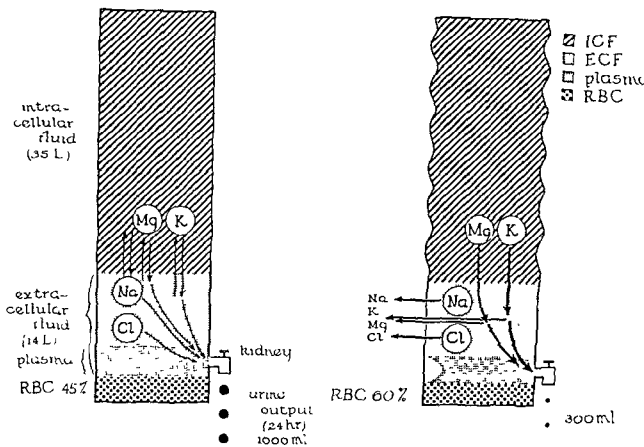
There are two types of dehydration:

1. *Water and electrolyte depletion (hypotonic dehydration).* This occurs where there is a loss of both body salt and water such as commonly occurs in the severe vomiting of pancreatitis or intestinal obstruction, in the severe diarrhea of ulcerative colitis, or in the case of external intestinal fistula.

The symptoms are due to sodium and fluid deficiency and occur early. There are thirst, weakness, oliguria, and a fall in blood pressure, and there may be peripheral circulatory collapse.

In the early stages of hypotonic dehydration, the fluid and electrolyte loss is chiefly from the extracellular compartment. The loss of salt is relatively greater than the loss of water and causes a hypotonicity of the extracellular fluid. As the dehydration process continues, the intracellular compartment becomes involved with a shift of water and potassium across the cell membrane in an attempt to make up the deficit in the interstitial fluid. Sodium then enters the cells, further decreasing the extracellular level of this ion. The newly shifted potassium is lost in the urine and via the abnormal routes of vomiting and diarrhea. The kidney conserves water, sodium, and chloride, but water continues to be lost via the skin and lungs.

2. *Pure-water depletion (hypertonic dehydration)* There is a loss of water without any loss of electrolytes, leading to hypertonicity of the extracellular fluid. This situation occurs in those conditions where there is a lack of water intake, e.g., in starvation, or in cases where the patient is unable to swallow, such as in esophageal tumors with dysphagia, great weakness, or coma. When all water intake ceases, the body continues to lose water via the kidney, lungs, and skin. The urine output must be



THE NORMAL FLUID SPACES
IN A 70 Kg MAN

EARLY RAPID OR HYPOTONIC
DEHYDRATION

*Fig 22—A diagrammatic representation of the fluid spaces in a 70 kg man

The volume of the intracellular fluid is shown as 35 L. and that of the extracellular as 14 L. The concept of continuous shift of Na and K between the intracellular and extracellular compartments is indicated by the two directional line arrows and that of the continuous K loss by the normal kidney with a normal urine output, by a single fine arrow. The Mg ion behaves in a similar fashion to the K ion. There is only a small loss of Na and Cl in the urine, due to the kidney mechanisms, which preserve the normal body concentration of these ions. The hematocrit is normal and is indicated by RBC 45%.

Fig 23—Early rapid or hypotonic dehydration

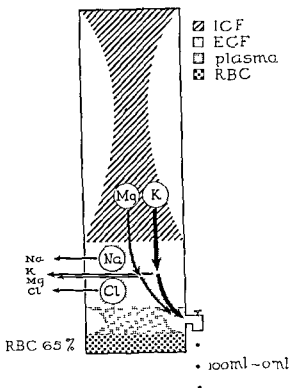
This condition results where there is loss of both water and electrolytes, e.g., in severe vomiting or intestinal obstruction. The magnitude of the electrolyte loss is relatively greater than the fluid loss. The dehydration is initially limited to the extracellular fluid, although there is a beginning instability of the intracellular compartment. There is a reduced urine output and a slightly increased K and Mg loss. There is no urinary Na or Cl loss, as the kidney is acting to conserve these ions. The hematocrit is significantly elevated.

at least 500 ml./24 hours in order to excrete nitrogenous waste products. The skin and lungs usually account for an additional loss of 1,000 ml. This total of 1,500 ml. is called the obligatory water loss. It can be seen that this continued loss of 1,500 ml/24 hours, without adequate replacement, will rapidly lead to dehydration. As there is little electrolyte

loss, and that chiefly potassium, the extracellular fluid becomes hypertonic. The cells put out water in an attempt to maintain intracellular and extracellular isotonicity. With this water shift, cellular potassium also moves across the cell barrier to be, in time, lost in the urine. As the lack of fluid intake is usually accompanied by a lack of caloric intake, cellular breakdown occurs, contributing to the potassium loss.

In contradistinction to the hypotonic dehydration, the water deficit is manifest equally

*Figs 22, 23 depict the direction of fluid and electrolyte shifts which occur during different degrees and forms of dehydration. The magnitude of these shifts is also roughly indicated.



LATE RAPID OR HYPOTONIC DEHYDRATION

Fig 24—Late rapid or hypotonic dehydration

This state occurs in untreated severe cases of fluid and electrolyte loss. There is now a marked depletion of water from both compartments. The K loss in the urine is excessive, although the urine output is markedly reduced. There is also a loss of Mg. There is no urinary Na or Cl loss. The degree of hemoconcentration, as evidenced by the hematocrit, is high.

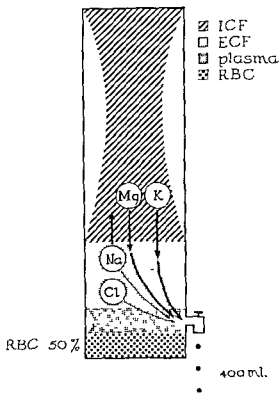
throughout the intracellular and extracellular compartments.

Signs and symptoms are severe thirst, weakness, dry, loose and inelastic skin, and oliguria, and there is no history of abnormal fluid and electrolyte losses.

Parenteral Fluid in Surgical Patients

The parenteral fluid requirements of surgical patients are estimated by a study of the three factors which bring about dehydration.

1. The deficit which has already resulted prior to the institution of therapy. This is the sum of the abnormal losses and the continued obligatory losses which have continued since the onset of the illness.



SLOW OR HYPERTONIC DEHYDRATION

Fig 25—Slow or hypertonic dehydration

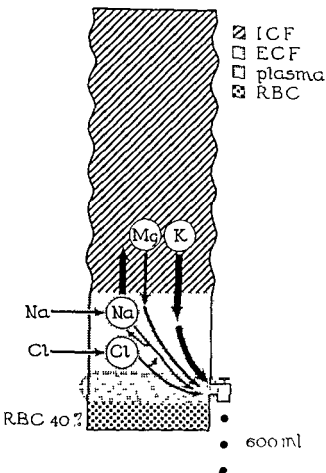
This is a condition that results from water starvation without any extraordinary loss of electrolytes. The dehydration is manifested equally in both the intracellular and extracellular compartments. There is increased K and Mg loss in the urine as a result of the intracellular fluid shift. There is an increased shift of Na into the cells to compensate for the ionic K loss. There is a slight loss of Na and Cl in the urine, as the renal conservation mechanism of these ions has not yet come into effect. The urine output is reduced. The hematocrit tends to be elevated.

Treatment consists of rehydration by isotonic carbohydrate solutions. K may be necessary in late cases.

2. The future daily normal fluid requirements of the patient. A basic quantity of 1,500 ml. for obligatory loss and 1,000-1,500 ml. for urinary output should be allowed.

3. The correction of continued abnormal loss from vomiting, continuous gastric suction, etc.

In all surgical patients who come to operation, it is convenient to divide the fluid requirements of each patient into three time phases: the preoperative phase, the operative phase, and the postoperative phase.

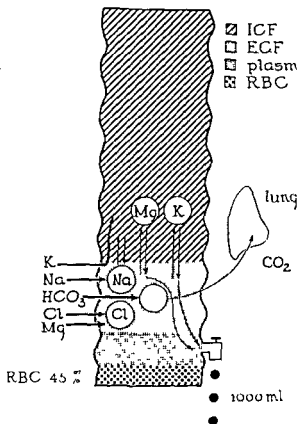


LATE RAPID DEHYDRATION TREATED WITH NaCl ALONE

Fig 26.—Late rapid dehydration treated with NaCl alone

The NaCl solution tends to overhydrate and expand the extracellular fluid compartment and to some extent the intracellular compartment as well. The excess of the Na ion tends to increase Na shift into the cells and to increase K and Mg loss in the urine. There is an increased urinary loss of Na and Cl as a result of the kidney's efforts to reduce the overabundance of these two electrolytes. The urine output lags behind the fluid intake. The hematocrit is reduced because of the retained fluid. Clinical edema may be present.

The Preoperative Phase.—The preoperative phase may not present any problem in the case of a healthy individual whose fluid needs are met by a regular oral intake of food and water. However, the problem may be complicated by a prolonged period of wasting illness, vomiting, shock, or hemorrhage, and careful parenteral therapy will then be necessary.



LATE RAPID DEHYDRATION TREATED WITH BALANCED ELECTROLYTES

Fig 27.—Late rapid dehydration treated with balanced electrolytes

The potassium deficit is met by KCl, the Mg deficit by $MgSO_4$, the Na deficit by NaCl and $NaHCO_3$, and the Cl deficit by the KCl and NaCl. Excess Cl administration is avoided by the use of $NaHCO_3$, as the HCO_3 is eventually excreted by the lungs as CO_2 . The water deficit in both compartments is corrected. The urine output and hematocrit are normal.

The history and physical examination afford the basis for estimating the amount and type of the fluid and electrolyte deficiency. The frequency, severity, and type of vomiting must be recorded. The duration of the illness enables one to estimate the amount of obligatory water loss. A knowledge of the urinary output is all-important. The physical examination shows the presence of fluid which is not available to the body, e.g., in edema, gastrointestinal ileus, etc. The degree of intestinal motility is an important point. Lack of intestinal motility usually means that there

is a large noneffective pool of water within the lumen of the bowel. Examination of the facial expression, tissue turgor, and blood pressure should always be made. The amount of thirst, the presence of sweat, and the condition of the peripheral circulation also give evidence of the degree of dehydration.

Laboratory Examinations.—There are several laboratory examinations which should be carried out in each case of dehydration. It should be remembered, however, that these laboratory tests do not afford a diagnosis of the type of dehydration. This must be made on clinical grounds. The following is a list of the determinations which are commonly used:

1. Red blood count, hemoglobin, and hematocrit
2. Urinalysis
3. Blood nonprotein nitrogen
4. Serum electrolyte estimations of sodium, potassium, and chloride
5. Carbon dioxide content of the blood
6. Blood volume estimations, using T-1824, Evans' blue dye, or radioactive tracer substances such as phosphorus, iodine, or iron
7. Serial determinations of the body weight

The Operative Phase.—Fluid and electrolyte requirements during the operation are mainly supportive. Large amounts need not be given except in the case of the patient with a high fever, or during excessive sweating as may occur on a very hot day. The most important fluid given in the operating room is the replacement blood transfusion. The time to administer blood is during the time that the actual loss is taking place. Thus the intravascular shift of the interstitial fluid in an attempt to maintain plasma volume is avoided.

The Postoperative Phase.—The fluid requirements should be planned to maintain the urinary output at between 1,000 and 1,500 ml/24 hours. This amount may have to be increased if there is evidence of poor urinary function. Due allowance must be made for the obligatory losses of the skin and lungs, and for the abnormal losses which may occur chiefly via continuous nasogastric suction, gastrointestinal fistula, ileostomy, etc.

Sedation

It is essential that every surgical patient have a good night's sleep before the operation. Rest must not be left to chance but should be assured by the use of a suitable soporific drug, such as one of the short-acting barbiturates. The sedative is preferably given by mouth, but subcutaneous or intravenous injection may at times be necessary.

Care of the Gastrointestinal Tract

No useful purpose is gained by the routine use of enemas or purgation. The use of these drastic measures should be limited to those patients in whom an empty bowel is necessary for the successful completion of an operative procedure, and to those patients in whom a period of postoperative immobilization is anticipated. In these latter patients it is usually sufficient to administer a simple enema on the night before the operation. However, in the case of patients who are scheduled for operative procedures on the colon or rectum, cleansing enemas should be given on the night prior to operation and repeated early on the morning of operation. Care should be taken that the enema be completely expelled, an effect best accomplished by allowing the patient to walk about for a time following the administration of the enema. Breakfast should be withheld for all patients who anticipate a morning operation which requires general anesthesia. However, for patients listed for afternoon operations, an easily assimilated breakfast should be given early in the morning. Patients who require minor surgical procedures under local anesthesia are best advised to eat their normal breakfast or other meal.

Skin Preparation

A wide area of skin about the proposed incision should be carefully prepared. All hair should be removed by meticulous shaving, and the area should be washed with soap and water. Occasionally it is advisable to paint the prepared area with an antiseptic solution and cover it with a sterile towel.

Preoperative Medication (See Chapter 8, Anesthesia.)

PREOPERATIVE AND POSTOPERATIVE CARE

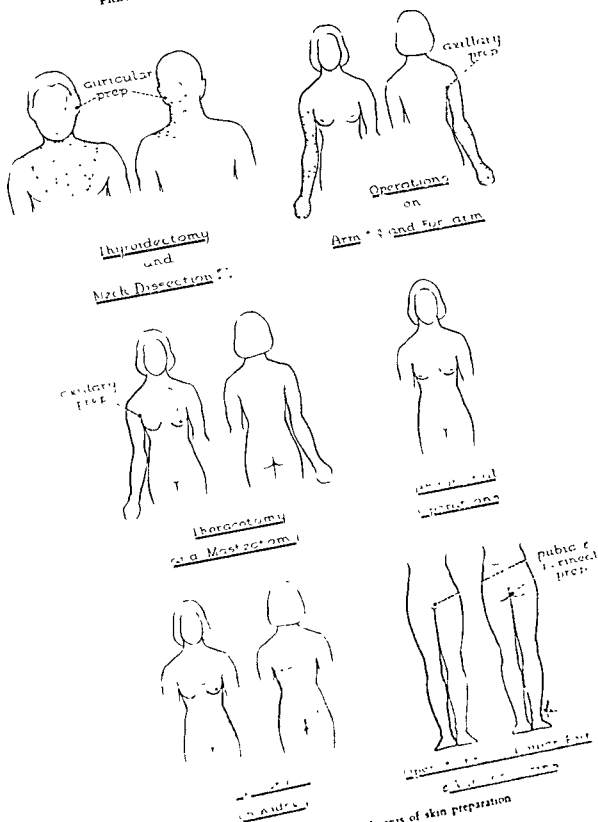


Fig. 29 Diagram of areas of skin preparation

POSTOPERATIVE MANAGEMENT

The Endocrine and Metabolic Responses to Injury

Every surgical operation is followed by a period in which the patient needs special care in order to minimize his discomfort and to prevent the complications. There is a systemic reaction to bodily injury characterized by various changes principally in the adrenal cortex, posterior pituitary, and the metabolism of protein and electrolytes. This is a normal response occurring in the healthy individual which presumably is due to a compensatory reaction to a state of stress.

Endocrine.—

Adrenal Cortex.—Following operation or injury, the adrenal cortex secretes large quantities of hormones which cause metabolic changes in the patient subjected to this stress. These changes are nonspecific in character and generally are related to the magnitude of the injury. These adrenocortical changes may be measured by the eosinophil response and by the 17-ketosteroid and aldosterone levels. The normal eosinophil count in the adult ranges, on the average, from 150 to 500. Following a surgical procedure of medium severity, the count rapidly falls to about 10% of normal, if the injury is of major severity the count may fall to zero. After moderate trauma the eosinophil count will return to normal within 24-48 hours, while after major trauma the count remains low for 2-5 days. If the injury is continued as in major burns, the eosinophil count remains low for a much longer period, i.e., up to 2 weeks.

As in the case of the eosinophils, the urinary *hydroxycorticoids* are affected by the degree of trauma. A moderate degree of injury produces an increase from the normal excretory level of 5-10 mg/24 hours, up to 20-25 mg/24 hours. Major injuries produce increases up to 50 mg/24 hours. The increased excretion lasts about a day after moderate injury, 3-5 days after major injury, and up to 2 weeks following a severe burn. The blood steroids follow a similar pattern after trauma.

Following trauma the urinary level of *aldosterone* (normal 4-8 μ g.) rises rapidly for 24 hours and then falls to normal. This rise

appears to be due to either an increased output of ACTH or psychic stress. The sodium and potassium response which accompanies this phenomenon cannot be ascribed to the rise in aldosterone but is due to the increase in glucocorticoids.

Posterior Pituitary.—Following surgery there occurs a change in water metabolism characterized by a decreased water excretion and a tendency of the body to conserve water. While not proved to be due to the antidiuretic hormone liberated by the posterior pituitary, the evidence strongly suggests that this is the case.

Thyroid.—There is an increased oxygen requirement following injury. This is readily measured by serial B.M.R. determinations which show increases up to 50-60 following injury and a return to normal in 5-6 days.

Metabolic.—Following operation or injury there is an increase in nitrogen loss in the urine. In surgery of moderate degree there is a slight increase in the nitrogen loss, which is in part due to starvation, in part due to stress. Following major surgery or trauma, the nitrogen loss is greatly increased, up to 15 Gm/day, and even as high as 25 Gm/day in severe crushing injuries and burns. Negative nitrogen balances may continue for a period of 1-2 weeks, even when the nitrogen intake is normal. During most of this catabolic phase, nitrogen appears in the urine as urea and occurs chiefly as the result of breakdown of skeletal muscle plus the general effect of starvation in the early posttraumatic phase. In addition to the protein derangements which result from injury, similar upsets occur in the metabolism of carbohydrate, fat, and vitamins.

Electrolytes.—Following injury potassium is lost as a result of starvation and the catabolic effect of injury. After major trauma, the urinary potassium loss in the first 24 hours ranges from 50-70 mEq. This amount decreases to 20-30 mEq per day within 3-4 days. This potassium loss is greater than can be accounted for by starvation. In starvation the potassium-nitrogen ratio is about 3 mEq/Gm., but following injury the ratio is about 5-10 mEq/Gm.

The excretion of sodium and chloride is contradistinguished to potassium is reduced fol-

lowing injury. This conservation of extracellular ions is greater than can be accounted for by starvation alone. The serum sodium levels are reduced in spite of the retention of sodium, a reduction probably due to a retention of water and a shift of sodium into the intracellular compartment.

If the body is to survive following injury, these metabolic changes must take place. Whether such changes are caused by or accompany the alterations in adrenal steroid metabolism is uncertain, but they do occur together.

Patients who have had major surgical procedures require continuous and expert observation during the initial postoperative phase. This management is ideally carried out in a well-equipped recovery ward which is staffed by personnel, professional and nursing, specially trained in the use of resuscitation measures and in the accurate recording of data. Patients should remain in such a ward until the acute postoperative phase is over.

Pain

Pain in the operative area is often accentuated by the anxiety of the patient. The most effective method to relieve pain is the hypodermic injection of morphine sulfate, gr $\frac{1}{8}$ - $\frac{1}{4}$ (7.5-15 mg.), repeated at 4-hour intervals when necessary. This drug should be used with caution, especially in the older age groups because of its depressant effect on the respiratory center. Care should be taken that doses of morphine larger than absolutely necessary are not used; 10 mg. of morphine sulfate/70 kg. of body weight will, under usual circumstances, afford adequate pain relief. Doses in excess of this amount lead to a great increase in uncomfortable side effects, e.g., nausea, vomiting, and respiratory depression, and add as a rule little to the degree of pain relief. Of course, if severe pain occurs, larger doses are indicated. Demerol, 100-150 mg., may be substituted for morphine. This drug produces less respiratory depression and does not cause the unpleasant side effects of nausea and vomiting, which so frequently occur following the administration of morphine. It should only be necessary to continue these drugs for the first 24 hours postoperatively. If discomfort per-

sists, milder analgesics such as codeine and acetylsalicylic acid may be given. Apprehension is best relieved by reassurance, supplemented in some cases by suitable barbiturates. Chlorpromazine is a useful adjunct, both as an analgesic and a sedative. The combination of chlorpromazine with Levo-Dromoran is particularly useful in the pain of advanced malignancy. The use of intravenous ethyl alcohol 5% and intravenous procaine 1% is frequently helpful in controlling pain on a short-term basis. Local nerve blocks occasionally are helpful.

Fluids

In the immediate postoperative period, the fluid requirements of the surgical patient are increased due to operative blood loss, increased sweating, gastric or other intestinal drainage, and vomiting. In the absence of nausea and vomiting, the oral administration of water may be started in small amounts as soon as the patient has recovered from the anesthetic and may be increased as rapidly as his tolerance permits. If the patient cannot take his fluid requirements by mouth, parenteral fluids should be given in such an amount as to ensure a urinary output of 1,000-1,500 ml. daily. It is extremely essential that careful records be kept of the fluid intake and output of each patient, in order that the fluid balance may be regulated. (See Chart 1.)

Diet

The details of diet vary widely with the various operative procedures. The aim should be to return the patient to a full and adequate oral intake as soon as possible. Following minor surgical procedures, and in the uncomplicated case, the patient can take enough food to maintain his nutrition, but after major surgical procedures it is frequently necessary to supplement the oral intake by parenteral means.

The important points of any dietary program are as follows:

1. To resume a normal oral intake at the earliest moment.
2. To supply a maximum amount of nourishment in a volume which can be readily taken by the patient. This nourishment must be presented in a palatable form.

ROYAL VICTORIA HOSPITAL

DAILY INTAKE AND OUTPUT

NAME _____ CASE No. _____ WARD _____

[illegible]

MR 20

Chart 1.

3 To supply nourishment, by special dietary means, to patients who have undergone operations which prevent a sufficient oral intake, e.g., to postgastrectomy patients for whom supplementary intravenous amino acids may be used; to postjejunostomy and gastrostomy patients for whom specially prepared liquid feedings are required

The following basic formula affords a balanced diet for patients following a gastrostomy or a jejunostomy. This concentrated formula must be reached after a 14-day period of graduated feeding increases.

Gastrostomy and Jejunostomy Feeding

Whole boiled milk	1,500 ml
Water	500 ml.
Dextri-Maltose (No 1 or 2)	100 Gm
Strained liver	4 (3½ oz) tins
Fat emulsion (Lipomul)	100 ml.
Protein powder (Gestal)	60 Gm.

Analysis

Protein	135 Gm.
Fat	115 Gm.
Carbohydrate	190 Gm
Calories	2,335
Volume	1,650 ml

Vitamins	adequate
Minerals	adequate
Sodium	47.24 mEq/L
Potassium	50.52 mEq/L
Chlorine	9.05 mEq/L
Phosphorus	57.4 mEq/L

The feedings are increased according to the following plan:

1st day	Whole boiled milk	500 ml.
	Water	500 ml.
3rd day	Whole boiled milk	1,000 ml.
	Water	500 ml.
5th day	Whole boiled milk	1,500 ml.
	Water	500 ml.
	Dextrin-Maltose	
	(No. 1 or 2)	50 Gm.
7th day	Whole boiled milk	1,500 ml.
	Water	500 ml.
	Dextrin-Maltose	
	(No. 1 or 2)	100 Gm.
	Strained liver	1 (3½ oz.) tin
9th day	Whole boiled milk	1,500 ml.
	Water	500 ml.
	Dextrin-Maltose	
	(No. 1 or 2)	100 Gm.
	Strained liver	2 (3½ oz.) tins
11th day	Whole boiled milk	1,500 ml.
	Water	500 ml.
	Dextrin-Maltose	
	(No. 1 or 2)	100 Gm.
	Strained liver	3 (3½ oz.) tins

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13th day	Whole boiled milk	1,500 ml
	Water	500 ml
	Dextri-Maltose	100 Gm
	(No 1 or 2)	4 (3½ oz) tins
	Strained liver	100 ml
	Lipomul	

Most patients are benefited by dietary supplements in the form of high protein drinks which should be given in the preoperative and postoperative periods. A useful and palatable formula is as follows:

High Protein Eggnog

<i>Recipe</i>	100 ml
Cream 15%	1,300 ml
Milk	5 only
Eggs	20 Gm
Sugar	75 Gm
Skim milk powder	30 ml
Vanilla	1,700 ml
Volume	

Analysis per liter

Protein	60 Gm
Fat	55 Gm
Carbohydrate	75 Gm
Calories	1,036
Sodium	36.0 mEq/L
Potassium	54.7 mEq/L
Chlorine	42.7 mEq/L
Phosphorus	41.8 mEq/L

Oral feedings are not to be attempted until the motility of the gastrointestinal tract has returned to normal. The resumption of normal motility can be ascertained by abdominal palpation, abdominal auscultation, and by the evidence of the passage of flatus per rectum. Stomach emptying can be gauged by the aspiration, at timed intervals, of measured amounts of fluid which have previously been introduced into the stomach via an indwelling nasogastric tube.

Care of the Lower Gastrointestinal Tract

The diet during the first few postoperative days ensures little residue. There is, therefore, no necessity for an evacuation before the third postoperative day. During this time, a rectal tube may be used to relieve flatus. If the bowels do not move by the third or fourth postoperative day, a small enema may be given. If enemas are ineffectual, rectal examination should always be done to exclude impaction of feces in the rectum. Fecal im-

paction can also produce symptoms of diarrhea and tenesmus.

Should fecal impaction occur, the feces should be softened by oil retention enemas, and if no evacuation occurs, manual removal becomes necessary.

Ambulation

The patient should be turned on alternate sides every hour and should be encouraged to breathe deeply and to cough. His respiratory excursions may be increased by the administration of carbon dioxide (5%) and of bed as soon as possible, but no hard and fast time schedule should be followed; rather each patient should be judged individually. His general condition, the nature and extent of the operation, and the presence or absence of complications must be considered. It is important to insist on early active movement. *The practice of lifting a patient into a chair where he remains immobile until lifted back into bed does not constitute early ambulation.* Many patients, including those who have had abdominal operations, may be allowed up in 24 hours, allow to walk to the bathroom after 48 hours, and within 4-5 days may be permitted to be in and about most of the time.

Such a program adds immeasurably to the well-being of the patient and results in fewer pulmonary complications, briefer convalescence, and easier nursing care. The incidence of wound disruption and postoperative hernia has not been increased. However, phlebotrombosis and subsequent pulmonary embolism may still occasionally occur in patients who have followed this regime.

Care of the Wound

In the so-called clean case, in the absence of fever, undue pain, or discharge in the operative area, the dressings need not be disturbed until the time for removal of suture. Heavy dressings should be avoided. Repeat inspections of the wound are unnecessary, in addition to the patient's discomfort, and may predispose to infection.

All changes of dressing must be done using the most scrupulous aseptic technique. In order to avoid infecting himself, or the trans-

PREOPERATIVE AND POSTOPERATIVE CARE

in and feel the ribs expanding against the hands. On expiration, a gentle pressure should be given with the hands at the end of the movement.

The patient is taught localized breathing exercises, that is, diaphragmatic and lower lateral costal breathing. It is most important to teach these exercises for at least two days prior to operation, in order to thoroughly ventilate the lungs and also to ensure that the patient fully understands what he will have to do postoperatively. If the patient does not know this before the operation, it is often very difficult to teach him afterward when he is in a state of shock and suffering pain. Most patients require treatment for about three days after the operation—they are treated twice a day if necessary.

Pulmonary Embolism.—Pulmonary embolism usually follows thrombosis of the veins of the legs and pelvis. In contrast to pneumonia and atelectasis, pulmonary embolus rarely occurs before the end of the first week after operation. Fatal embolism is often preceded by smaller, nonfatal infarcts.

The clinical manifestations depend upon the size of the embolus and its location. If a main pulmonary artery is occluded, the symptoms are those of acute respiratory distress with circulatory collapse. If a smaller branch is blocked, the patient has pain in the affected side of the chest, dyspnea, cyanosis, fever, and hemoptysis. X-rays in the early stages frequently fail to demonstrate the lesion.

Treatment.—Careful daily examination of the legs for signs of thrombosis should always be done. At the first sign of involvement of the veins, anticoagulant (Marcumar) therapy should be instituted. If pulmonary embolism occurs, antishock measures such as papaverine, atropine, and antispasmodics should be instituted and antispasmodics should be given. Oxygen and anticoagulant therapy must be continued. If recurrent embolism occurs, ligation of the inferior vena cava should be carried out.

Fat Embolism.—Fat embolism usually follows severe trauma and fractures and is due to the presence of fat globules in the circulating blood. The etiology is obscure and the condition may be due to either a disturbance in general fat metabolism or the liberation of fat globules at the site of trauma.

The symptoms are predominantly cerebral or pulmonary. The patient is restless, confused, and possibly comatose and may develop dyspnea, cyanosis, and signs of pneumonitis. Petechiae are often present over the upper thorax and neck. The diagnosis is established by the presence of fat in the sputum or urine. Treatment is directed toward the relief of shock and pulmonary complications.

Gastrointestinal Complications

Nausea and Vomiting.—Nausea and vomiting are common in any patient who has had a general anesthetic and are aggravated by excessive intake of fluids or food in the immediate postoperative period. Many patients are sensitive to morphine, which may prolong and increase vomiting. If vomiting is prolonged, it becomes potentially dangerous because of the resulting dehydration and chloride loss. In such cases, continuous gastric suction should be instituted and parenteral fluids given.

The use of certain proprietary drugs, Dramamine, Gravol, or Stemetil (prochlorperazine), by oral, intravenous, or rectal routes is useful in the so-called idiopathic nausea of the postoperative period. On no account should these drugs be used to alleviate the nausea and vomiting of postoperative ileus or some other similar pathologic state.

Abdominal Distention.—Following every abdominal operation a certain degree of temporary paresis of the intestines occurs, but the bowel musculature quickly regains its tone. Accumulation of gas within the intestinal lumen and the ineffectual efforts to expel it result in the so-called *gas pains*. The atony persists in some cases, due to prolonged or rough handling of the bowel, retroperitoneal hemorrhage, peritonitis, or operations on the retroperitoneal tissues. If this complication is not recognized early and treated promptly, increasing distention will result. The major amount of intestinal gas is derived from swallowed air, with only a small proportion coming from fermentation of food and interchange of gases with the blood stream. It is much easier to prevent than to treat *ileus*. In the early stage, a rectal tube may permit the passage of flatus with marked relief. Early prompt use of gastric intubation with continuous suction will

prevent distention in the majority of cases. Intestinal intubation with the Miller-Abbott, Harris, or Cantor tube will overcome ileus if the tube can be passed into the small bowel. In our experience, a single wide lumen tube fitted with a mercury bag, such as is provided in the Cantor tube, is the most satisfactory type. (See Fig. 29.) Drugs have a limited field in the treatment of intestinal ileus. Morphine enhances both the tone and the peristaltic action of the intestine, especially of the small bowel. (See Chapter 24, Intestinal Obstruction.) As a rule, no drugs appear to be of value until peristalsis is resumed.

dilated stomach. Continuous suction on the stomach tube should be maintained until the gastric tone is restored. One questions whether this is a true disease entity or a manifestation of paralytic ileus.

Hiccough.—Postoperative hiccoughs may occur following operations on the abdominal viscera, the genitourinary tract, the diaphragm, or the central nervous system. It is thought to be a reflex phenomenon due to stimulation of the afferent nerve terminals in the diaphragm. It is usually transitory and responds to symptomatic treatment, but it may be prolonged and very refractory to therapy.

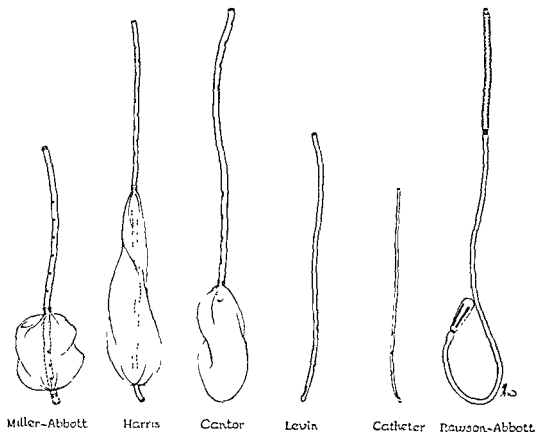


Fig. 29—Types of nasogastric tubes in common use

Acute Dilatation of the Stomach.—This condition may follow any operation and is regularly associated with paralytic ileus. There is loss of gastric tone, and large quantities of gas and fluid accumulate in the stomach. The enlarged dilated stomach can be palpated, and large quantities of fluid are vomited. The treatment depends upon early diagnosis and the immediate removal of the contents of the

Inhalation of carbon dioxide (5-10% in oxygen) is often effective. As hiccough frequently accompanies an ileus or stenosis in the intestinal tract, gastric suction may bring relief. Chlorpromazine given parenterally will usually relieve hiccough of idiopathic origin. In the intractable case it is occasionally necessary to interrupt the phrenic nerve, either by local anesthesia or operative procedures. If

PREOPERATIVE AND POSTOPERATIVE CARE

this is done the patient first should be fluoroscoped in order to determine which side of the diaphragm is producing the hiccough.

Parotitis.—This condition is an uncommon postoperative complication. The invading organism, usually a staphylococcus, enters via the parotid duct. A dirty mouth in a dehydrated patient is undoubtedly a predisposing factor, and the condition generally occurs in debilitated individuals.

Pain, swelling, and tenderness in the parotid region accompanied by pyrexia appear within a few days of operation, and pus can often be expressed from the opening of the parotid duct.

The treatment of oral sepsis and the prevention of dehydration will prevent the occurrence of parotitis. Once the infection is established, antibiotic therapy is of great value, combined at times with small doses of x-ray to the parotid region.

Urinary Complications

Urinary Retention.—Inability to void is common in the immediate postoperative period and is, as a rule, transitory. Many patients experience difficulty in voiding while recumbent, getting the patient out of bed frequently enables him to empty his bladder. Parasympathetic drugs are sometimes useful. If such measures fail, catheterization is necessary. Overdistention of the bladder predisposes to infection, and catheterization should be done before overdistention occurs. Great care must be taken to avoid introducing bacteria into the bladder during this procedure. If retention persists, an indwelling catheter may be introduced, and tidal drainage is occasionally indicated.

Oliguria.—Some degree of oliguria always occurs in the first postoperative day. This is due to a variety of factors: fever, vomiting, intake, excessive sweating, the specific effect of hyperventilation, the effect of the use of adrenal cortex on the kidney, and the use of continuous gastrointestinal suction. Oliguria is arbitrarily assumed to be present if the urinary output is less than 500 ml./24 hours. This amount is the minimum urinary output required for the excretion of nitrogenous waste products if the renal function is normal. This initial period of oliguria is not alarming, par-

ticularly if the urinary specific gravity is high. Persistence of oliguria means either an inadequate fluid therapy or renal damage. If the specific gravity is persistently low or if the patient has a marked positive fluid balance or if he develops edema, impaired renal function should be immediately suspected.

Anuria.—Anuria may complicate surgical operations, especially when accompanied by hypotension and shock. Dehydration and previous kidney disease are predisposing factors. Following severe burns, incompatible blood transfusions, etc., lesions of the lower nephron nephrosis type occur, and the functional derangement may lead to complete anuria. In many cases the kidneys begin to function spontaneously after a few days. Great care must be taken that the patients are not overloaded with fluids during this period. (See Chapter 31, Genitourinary System.)

Wound Complications

Infection.—Infection should not occur in a clean case. If it does, a careful review of the operation and dressing techniques must be made. Infection is usually restricted to a small part of the wound, but a generalized wound infection and spreading cellulitis may occur. Clinically the patient runs a normal course for several days and then develops fever and pain at the operative site. Examination of the wound may show swelling and redness, but often a localized point of tenderness is the only indication of underlying infection. The wound should be reopened, the pus evacuated, and antibiotics administered.

Hematoma.—Hematomas may occur in any operative wound, the incidence is inversely proportional to the surgeon's care in effecting hemostasis. Small hematomas will be absorbed spontaneously but predispose to infection. Large hematomas should be evacuated under anesthesia and the wound resutured, as retained blood clot markedly delays wound healing.

Disruption of the Wound.—This condition occurs more commonly in the aged and obese, in patients with debilitating disease, such as cancer, and in those who experience vomiting, distention, or cough in the postoperative period. Incomplete wound disruption is the

basis for almost all incisional hernias. It commonly occurs about a week after operation.

Wound disruption should be suspected if there is any constant outpouring of straw-colored or blood-stained fluid. Frequently the patient experiences a sensation of something breaking or, as he says, "letting go," particularly during a paroxysm of coughing. A small piece of omentum appearing in the wound should not be confused with subcutaneous fat.

To minimize the incidence of wound disruption, the patient's nutritional status should be satisfactory before operation; the incision should be closed carefully and without tension on the suture line; and wounds of debilitated patients should be closed with through-and-through nonabsorbable sutures.

Treatment.—The wound should be immediately resutured with through-and-through nonabsorbable sutures, unless the patient's condition is so critical that any operation is contraindicated, in which event adhesive strapping may serve as a temporary measure.

Headache

Headache is a common complaint following spinal anesthesia. It is frontotemporal and/or suboccipital in location. The pain is usually absent when the patient is lying flat in bed and develops when the patient sits up or stands. The pain is aggravated by sneezing, coughing, and bodily exertion.

The cause is somewhat equivocal, but it is most likely due to a slow leakage of spinal fluid through the puncture site in the fibrous, inelastic, and slowly healing dura. The loss of more than 10% of the spinal fluid volume causes intracranial changes, such as dilatation of the intracranial vessels, chiefly the veins, and some increase in the brain volume. Occasionally the headache is associated with a low-grade aseptic meningitis.

Headache may also be caused by extracranial muscle spasm, due to trauma from the lumbar puncture, posture on the operating table, and undue postoperative immobilization.

The treatment is unsatisfactory. The pain will normally pass off within a week. Great relief is afforded by strict recumbency. Analgesics should be used. Occasionally intracranial vasoconstrictor agents, such as ergotamine in

combination with caffeine, may be given in the proprietary tablet *Cafergot* (100 mg. caffeine nitrate and 1 mg. of ergotamine), two tablets every 30 minutes, for 4-6 doses. Hydration of the patient will also prove useful.

Skeletal muscle spasm is helped by local physiotherapy and sedatives.

PATIENTS PRESENTING SPECIAL PROBLEMS

Infants and Children

Infants and children are particularly susceptible to disturbances in metabolism as a result of operative trauma. They require a much greater caloric, water, and salt intake per kilogram of body weight than do adults.

The effects of dehydration are more profound and occur more rapidly in infants and young children. The average daily fluid intake of infants and young children is approximately 700 ml/24 hours, and the urinary output is of the same order. The extracellular compartment of these patients is about $\frac{1}{10}$ that of an adult, i.e., $1\frac{1}{2}$ L. Therefore, any decrease in the fluid intake will have a rapid and profound effect on the small amount of body fluid. Likewise, any increased loss of fluid, either through vomiting or excessive sweating, will soon deplete the extracellular compartment and lead to a severe dehydration. The normal infant requires about 150 ml of fluid/kg of body weight daily. In the surgical patient this amount should be given by a slow, continuous intravenous drip, the rate not to exceed 30-60 ml/hour. The blood volume of infants is extremely small. Therefore, any operative procedure should be carried out with a replacement blood transfusion. This should not exceed 25 ml/kg. at any one time, unless there is an inordinate degree of blood loss.

The Elderly Patient

The elderly patient reacts less favorably to trauma than does a younger person. Advanced age is frequently associated with nutritional deficiencies and cardiovascular, renal, or respiratory disease, any of which will add to the operative risk. However, with careful preoperative and postoperative care, proper choice of anesthetic agents, and meticulous surgical

technique, the average elderly patient will come through even an extensive surgical procedure without difficulty.

The postoperative phase of treatment is most important, for patients even with outwardly good cardiovascular or renal function may develop a breakdown of these systems, and special precautions must be taken to forestall any such event. An adequate urinary output is most important to ensure that the nitrogenous waste products are excreted, because the renal concentrating power may be reduced.

To help guard against cardiac complications, salt intake should be restricted for several days prior to any major surgical procedure. Intravenous feeding must be done with great care to avoid overloading the circulation with ill-chosen or rapidly administered solutions.

Respiratory complications frequently develop in the elderly patient from concomitant chronic respiratory disease or from inability to cough up retained bronchial secretions. Assiduous nursing care and prophylactic chemotherapy will prevent most complications of this type.

It should be realized that the chronologic age of a patient is a poor guide to his physiologic state, and the planning of therapy should be based on his physiologic age. Persistent vomiting in an elderly patient does not rapidly deplete the patient's reserve of water and salt because his gastrointestinal secretions are smaller in volume and have a lower electrolyte content. However, the blood volume of elderly patients, particularly if their nutrition has been poor, is frequently low, and small losses of fluid may lead to peripheral collapse. This small blood volume, the so-called state of chronic shock, should be treated with judicious preoperative whole blood transfusions, and extremely carefully judged replacement blood transfusion during any operative procedure.

The Patient With Cardiovascular Disease

The patient with cardiovascular disease, whether young or old, who undergoes a surgical procedure does so with added risk. However, except in the presence of cardiac decompensation, or following a recent cardiac accident, or in severe congenital heart disease, this risk should not be inordinately great.

Patients with chronic rheumatic heart disease, angina pectoris, and hypertension without stand extensive surgical procedures without difficulty, if certain precautions are observed. The preoperative sedation should be sufficient to allay apprehension, yet not enough to cause marked cerebral or respiratory depression. Anesthetic agents must be administered with extreme care to avoid struggling during the stage of excitement, and anoxemia and hypotension must be prevented at all times. Intravenous fluids must be given very carefully to avoid overloading the right side of the heart, with the dangerous consequence of pulmonary edema. Nonelectrolyte crystalline solutions are usually well tolerated, but sodium chloride is best avoided because of its tendency to increase the blood volume. Whole blood and blood plasma should only be given to replace their loss.

The most important practical point in assessing the operative risk in cardiac patients is the degree of exercise tolerance and the presence or absence of cardiac decompensation. The use of digitalis and quinidine is not indicated, unless cardiac irregularity or failure develops during the postoperative period.

The Patient With Renal Disease

The patient with chronic renal insufficiency constitutes a great problem to the surgeon. The impaired powers of concentration and selective absorption of the renal tubules require that special attention be paid to fluid and electrolyte requirements, to ensure adequate excretion of waste products and a satisfactory electrolyte balance. The greater the degree of impairment of function, the greater the urine volume required. Renal failure or shutdown, which can occur with great suddenness in cases with impaired renal function, usually occurs after a period of hypotension or dehydration, and these events should be avoided at all costs. Acute renal disease, such as pyelitis and nephritis, should be treated before any but emergency surgical procedures are carried out.

The routine urinalysis is the best guide to the detection of renal disease. The finding of albumin, casts, red blood cells, or a low fixed specific gravity is sufficient to warrant a complete urinary tract investigation and should

include biochemical studies, e.g., NPN and creatinine, urea clearance test, a test of the excretory power of the kidney by the phenol-sulfonphthalein reaction, and a test of the urinary concentration power of the kidney (Mosenthal). Special roentgenologic tests and instrumental examination of the kidney and lower urinary tract should also be carried out

The Patient With Respiratory Disease

Chronic respiratory disease augments the risk by leading to an increased incidence of postoperative pulmonary complications. With the impaired respiratory excursion and the deficient aeration of the lungs, most anesthetic agents are poorly tolerated. Careful attention to oxygen supply and a free airway is most essential during operations on patients with chronic respiratory disease. Postural drainage with parenteral and aerosol chemotherapy should be used during the preoperative and postoperative phases.

Acute respiratory infections necessitate the postponing of elective surgical procedures. If an emergency arises, inhalation and parenteral chemotherapy should be employed. In any case of pulmonary disease, particular effort must be directed to the clearing of tracheal and bronchial secretions by coughing, tracheal aspiration, early movement, and ambulation.

The Obese Patient

Obesity, particularly in the older patient, increases the operative risk, first, because of its frequent association with metabolic disturbances or with organic, cardiac, or respiratory lesions, and, second, because of the difficulties encountered in carrying out surgical procedures, especially those within the abdomen.

Weight reduction should be attempted prior to elective surgical procedures. However, in the event of emergency, the added risk must be accepted.

Diabetes in the Surgical Patient

Surgery in the Presence of Diabetes.—The surgical management of the diabetic patient necessitates close cooperation between physician and surgeon. If the hypergly-

cemia is not controlled, the incidence and severity of infections will be increased, delayed wound healing may occur, and glycosuria, acidosis, and coma may result. In the presence of diabetes, operations may be performed with comparative safety so long as the diabetes is under control. It should be remembered, however, that the operative trauma will temporarily aggravate the diabetic state.

Emergency Operations.—These present a difficult problem. In infections and trauma, the needs of the body for insulin are increased, and the dosage must be adjusted accordingly. The operative risk in these patients is greatly increased. If a true emergency exists, every attempt should be made to control acidosis, but if the added risk is both necessary and appreciated, it may still be advisable to proceed with surgery even in the presence of ketosis.

The acute abdominal case is of special interest. In many of these, e.g., acute appendicitis, the symptoms are milder in the diabetic. It should also be appreciated that in patients in diabetic acidosis or impending coma, acute abdominal symptoms may develop, which may be easily confused with those of appendicitis and pancreatitis.

Localized collections of pus in the diabetic patient may be called a diabetic emergency. The presence of retained pus has an adverse effect upon the diabetes, making its control difficult or impossible. As drainage operations outside the abdominal cavity are of relatively short duration and can be done under light anesthesia, operation can be undertaken without delay, even in the presence of uncontrolled diabetes.

Preoperative Care.—It is important that the glycogen and protein reserves of the liver of the diabetic patient be augmented by a diet containing adequate amounts of carbohydrate and protein. Because of the widespread arteriosclerosis, a careful evaluation of the cardiac and renal status should be made before any elective operation.

In general, protamine insulin is more difficult to use in the immediate preoperative and postoperative periods, and so a change should be made to the quick-acting crystalline form which permits more exact regulation. On the

day of operation the caloric needs of the patient can be met by intravenous glucose, which should be covered by insulin (1 unit of insulin per 5 Gm. of glucose). No attempt should be made to render the urine completely sugar-free because of the dangers of hypoglycemic reactions. It is safer to maintain a moderate degree of hyperglycemia in the preoperative and postoperative periods than to risk the development of insulin shock.

Anesthetic.—In the selection of an anesthetic, three factors should be kept in mind:

1. **Toxicity.** General anesthetics, especially chloroform and ether, should be avoided if at all possible, because there is a risk of producing fatal damage to a glycogen-depleted liver.

2. **Length of action.** The sooner after operation the patients are able to take carbohydrate food, the easier will be the management and the less likely the development of ketosis.

3. **Liability to produce postoperative vomiting.** The anesthetic of choice, therefore, is one with a low toxicity, rapid elimination, and a low incidence of nausea and vomiting. For operations of a minor nature, nitrous oxide and oxygen should be used. Even in abdominal surgery these gases combined with curare seem free from deleterious effects. For major surgery, both nitrous oxide supplemented with curare and spinal anesthesia are the agents of choice.

Postoperative Care.—An adequate fluid intake is important for the diabetic patient. During the operative period blood and saline may be used as necessary. Glucose, if required in the postoperative period to meet caloric requirements, should be covered by insulin, though care should be taken to avoid hypoglycemia. Oral feedings should be given as soon as tolerated and increased until an adequate diet is attained.

The following routine may be used as a guide for the insulin and carbohydrate requirements for diabetic patients who are undergoing surgical procedures. It is most important that a clear description of the diabetic state, including the insulin requirements, should accompany the patient to the operating room so that intravenous fluids may be cov-

ered by the necessary insulin. The standard form which is used at the Royal Victoria Hospital for this purpose is as shown in Table 6.

General Considerations.—

1. Other than emergencies, all operations, if possible, should be performed in the morning.

2. Use crystalline zinc insulin for the immediate postoperative period.

3. The diet should have contained at least 200 Gm. of CHO/day on the two days preceding the operation. There should be no acidosis, and the patient should be free from glycosuria in a 24-hour specimen of urine.

4. The blood sugar should be slightly elevated or within the normal range.

5. Less insulin will be required postoperatively if the operation reduces sepsis. The decreased insulin requirement is gradual and is usually not apparent for 12 hours.

6. Preoperative sedation and the choice of an anesthetic agent should be no different from those of the nondiabetic patient.

Specific Considerations.—

1. Day of operation—major surgery: If the patient has been on protamine zinc insulin, give no insulin and no breakfast.

It is safer to place the patient on crystalline zinc insulin when the operative procedure is a major one, when the convalescence is expected to be prolonged or stormy, or when in doubt. The dose of insulin required can be calculated by estimating the preoperative requirements per 10 Gm of carbohydrate, i.e.:

Protamine zinc = $\frac{2}{3}$ crystalline zinc
 NPH insulin = $\frac{1}{3}$ crystalline zinc
 Patient's intake of carbohydrate was 250 Gm
 Patient's insulin requirement was PZ 21
 CZ 12

Therefore the amount of CZ insulin was 48 units
 = 2 units of CZ insulin/10 Gm carbohydrate
 or 10 units of insulin for every 50 Gm of glucose

Fractional urines should be used to help judge insulin requirements.

2. On return from the operating theater, give 1,000 ml of 5% glucose-saline intravenously, with the number of units of CZ insulin subcutaneously as estimated above.

3. No oral feedings should be given during the first 24 hour postoperative period.

TABLE 6
FORM ACCOMPANYING DIABETIC PATIENT TO OPERATING ROOM

Patient's last name				First	Age	Sex	Case No
Duration of diabetes		Mild	Moderate	Severe	Stable	Unstable	
Complications of diabetes							
Maintenance		Diet			P.	F.	C
Maintenance		Insulin					
Preoperative preparation							
Date of last blood sugar		AC		PC		PCL	
Time of last urinalysis		Sugar			Acetone		
Suggested fluid in O R and recovery room							
Suggested insulin coverage		(1) In bottle			(2) SC		
<div style="text-align: right;">_____ MD.</div> <div style="text-align: center;">Physician</div>							

ANESTHETIC SECTION

Time		Type of anesthetic		Duration	
Fluids in O R type ml		Time of infusion	Beginning	End	Insulin in O R
Fluids in recovery room ml		Time of infusion	Beginning	End	Insulin in R R.
TOTAL FLUID	ml	TOTAL			
Time discharged			Anesthetist		
Remarks					

4. In the second 24-hour period postoperatively, give 20 Gm. fluid feedings every 3 hours for six feedings. Preceding every second feeding, the required number of units of CZ insulin should be given.

5 The diet on the first day of three meals should be protein 70 Gm., fat 50 Gm., and carbohydrate 150 Gm., assuming the preoperative diet had been protein 70 Gm., fat 50 Gm., and carbohydrate 250 Gm

Minor Surgery, and Surgery Carried Out Under Local Anesthesia—

1. The preoperative insulin dosage is the same as that described above under major surgery

2 On return from the operating theater, give 1,000 ml. of 5% glucose saline, intravenously, with 16 units of crystalline zinc insulin, subcutaneously

3. At 6 P.M., 8 P.M., and 10 P.M. on the day of the operation, give 200 ml of orange juice

4. Give 12 units of insulin before the 8 P.M. feeding, i.e., at 7:30 P.M.

5. On the following day, three meals may be instituted and preoperative control re-established

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Chapter 6

Injuries Due to Physical Agents

Thermal, Irradiation, Electric, and Chemical Trauma

Hamilton Baxter, MD

THERMAL BURNS

The prevalence of injuries from heat may be realized when it is shown that nearly 8,000 people in the United States and an estimated 60,000 in the world die each year from this cause. These figures become insignificant when compared with the hundreds of thousands of people who are hospitalized for this type of injury and suffer various degrees of disfigurement each year. When one calculates the staggering number of man-hours of work lost to industry, the hospital beds required, and the hours of medical and nursing care needed, it is indeed remarkable that so few preventive measures have been adopted. The widespread use of noninflammable or fire-resistant clothing by the civilian population would greatly reduce the number of cases requiring treatment on this continent each year.

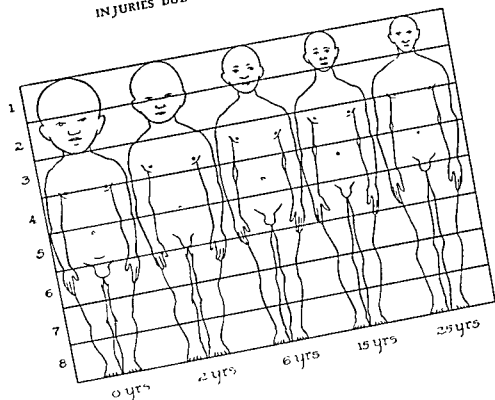
Between World Wars I and II certain marked physiologic disturbances in the burned patient were observed. First the deficiency of electrolytes and fluid was noted. Then it was shown that plasma was extravasated into the burned area with resultant hemoconcentration and that plasma administered intravenously was beneficial. Finally the infective phase of burns was recognized and various therapeutic measures advocated for treatment. As a result of the large number of burns suffered by civilians and military personnel in World War II, in-

tensive research was carried out, and several major advances in therapy resulted. These may be summarized as follows:

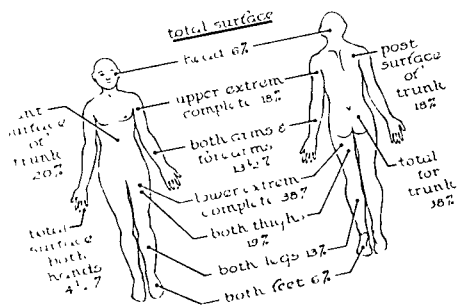
1. The development of improved methods of treating shock and hemoconcentration
2. The wide adoption of the occlusive pressure dressing and the exposure method
3. The dermatome, which enabled surgeons to obtain accurately and easily any thickness and amount of skin grafts desired
4. The discovery and use of sulfonamides, penicillin, and newer broad-spectrum antibiotics
5. Improvement in methods of nutrition
6. The resurfacing of granulating areas at the earliest possible time, which automatically prevents most of the chronic sequelae of burns

The original classification by Dupuytren of the severity of a burn into six degrees was simplified, for reasons of practical convenience, to include only the following three divisions: *first degree burn*, which causes reddening of the skin; *second degree burn*, which involves the deeper layers, although regeneration is possible from the remaining hair follicles, sweat, and sebaceous gland ducts; and *third degree burn*, which destroys the entire skin and any or all deeper structures, including bone. Thus, in modern terminology, the third degree burn includes the fourth, fifth, and sixth classifications by Dupuytren. Healing of

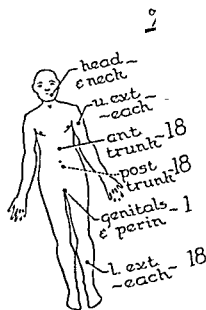
INJURIES DUE TO PHYSICAL AGENTS



A



B



C

Fig. 30—A, A comparison is made of the changes in per cent surface area of the head and extremities between children and adults
 B, Berkow's method of calculating the extent of burns
 C, "Rules of Nine" proposed by Wallace

a first degree burn is complete in a few days, while one of second degree will require about two or three weeks, depending on the number and viability of epithelial structures remaining. A variable amount of scar formation results. The third degree burn, if small, may heal by epithelial ingrowth from the edges or by contracture, but large areas should be skin grafted as soon as the depth of the burn is evident. Although a number of methods for determining the depth of thermal burns have been proposed, the most reliable test is that of visual inspection at intervals, due allowance being made for the region of the body and the usual hair distribution.

The Berkow table for adults and its modification for children should be used to calculate the percentage of the body surface burned, so that the amount of plasma and fluids to be administered may be determined. For example, the percentage of surface area of the head of a 1-year-old child is twice that of a 15-year-old child. As an alternative method Wallace has proposed the "Rules of Nine" and divides the various extremities and the anterior and posterior aspects of the trunk into areas of 9% or multiples thereof. By either method a rough estimate of the areas of first, second, and third degree burns should be charted for comparison with the final outcome. Very young and aged individuals are more liable to suffer from shock than healthy adults. Burns of the face, abdomen, and buttocks in children, although limited in area, sometimes result in shock. Severe burns of more than 50% in patients over 50 years of age very frequently result in death. For every decade over 50 years of age, the percentage of body burn which will cause a high mortality decreases markedly. Patients over 60 years of age may die from a relatively minor burn because of coexisting conditions, such as cardiac, pulmonary, renal, or arteriosclerotic disease. Malnutrition or anemia may require treatment as well as the burn.

Physiologic Derangements

Shock.—Immediately following a burn, pain is experienced which varies with the type of injury. This may cause syncope or death within an hour or two despite prompt therapy.

Blood Flow.—Due to the painful sensations, there is a brief period of blanching of

the skin followed by vasodilatation of the vessels of the skin and subcutaneous tissue. Unless charring has occurred, there is a marked increase in capillary permeability with extravasation of plasma, particularly the albumins, into the interstitial spaces. The more extensive the burned area, the greater will be the amount of plasma lost from the vascular system. This results in hemoconcentration, which is well established in a few hours and is so characteristic of thermal burns.

Loss of Plasma and Increase in Lymph Flow.—The protein content of the escaped fluid is estimated at about 4%, and from 0.4-2.3 Gm. of nonprotein nitrogen may be lost per square inch of burned surface in 24 hours. The albumin/globulin ratio of bleb fluid is greater than that of plasma. The flow decreases as the burn heals. The lymph return is increased from a burned area, and this flow is not reduced by an occlusive burn dressing. No definite toxic substance has been found in the lymph returning from an area of thermal burn in man. For several days the total lipids, total cholesterol, and fatty acids are markedly elevated in lymph flow from a burned area but remain unchanged on the unburned side.

Anemia.—Immediately following the burn there is local heat destruction of red cells in the area of burned skin and subcutaneous tissue. This anemia is temporarily masked by the hemoconcentration which occurs. Cope has shown that in extensively burned patients not over 10-12% of the red cell mass was destroyed by the initial thermal trauma. While large amounts of whole blood may not be required in treatment of burn shock during the first 48 hours, it is necessary to give frequent transfusions to almost all severely burned patients after the first week. Increased fragility of red cells, reduced synthesis of hemoglobin, and bleeding from the burn wound contribute to the progressive anemia which continues until the granulating areas are covered with skin grafts and infection is overcome.

Permeability.—The burned surface permits flow of diffusible substances in either direction. Penicillin, Aureomycin, erythromycin, and other antibiotics are present in the plasma exuding from the burned surface in amounts which would encourage bacteriostasis. Al-

INJURIES DUE TO PHYSICAL AGENTS

though bacteria do not invade intact epithelium of a burned surface even if coagulated, once this defense is broken, they may cause local inflammation or bacteremia if favorable conditions arise

Fluid, Colloid, and Electrolyte Imbalance in the Burn Wound.—The burn wound draws water, protein, and electrolytes from the injured and permeable capillaries into the extravascular spaces, and thus the wound swells with edema. If the burn is limited, the patient will survive without special fluid therapy. On the other hand, unless early and adequate replacement therapy is given in extensive burns, the patient may die. Burn trauma not only increases the permeability of the capillary membranes so that much plasma is lost into the wound, but the nervous reflex mechanism controlling blood flow is disrupted so that increased arterial flow to the burned area occurs, and this in turn augments the filtration of fluid into the wound. The burn wound fluid has a much higher concentration

of plasma protein than that of normal tissue and lymph. Following a mild burn the protein concentration of bleb fluid may be no greater than 3 Gm. % and after a moderate burn no more than 4 Gm. %. A severe burn rarely gives a concentration of over 4.5 Gm. %. The rate of edema formation is relatively proportional to the severity of the burn. With increasing tissue pressure, the lymphatic flow is at first rapidly increased, but as edema pressure reaches a maximum, the lymph flow decreases but does not return to normal. In burns in human beings, edema appears to reach its maximum between the 36th and 42nd hour. Apparently capillary permeability has decreased sufficiently so that filtration no longer exceeds resorption of fluid. The cycle of changes is shown in Figs 31-38.

Pathology.—The thermal agent may produce various degrees of injury: erythema, edema, or even necrosis. These three types of damage may be present in different areas of a burn. Flame burns are likely to produce

Fig 31.—The normal protein levels of plasma and interstitial fluids are shown, as well as the movement of extracellular and intracellular electrolytes.

Fig 32.—The initial effect of the burn upon protein concentration of plasma is an increase. The protein concentration of the burn wound fluid, although much higher than that of normal tissue fluid and lymph, rarely rises above 4.5 Gm.

Fig 33.—The part of the plasma protein which did not pass with the plasma fluid into the burn wound remains in circulation, and its elevated colloid osmotic pressure draws fluid from the unburned tissue into the blood stream, so that a dilute plasma is the second phase of protein and fluid redistribution after a burn. During the initial phase (Fig 32), the concentrated plasma could not resorb fluid from the burn wound because of the damaged capillary membrane.

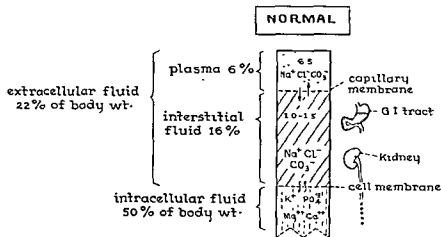
Fig 34.—Here the wound has continued to swell. More fluid has been drawn from the interstitial space into the blood stream with a rise of colloid protein in the interstitial fluid. Water is drawn from the cells into the extravascular space, and with it the intercellular electrolytes, potassium and phosphorus are believed to pass out of the cells. Kidney function ceases and permanent renal damage may occur. Death may occur partly from dehydration, partly from anoxia.

Fig 35.—If the burn is not extensive, redistribution of fluid and colloid occurs. With healing of the capillary membranes, leakage into the wound diminishes, and protein concentration of the fluid escaping into it is diminished. Lymph flow into the blood stream continues, and the wound edema subsides. The plasma recovers its normal pattern, and the cells regain their normal content of water and electrolytes.

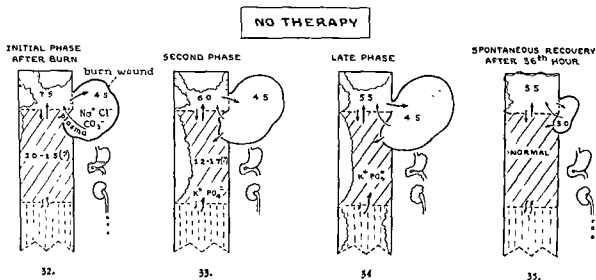
Fig 36.—This shows the effect of using whole plasma alone. The amount of plasma given equals the weight of fluid lost in the burned area. It is good but not ideal therapy since there are enough water and electrolytes but too much protein. Administration of whole blood alone gives somewhat similar results.

Fig 37.—When sufficient electrolyte solution is given to refill the circulating plasma volume the fluid distributes itself generally. The protein content of all spaces is diluted. Widespread edema occurs, and pulmonary edema may develop. The cardiovascular and renal functions tend to fail.

Fig 38.—A mixture of colloid and electrolyte solutions is injected into the blood stream in such concentration and volume as to replace completely the water, electrolytes, and protein lost in the wound. Much or all of the electrolyte solution may be given by mouth if desired. This provides ideal therapy for burn shock.



31.

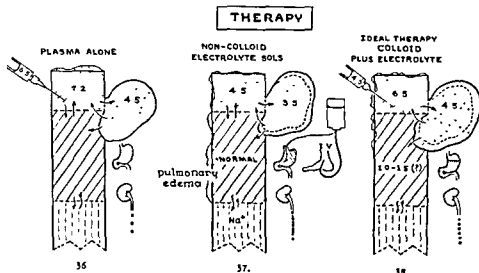


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Figs 31-38—(See opposite page for legends)

dry necrosis, while steam, hot water, and other fluids tend to cause a moist necrosis. A *first degree burn* is marked grossly by hyperemia, and if sections are examined, a mild exudate of serum and leukocytes will be found. Desquamation is followed by regeneration and healing. A *second degree burn* is characterized by vesication and various degrees of injury to the dermis. Edema of the skin occurs with bleb formation and hyperemia of the deeper elements, and necrosis of part of the dermis. Regeneration occurs from the remaining hair follicles, sweat, and sebaceous glands. The *third degree burn* is marked by necrosis of the whole thickness of skin. An eschar forms which sloughs off, leaving an ulcer. Epithelial regeneration can occur only from the margins of the burn. This is the type of burn in which severe contractures occur if skin grafting is not performed at an early stage.

Systemic Manifestations

Primary shock is of neurogenic origin and results in generalized vasomotor collapse. It is seen shortly after injury and is characterized by pale, clammy, moist skin, shallow respirations, and a weak pulse. In severely burned patients, death may occur from this cause before a physician can institute treatment, or even in spite of it.

Secondary shock occurs subsequent to primary shock, lasts for 2-3 days, and is characterized by hemoconcentration and the escape of plasma into the tissues.

Due to the increased red blood cell concentration and increased viscosity of the blood, there is decreased cardiac output and decreased blood flow to certain vital organs. The liver and kidney are particularly affected, and due to decreased blood flow, renal function may be drastically reduced so that the tissues and tubules of the kidney may never regain their function, and death from uremia may result.

The normal functions of the liver are retarded when there is an inadequate supply of blood and oxygen, so that glycogen cannot be stored, and deamination is prevented.

Lungs.—Direct exposure to flame or hot gases may cause some injury to the respiratory tract, usually only as far as the trachea. The bronchiolar epithelium is more likely to be

injured by chemically irritating gases. Carbon monoxide poisoning and lowered oxygen content of air may become major lethal factors when fire occurs in enclosed spaces. Early tracheostomy is important if there is evidence of pulmonary injury due to heat or irritating gases.

Adrenals.—Following burns as well as other forms of stress, there is adrenal hyperactivity mediated through the pituitary with increased output of adrenal hormones, as revealed by eosinopenia and a rise in output of urinary ketosteroids. In certain systemic conditions the adrenal may not be able to react adequately, with subsequent collapse.

Thyroid.—In chronic burn patients with extensive granulating areas, hyperactivity of the thyroid gland has been postulated. There is a rapid pulse, considerable weight loss occurs, and the basal metabolic rate may be greatly increased. In some patients a basal metabolic rate of plus 40 has been recorded. However, this is not due to increased activity of the thyroid gland but to increased oxygen consumption caused by proliferating granulation tissue. It disappears when the burn heals or is skin grafted.

Acute toxemia overlaps secondary shock and lasts for 7-10 days. Those who survive secondary shock and subsequently die usually do so in this period. Whether it is due to toxic substances or to physical changes in the circulating blood or is caused by anemia and septic infection is uncertain. When extensive second and third degree burns of from 30-80% of the body surface occur, death frequently results in spite of intensive colloid and electrolyte replacement therapy. In elderly individuals, death may be due to the additive effect of coincident cardiac, renal, pulmonary, or systemic diseases, together with the thermal burn. However, in young, previously healthy adults, a syndrome characterized by disturbance of electrolyte levels, marked mental depression, and peripheral vascular collapse without evidence of invasive clinical infection from the burned surface may occur. Various causes have been attributed to this syndrome, such as loss of function of the skin as an organ, toxic effects of undetected septicemia, or collapse of the pituitary-adrenal endocrine system. The

toxemia in burns is probably a manifestation of invasive infection and septicemia. At times this downward progression may be altered to complete recovery by combined autogenous and homologous skin grafting of granulating areas, adequate blood transfusions, and good nutrition. Nevertheless, in some patients the pulse becomes rapid and the skin cold and cyanotic, and hyperpyrexia may develop. Restlessness progressing to stupor and a final fall in blood pressure indicate approaching death.

Chronic sepsis is due to the retention of pus under dead skin or wound granulations and is eliminated by removal of eschar and skin grafting.

TREATMENT OF BURNS

First Aid

Minor Burns.—Very minor burns about the home, particularly in children, are the cause of much screaming on the part of the child,



Fig. 39—Marked emaciation which develops in patients with extensive third degree burns when skin grafting is delayed beyond the optimum period

In children, convulsions often precede the demise. Sudden respiratory failure may occur, and increased intracranial pressure with compression of the medulla oblongata has been found. Microscopically, severe interstitial edema and ganglion cell changes, most marked in the hypothalamus, have been noted.

anxiety to the parents, and frequent calls to the doctor. For psychologic reasons mainly, but also to protect the wound from trauma and to minimize discomfort, treatment should be initiated by the parent. The part should be cleansed, grease or other contaminants should be wiped away with absorbent cotton, and

sterile gauze dressings lightly bandaged to the burned part. After a few days the dressing may be changed. The great majority will have healed, and the few deeper injuries may be treated by the doctor as required.

Serious Burns.—Emergency treatment should consist of wrapping the burned area in sterile (or clean) dressings, towel, sheet, or blanket and transporting the patient to hospital as soon as possible. Give morphine, 10-16 mg., subcutaneously or intravenously, as indicated.

Immediate Definitive Treatment

General Therapy.—

1 Sterile masks for attendants and patient if possible. Remove burned clothing and place on sterile sheet in dressing room or operating room. Take blood for hemoglobin, hematocrit, hemogram, blood proteins, and electrolytes. Ask or otherwise determine weight of patient.

2 Estimate extent of burn accurately, using Berkow's Table or the "Rules of Nine" proposed by Wallace.

3 If burn shock may be anticipated (15% of body surface or more in healthy adults, or 10% in babies, young children, or elderly persons), do not wait for signs of shock to appear, but give intravenous colloid or electrolyte solutions according to the following formula.

First 24 hours

(a) 1 ml./kg. for each 1% body surface burned of blood, plasma, or plasma substitute (give part whole blood and part plasma, dextran, or polyvinyl pyrrolidone).

(b) 1 ml./kg. for each 1% body surface burned of electrolyte solution, physiologic saline.

(c) 2,000 ml. of 5% dextrose in water. The colloid and electrolyte solutions should be administered intravenously, using a large needle or a cannula inserted in the vein following a cut down. In the first 8 hours, one half the total estimated fluid requirements for the first day should be given, one quarter in the second 8 hours, and one quarter in the last 8 hours. If no vomiting is present, 2,000 ml. of 5% dextrose may be given by mouth, otherwise the intravenous route is used.

Second 24 hours

Reduce colloid and electrolyte requirements to one half the total amount for the first 24 hours. The 2,000 ml. of 5% dextrose in water is continued.

4 Obtain urine for *urinalysis*, insert Foley catheter in patients with burns of 15-20% or

over. Maintain hourly urine output of 25-50 ml. in adults and 15-25 ml. in children. Adjust rate of fluid administration to maintain this output.

TABLE 7
REQUIRED URINE OUTPUT FOLLOWING BURNS

AGE	ML. PER HOUR (AVERAGE)
4-7 years	25
7-10 years	30
10-14 years	33
Over 14 years	50

5. *Antibiotic therapy* should be commenced without delay. Large quantities of broad-spectrum antibiotics should be used. It has been shown that these are present in considerable quantities in the plasma escaping from the burned surfaces and that bacteriostatic levels are obtained against most hemolytic streptococci and micrococci, which predominate the bacterial flora of recent burns. An enhanced effect may be obtained by combining penicillin with streptomycin, or chlor-tetracycline with oxytetracycline. Subsequently, the most effective antibiotic may be chosen, following culture and sensitivity tests to antibiotics of various bacteria obtained from the burn wound. The patient should be questioned regarding sensitization to antibiotics, and those known to cause reactions should not be administered.

6. *Oxygen* should be administered by nasal catheter, mask, or tent if there is evidence of burns of the mouth and larynx. Inhalation of steam or hot irritating gases may cause obstruction of the trachea or edema of the lungs. A tracheostomy may be necessary to facilitate aspiration of the bronchi. Pulmonary damage due to irritating gas or vapor may necessitate reduction in the total amount of fluid administered, electrolyte solutions being replaced in varying degrees by whole blood.

7. Give a booster dose of *tetanus toxoid* or 1,500 units of tetanus antitoxin.

8. It is generally agreed that the use of *corticotrophin* or *cortisone* should be limited to the treatment of very severely burned and critically ill patients during the early stages of the trauma. Remarkable recovery has occasionally been observed following an initial dose of 800 mg. of cortisone, which was reduced

rapidly to the usual therapeutic dose during the course of a few days

9. Following a minor burn, patients can usually take a fairly substantial diet by mouth. However, following an extensive burn, the patient may tolerate only a limited amount of fluid or soupy diet. In some instances vomiting occurs, and all fluids must be given intravenously. It may be necessary to provide continuous gastric suction by means of a nasal catheter for a few patients, because of persistent vomiting. In such cases, blood electrolytes should be determined at intervals and measures taken to restore normal levels of the various electrolytes.

Local Therapy.—The total area of a burn and some estimate of depth of injury should be carefully recorded, since these factors determine to a considerable degree the amount of plasma and electrolyte therapy administered. The fact should never be forgotten that *treatment of the general condition of the patient takes precedence over local care*, unless two teams are immediately available. Failure to observe this rule has permitted many patients to slip into irremediable shock while a perfect dressing was being applied. If at all possible, the patient should be taken to an operating room where, under aseptic conditions, the burned areas are cleansed and application of a dressing is carried out. Adequate doses of morphine are given, but general anesthesia is not employed. The local care of the burn wound is the same whether the exposure method or an occlusive dressing is to be applied. Gross dirt or oil is removed from the burned area with large quantities of warm sterile water and hexachlorophene, 1% cetrimide, or white soap on cotton balls or sponges. Blisters are drained, but the skin over them is not removed. Any loose tags or rolls of epithelium are removed with scissors. Cleansing is carried out with a minimum of trauma to the exposed dermis.

Despite the imposing list of over 70 methods of treating burns, it should be recognized that variations in treatment must be made because of the age of the patient, the location and depth of the burn, and the agent causing the burn. The purpose of any method of local treatment is to limit infection and minimize further skin damage. This may best be achieved

by promoting dryness and relative immobility of the burn wound. These conditions may be accomplished by one of two techniques generally used at present—the *occlusive dressing method* and the *exposure method*. Each has its own advantages and disadvantages and indications for use, depending upon the site and type of burn.

OCCCLUSIVE DRESSING TECHNIQUE—The burned surface is dressed with dry fine-mesh gauze or gauze impregnated with petrolatum or other bland ointment. After applying a number of layers of flat gauze dressings the whole is covered with a bulky layer of cotton waste which is held under moderate pressure by an elastic bandage or adhesive tape.* Plywood or plaster splints may be applied to prevent movement. The dressing is not changed for 7-14 days, depending on evidence of infection. This should be carried out in the operating room, with preparation having been made either to debride necrotic tissue or to apply skin grafts.

Advantages—

1. The dressing provides pressure on the burn wound, which may reduce *very slightly* the amount of fluid lost into the extravascular spaces.
2. The thick dressing provides protection from cold and bacterial or other contamination.
3. When thermal burns are combined with fractures, the dressing may be reinforced with splints or plaster, to provide protection for the combined injuries.

Disadvantages—

1. The dressings require much storage space and periodic resterilization.
2. Application of the dressing requires a moderate amount of time and trained medical personnel.
3. When the dressing is applied too tightly, it may cause a tourniquet effect or nerve damage with permanent injury to an extremity.
4. Respiratory movements of the thorax and abdomen are restricted.
5. Hyperthermia may be caused when a large area of the body is covered with the dressing.

*A large occlusive burn dressing, prepared in two sizes (18 x 18 inches and 18 x 36 inches) and manufactured commercially may be used if desired.

6. It must be removed to inspect the burn lesion at intervals.

7. In extensive deep second degree burns the plasma exudate may soak through after a few days and require a change of dressing to prevent ingress of bacteria from the outside.

Exposure Technique.—Apparently this method was first used at least 60 years ago, but did not gain many adherents at that time, probably because of the difficulty in controlling infection and also due to the fact that skin-grafting techniques were primitive and usually delayed too long after the burn. This resulted in suppuration, serious systemic reactions, and late burn contractures. Recent experiences with this method have shown that various parts of the body can be quite satisfactorily exposed by different methods of elevation or suspension, so that contact with bed clothing or other objects is prevented. The burned area is cleansed as in the occlusive dressing method but is left exposed to the air of the ward. Nursing care is concentrated on protection of the burned surface, and a bed-cradle, covered with a clean sheet and blanket if necessary, will help to keep the patient warm. The limbs are positioned to avoid contact with the bed clothing and to limit edema and are restrained to prevent cracks in the crust. This limitation of movement is achieved by a clove-hitch soft bandage applied to unburned parts of the limbs. In partial-thickness burns, a dry, brown crust forms in 48-72 hours, while the full-thickness burn caused by fire presents from the first a hard, dry eschar.

HEAD AND NECK.—The exposure method is particularly suitable for these areas and prompt healing occurs unless the burns are very severe. If the eyelids have been deeply burned, tarsorrhaphy should be performed early to avoid the formation of corneal ulcers; skin grafts are applied as soon as possible. Any discharge from the eyes, nose, or mouth is carefully removed at intervals. The neck should be kept in hyperextension. If the cranium or other bones of the body are exposed, multiple small drill holes should be made in the bone. Split skin grafts may then be applied when sufficient granulations have grown through the holes and proliferated.

UPPER EXTREMITIES.—Burns of the hand and forearm may be treated by keeping the elbow flexed and the hand elevated. When almost the entire upper extremity is involved, it is possible in most cases to support the limb by sterile linen slings or other supports at areas which are not seriously damaged. For example, the palm is frequently only slightly involved, and a sterile, padded support placed in it will aid in elevating the arm without jeopardizing healing of the hand. Suspension of the hand and arm may be obtained by passing steel sutures through the nails and attaching them to a spreader. If the fingers are deeply burned circumferentially, it is advisable to use the occlusive dressing method with the wrist extended and all joints of the fingers flexed to 30 degrees in the position of function. In certain cases of third degree burns, the eschar may be excised on the 3rd-5th day and skin grafts applied immediately.

THE TRUNK.—Only one side of the trunk may be successfully treated by the exposure method. With a cooperative patient it is possible to expose a burn involving the back, one side, and part of the abdomen by positioning the individual on one side. Circumferential burns of the trunk are not suitable for treatment by the exposure method.

GENITALIA AND PERINEUM.—These areas heal most successfully when treated by the exposure method.

Farmer has proposed that burned areas treated by the exposure method should be dusted with aluminum powder frequently until a dry eschar has formed. It was observed that the eschar was thinner and more pliable and permitted movement of joints better without cracking.

LOWER EXTREMITIES.—The method of fixation varies with the distribution of the burns. Children with burns of the buttocks and upper parts of the thighs tolerate a gallow's suspension splint very well. When the entire circumference of the legs has been burned, they must be elevated so that there is no contact with bedclothes. The feet and ankles are not involved in most burns of the lower extremities, and the weight of the legs may be supported by resting the feet on sponge-rubber pads, placed on an adjustable stand which may be raised to obtain complete exposure of the af-

fectured areas. In this fashion, burns of the legs up to the buttocks and perineum may be successfully treated.

Advantages—

1. Plasma loss from the surface of the burn is minimal after the crust forms. Formation of this usually requires 48-72 hours and occurs more rapidly in a cool room.

2. Infection is less common than with occlusive dressings, and the patients are more comfortable once the crust forms.

Disadvantages—

1. Circumferential burns of the trunk are not suitable for treatment.

2. Although superficial burns of the hands may be treated by the exposure method, it is advisable to use the occlusive dressing technique for deep burns of the hands and feet, since interference with blood supply to these extremities may occur by a contracture of the crust when the exposure method is used in deep burns.

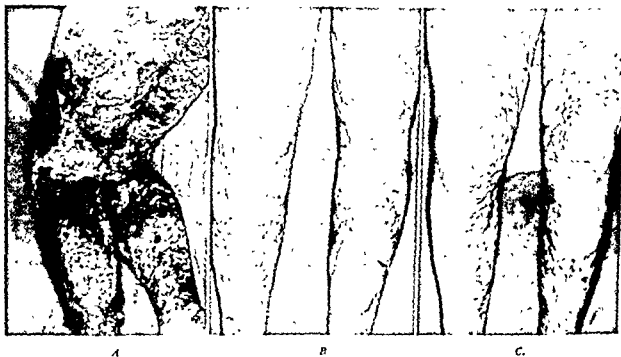


Fig. 41—A, Circumferential burns of both legs, mainly third degree. Dry, brown eschar separating just before skin grafting.

B, Posterior aspect of legs, following split thickness skin grafts from buttocks to ankles.

C, Anterior surface of legs showing skin grafting of thighs and lower legs shortly after healing of grafts.

3. There is less odor and fever, and smaller doses of narcotics and antibiotics are required. The patient's appetite and morale are better.

4. Reduction of infection and suppuration reduces the conversion of partial to full thickness skin loss. Fewer patients require skin grafting, and hypertrophic scar formation is decreased.

5. A minimal number of hours of nursing and surgical care are required after the crust forms unless skin grafting is necessary. The expenditure of bulky surgical dressings is eliminated.

Intermediate Treatment

(From 48 hours until separation of burn crust or beginning of skin grafting)

General—

1. *Fluid intake* from the 3rd day on should be kept at a sufficiently high level to provide for an adequate output of urine and insensible fluid loss as well as that from the burned surface. If signs of pulmonary edema develop, fluids should be curtailed.

2. *Blood electrolyte levels* should be determined at intervals and measures taken to correct either excessively high or low levels.

3. *Antibiotic therapy* should be continued, but changes in the agent employed may be necessary because of allergic response, development of bacterial resistance or impairment of bone marrow.

4. *Blood transfusions* Determine hemoglobin level frequently. After treatment of burn shock, the need for red blood cells depends upon the volume of these cells destroyed immediately by heat. It has been estimated that not more than 10-12% of the red cell mass is destroyed in extensively burned patients. Red cell loss occurs from cells made more fragile and hemolyzed, sludging of blood, and later on from bleeding from exposed granulations and débridement of slough. The hemoglobin should be maintained at 85% or higher.

5. *Urimalysis* at regular intervals. The Foley catheter may be removed when an adequate daily urine output has been maintained for a number of days.

6. *Nutrition* As a result of lowered food intake, deranged nitrogen metabolism, fever, and infection, an individual suffering from extensive burns loses weight rapidly, and there is marked wasting of muscles. To combat this, a diet high in protein, calories, and vitamins should be given as soon as tolerated. A suitable diet consists of 150-200 Gm protein, 250-300 Gm of carbohydrate, and 100 Gm. of fat. Sulfur-containing foods should be provided, as well as large amounts of vitamin C and vitamin B complex. If vomiting is present in the early stages, intravenous therapy must be substituted until about the 3rd post-burn day, when most patients can take food. Blocker has employed a small pump to force a semifluid diet continuously through a nasal catheter which is inserted into the stomach. Mildly burned patients can tolerate a higher food intake than those severely burned.

7. *Hyperthermia* Remove occlusive dressings if used, and place patient in oxygen tent with thermostat set at lowest temperature. If fever remains high, apply ice packs to unburned areas. Remove ice bags before temperature reaches normal so that hypothermia will not occur.

8. *Hormone therapy* Intramuscular injection of 10-50 mg of testosterone daily has been found of value in improving the general condition of the debilitated burn patient. In

spite of earlier favorable reports, corticotrophin and cortisone should be employed only in the treatment of critically ill patients and then in large doses. It is generally agreed that they do not significantly depress formation of granulation tissue, impede the "take" of autogenous skin grafts, or prolong the life of homologous skin grafts in man.

9. *Active exercise* Planned bed exercises are most important in reducing nitrogen loss, maintaining vital capacity of lungs, and in improving morale. Less severely burned extremities should be exercised first, for while active movement is important, it should not interfere with healing of raw areas. Manipulation and passive stretching are condemned. Hydrotherapy is not indicated unless the stage of healing of the part can tolerate soaking in water.

It should not be forgotten that a severely burned individual requires endless encouragement and freedom from financial worry concerning his family or relatives.

Local.—

1. When a burn has inflicted a partial skin loss, the normal skin regeneration will finally separate the crust and expose a healed surface. This occurs in the average case from the 10th-14th day in superficial burns, and in deep second degree burns from the 14th-28th day. In third degree burns the depth of the burn may frequently be recognized after a few days, when the eschar becomes translucent so that the subdermal fat may be seen. The eschar also sinks below the level of the surrounding skin, which has been less seriously damaged. If the heat-coagulated skin is allowed to remain too long, pus will collect beneath it. This should be removed as soon as observed, by cutting away the eschar over the collection of pus and applying wet dressings to the exposed granulating area. When cracks develop in the dry crust, usually about joints, the area on either side of the crack should be excised and wet dressings applied to the raw base in preparation for skin grafting.

2. *Débridement.* Removal of third degree burn slough may be carried out by either surgical excision or various types of wet dressings. Surgical excision causes considerable blood

loss, and if large areas are removed, the patient's general condition may be adversely affected. The use of Dakin's solution, pyruvic, or phosphoric acids for chemical débridement of slough is uncommon, since enzymes such as streptokinase, streptodornase, trypsin, collagenase, and proteinase are now used for the enzymatic débridement of heat-denatured skin. The latter two enzymes do not damage viable cells of the skin or cause untoward reactions in patients, other than a slight increase in the amount of fever.

Late Treatment

(From beginning of skin grafting until completion)

Prior to skin grafting the hemoglobin should be at least 85% or higher, and transfusions should be administered as required to achieve this level.

1 When the burned area is relatively small and obviously third degree, it is sometimes advisable to excise the burned tissue completely and apply a split skin graft immediately. The period of hospitalization in selected cases is reduced to a minimum.

2 When large areas of slough have been débrided, either surgically or by enzymatic débridement, it is advisable to apply a pressure dressing for several days, so that a firm bed of granulating tissue will be prepared for the reception of the skin graft. If culture of the burn wound reveals the presence of pseudomonas or proteus which interferes with the "take" of skin grafts, ointments, or wet dressings containing neomycin or polymyxin applied for a few days are effective in overcoming these bacteria.

3 Full-thickness burns of 30% or more of the body surface will cause rapid deterioration of the general condition of the patient. In such patients the use of homografts of skin should be considered to increase the patient's chance of survival and reduce the size of the parasitic burn wound with minimal disturbance to the patient. The *alternate strip method* of applying a narrow autograft of skin, followed by a wider homograft until the granulating surface is covered, permits closure of large granulating surfaces without the subsequent breakdown and sloughing of the homograft which occurs when

homografts only are used. This may be achieved with autografts $\frac{1}{2}$ " wide alternating with strips of homograft $\frac{1}{2}$ " wide. The epithelium of the autograft proliferates and spreads across the dermis of the degenerating homograft. Thus, by making the patient's own skin extend coverage of granulating areas by proliferating over homografts, it is possible to obtain sufficient autografts of skin for resurfacing of critical areas such as the hands, face, and flexor surfaces of joints, while covering other areas with alternate strip grafts. Any granulating area over 2" in diameter is worth skin grafting. Even smaller wounds should be resurfaced when they are situated near joints, in areas where contractures may form, or on cosmetically important regions.

4. With early and adequate skin grafting, contractures of the extremities, neck, lips, and eyelids which caused such hideous deformities in neglected patients will no longer occur. The later plastic treatment of healed but scarred areas, in which sloughing of tendons or parts of the ears, the nose, or other features has occurred, is carried out by means of pedicle flaps, Z-plastics, or further skin grafting. Tendon grafts, cartilage, and bone grafts are inserted as required.

COMPLICATIONS OF BURNS

Many of the complications of burns, such as contractures of joints, distortion of tissues about the neck, mouth, nose, and eyelids, may be avoided if early and adequate skin grafting is performed. Curling's ulcer of the duodenum was found in 2% of burned patients and occurred more frequently in those with extensive burns. The condition was not diagnosed until marked epigastric pain or an acute gastrointestinal hemorrhage developed. Conservative medical treatment of the bleeding ulcer is advised unless operation is necessary to save life. Marjolin's ulcer is rarely seen now, due to improved methods of treatment of thermal burns. Corneal ulcers may be prevented by suturing of the upper and lower lid margins together, and skin grafting the burned surface as soon as possible. Loss of tendons may often be prevented by early coverage of the region with split grafts or a pedicle flap. Hypertrophic scars following thermal burns are probably less frequently seen, since newer

A.



B

C

Fig 41—A and B, Explosion of magnesium incendiary bomb in hand of factory employee, with charring of hand and extensive third degree burn of arm, face, and opposite hand.

C, Result following thick split skin grafting of arm and face with scalp graft to reconstruct eyebrow. The exposed bones of the forearm were wrapped in a pedicle flap to withstand the pressure of an artificial hand

INJURIES DUE TO PHYSICAL AGENTS

methods of treating burns have been developed. This condition may be defined as an excessive production of fibrous tissue within the limits of the wound, which gradually disappears after a few months. On the other hand, a keloid is due to an excessive production of fibrous tissue not only within the limits of the wound, but with invasion of the surrounding normal skin, which may assume considerable proportion in both linear extent and thickness. The treatment of large areas of keloid following burns is by excision and skin grafting. Immediately after healing of the skin graft, adequate roentgen or radium therapy is necessary to prevent recurrence, particularly in the margins of the skin graft.

CHEMICAL BURNS AND INJURY

Following injury with strong acids and alkalis, the affected areas should be washed with copious quantities of water as soon as possible to dilute and remove the chemical before reaction with the tissue and heat production can occur. The following solution which can be used as a buffer for either type of burn should be kept available, particularly in industries where strong acids and alkalis are used: monobasic potassium phosphate (70 Gm.) and dibasic sodium phosphate (150 Gm.) in water (850 ml.) The burn should be washed with the solution, and compresses be applied every half hour for 12 hours. It is nonirritating and does not injure the eyes. Following neutralization of the acid or alkali, routine burn therapy procedure may be continued. Burns from hydrofluoric acid must be immediately washed with water and covered with magnesium-oxide cream. Powdered lime should be wiped away carefully before water is used. Routine burn treatment is then given.

Phosphorus powders should be brushed off before applying water to the part. Burning phosphorus in contact with tissue should be treated by plunging the part in water immediately. The heat of burning phosphorus, and not the acids formed during combustion, causes the deep tissue coagulation and damage seen in these burns. The phosphorus is then scraped off carefully. The area should then be washed with a solution of sodium bicarbonate (2 tablespoons to a pint of water) followed by a 1% solution of copper sulfate, which will

coat any remaining particles with a protective layer of copper phosphide. Regular burn dressings are then applied. Following severe white phosphorus burns common in wartime, systemic therapy should be started immediately. Methionine should be given in glucose saline (100 mg./L.) to minimize liver damage. Isotonic solution made up of two thirds saline and one third sodium bicarbonate should be given by mouth. Adequate amounts of calcium and vitamin K should be supplied. Surgical excision of necrotic skin and deeper tissue and subsequent skin grafting of the defect are frequently necessary.

Magnesium, which burns at intense heat, approximately 3,500° C., causes deep destruction should a flare or incendiary missile explode accidentally. Following removal of any remaining metal and grossly charred tissue, a burn dressing should be applied until final demarcation of necrotic tissue occurs and reparative treatment may be started.

Mustard gas causes blistering and deep destruction of skin. Contaminated clothing should be removed and liquid, such as moisture on the skin, should be absorbed by dry gauze. Any remaining gas or liquid should be neutralized by Dakin's solution. Subsequently the area should be washed with soap and water, dried, and a burn dressing applied.

Lewisite, which contains arsenic, acts rapidly and causes intense irritation of the skin, so all contaminated clothing should be removed and treatment started as soon as possible. BAL (British antilewisite or dimercaprol, USP) has been used with considerable success. An intramuscular injection of 3 ml. of an oil solution is administered at the earliest possible moment and repeated every 4 hours for 4-5 doses. The total dose should not exceed 50 mg./kg. body weight during a 36-hour period. Locally, lewisite may be neutralized with hydrogen peroxide, followed by washing with soap and water. Skin blebs which contain arsenic should be drained and regular burn dressings applied.

ELECTRIC BURNS

Following a severe electric shock most patients are unconscious, and about three fourths require artificial respiration. This must often be continued for some time, and the heart

into the body and quite frequently involves muscle and bone, since the blood vessels provide an excellent path for conduction of electricity. In severe electric burns when there is surface charring and probably deep damage, it is advisable to wait until slough or sequestra have separated to avoid loss of salvageable tissue and risk of hemorrhage. Granulating areas may be resurfaced with skin grafts or pedicle flaps as indicated. Extremities which have been charred or show incipient gangrene should be amputated before infective or toxic symptoms appear.

RADIATION INJURY

Acute Total Body Radiation.—Explosion of an atomic bomb by military intention may be expected to produce about 15% of casualties suffering from radiation injury alone. The problems of triage are complicated by the fact that the sensitivity of man to massive total body radiation is not known. It is generally estimated that 400 r constitutes LD_{50} in man, but some will succumb to 200 r, while others will survive high dosage. Estimation of the amount of radiation received, by a dosimeter, is not completely reliable, since it or part of the body of the individual may be shielded. General those beneath the hypocenter of the bomb may be assumed to have received a higher dose of radiation than others a thousand yards or more toward the periphery. Clinically, patients suffering from acute radiation syndrome may be divided into three groups:

Group 1.—These have received supralethal dose and recovery is improbable. Vomiting starts radiologically or in a few hours, followed by sloughing, and death in a few

2. Hemorrhage, mainly due to profound thrombocytopenia

3. Anemia due to hemorrhage, increased rate of destruction of red blood cells, and lack of production of red blood cells by the bone marrow.

4. Disturbance of fluids, electrolytes, and acid-base balance by anorexia, vomiting, diarrhea, and fever. Although pretreatment by various measures will reduce the mortality in acute radiation injuries, no specific therapy is available as yet which is effective when administered following exposure. The only methods available at present are antibiotics, blood transfusions, and other supportive measures until regeneration of bone marrow occurs.

Acute Localized Radiation Injury.—Individuals employed in atomic energy plants or exposed to radioactive substances, x-ray machines, or fluoroscopes in hospitals or industrial laboratories may receive large doses of ionizing radiations over a limited area of the body. The hands are particularly liable to suffer. Early symptoms are edema and erythema, which become more intense in a few days. Vesicles form and may coalesce to form large blebs. Moderate or severe pain may be present. The injured area should be covered with a bland ointment, gauze, and dressing which reduce pain. Infection may be prevented by adequate antibiotic therapy. Depending upon the dosage of radiation received and the resistance of the tissues, the skin and deeper tissues may heal or slough. Separation of slough should be awaited, when skin grafts may be applied. Thus an apparently doomed finger may be saved from amputation. However, because of the effect of acute radiation in causing sclerosis and obliteration of blood vessels, the injured skin will undergo more rapid atrophic changes than it will with small dosage or chronic exposure to radiation. To avoid ulceration or neoplastic changes, it may become necessary to excise all degenerating areas and "recoat" them with a fresh, healthy thick skin graft.

Chronic Radiodermatitis.—This may result as a sequel to acute injury or from the cumulative effect of repeated small doses of radiation. Telangiectasia, atrophy of skin, depigmentation, ulceration, or keratoses may be present in any combination. Cancer may finally appear

must be functioning. Fibrillation or arrest of the heart requires treatment by cardiac massage within 5-10 minutes and may be attempted if conditions are favorable. A fibrillating heart must be stopped by a shock from the defibrillator and then restarted. Once the patient has been resuscitated, shock should be

treated in the usual way to bring the blood values to normal levels.

The frequency of these burns is decreasing because of the increased safety of construction of various electric appliances. They differ considerably from thermal burns, in that necrosis of tissue follows the path of the current deep



Fig. 35. A, Area of scalp necrosis with exposed bone following electric burn.
 B, Defect in skull after removal of necrotic bone. Dura was intact.
 C and D, Appearance after transfer of pedicle flap from abdomen to the defect in the scalp and calvarium, using the forehead as a carrier.

into the body and quite frequently involves muscle and bone, since the blood vessels provide an excellent path for conduction of electricity. In severe electric burns when there is surface charring and probably deep damage, it is advisable to wait until slough or sequestra have separated to avoid loss of salvable tissue and risk of hemorrhage. Granulating areas may be resurfaced with skin grafts or pedicle flaps as indicated. Extremities which have been charred or show incipient gangrene should be amputated before infective or toxic symptoms appear.

RADIATION INJURY

Acute Total Body Radiation—Explosion of an atomic bomb by military intention may be expected to produce about 15% of casualties suffering from radiation injury alone. The problems of triage are complicated by the fact that the sensitivity of man to massive total body radiation is not known. It is generally estimated that 400 r constitutes LD_{50} in man, but some will succumb to 200 r, while others will survive high dosage. Estimation of the amount of radiation received, by a dosimeter, is not completely reliable, since it or part of the body of the individual may be shielded. In general those beneath the hypocenter of the bomb may be assumed to have received a heavier dose of radiation than others a thousand yards or more toward the periphery. Clinically, patients suffering from acute radiation syndrome may be divided into three groups.

Group 1—These have received supralethal dosage and recovery is improbable. Vomiting starts immediately or in a few hours, followed by diarrhea, prostration, and death in a few days.

Group 2—A moderate amount of exposure has been received and survival is possible. Vomiting occurs on the day of exposure and subsides in 24 hours. There is a reappearance of symptoms in 1-3 weeks.

Group 3—Sublethal dosage has been received and survival is quite likely. There is no vomiting on the day of exposure and only mild symptoms occur subsequently.

In those who have a chance to survive, it is important to direct therapy toward the prevention or treatment of the following:

2. Hemorrhage, mainly due to profound thrombocytopenia.

3. Anemia due to hemorrhage, increased rate of destruction of red blood cells, and lack of production of red blood cells by the bone marrow.

4. Disturbance of fluids, electrolytes, and acid-base balance by anorexia, vomiting, diarrhea, and fever. Although pretreatment by various measures will reduce the mortality in acute radiation injuries, no specific therapy is available as yet which is effective when administered following exposure. The only methods available at present are antibiotics, blood transfusions, and other supportive measures until regeneration of bone marrow occurs.

Acute Localized Radiation Injury.—Individuals employed in atomic energy plants or exposed to radioactive substances, x-ray machines, or fluoroscopes in hospitals or industrial laboratories may receive large doses of ionizing radiations over a limited area of the body. The hands are particularly liable to suffer. Early symptoms are edema and erythema, which become more intense in a few days. Vesicles form and may coalesce to form large blebs. Moderate or severe pain may be present. The injured area should be covered with a bland ointment, gauze, and dressing which reduce pain. Infection may be prevented by adequate antibiotic therapy. Depending upon the dosage of radiation received and the resistance of the tissues, the skin and deeper tissues may heal or slough. Separation of slough should be awaited, when skin grafts may be applied. Thus an apparently doomed finger may be saved from amputation. However, because of the effect of acute radiation in causing sclerosis and obliteration of blood vessels, the injured skin will undergo more rapid atrophic changes than it will with small dosage or chronic exposure to radiation. To avoid ulceration or neoplastic changes, it may become necessary to excise all degenerating areas and "recoat" them with a fresh, healthy thick skin graft.

Chronic Radiodermatitis.—This may result as a sequel to acute injury or from the cumulative effect of repeated small doses of radiation. Telangiectasia, atrophy of skin, depigmentation, ulceration, or keratoses may be present in any combination. Cancer may finally appear.

1. Infection

INJURIES DUE TO PHYSICAL AGENTS

It should be stressed that such areas should be excised and skin grafted before neoplastic changes have developed.

Acute Radiation Syndrome.—It has been clearly established that radiations produce mutations and that, in general, mutations are harmful. It is difficult to estimate the extent of damage which will develop in future generations after mutant genes are induced by radiations. Most geneticists agree that the potential danger is great. It has been suggested that not

more than an average total lifetime dose of 10 r of man-made radiation should be administered to the reproductive cells. At the present time, about 3 r, on the average, are used for medical x-rays. Background radiation accounts for about 3 r, and the fall out from weapon-testing has been estimated at approximately 0.5%. The medical profession should avoid unnecessary x-ray exposure of the gonads of the patients, as long as medical diagnostic service is not impaired.

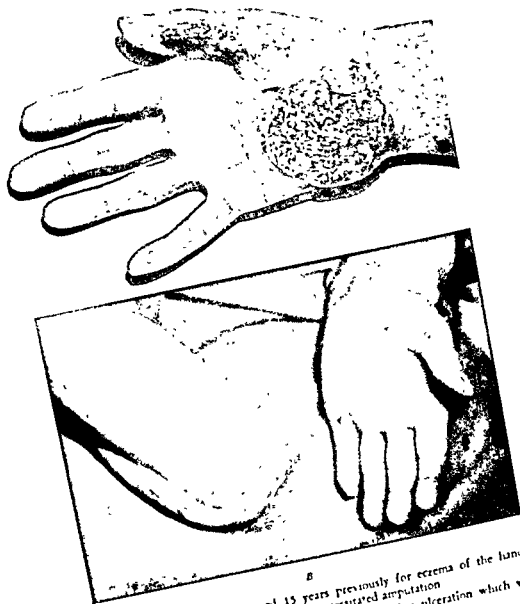


Fig. 46—A, X-ray therapy received 15 years previously for eczema of the hands, followed by development of squamous cell carcinoma which necessitated amputation
B, Healed amputation stump, with the other hand showing ulceration which will probably progress to neoplastic change

Ultraviolet Radiation.—Exposure to ultraviolet rays may cause a first degree burn, and if very extensive will result in shock and in some instances death. Extensive sunburn may be treated by bland ointments. Constant exposure to sunlight over a period of years, especially in blondes, may give rise to sailors' skin, a condition which resembles chronic radio-dermatitis and which not infrequently results in neoplastic changes in the skin.

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Early Management of the Severely Burned Patient (1955) (By Edwin H. Ellison, M.D., and Roger D. Williams, M.D., Columbus)	27 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn
Surgical Technique of the Several Types of Skin Graft (illustrates use of several new instruments for cutting of skin grafts and technique of application of thick-split and whole thickness grafts of skin to infected and uninfected wounds) (1952) (By Herbert Conway, M.D., New York)	32 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn

Injuries Due to Cold

Donald R. Webster, M.D.

Introduction

While isolated cases of frostbite are relatively common in cold climates in civilian life, it is only in military campaigns that large numbers of cases are seen. This can be a matter of grave concern, and history records the bitter price paid by armies unprepared to meet the exigencies of winter warfare. The casualties

from cold injuries may far outnumber those from enemy action. There is often difficulty in providing adequate clothing and shelter and for evacuating the wounded. Many must remain immobile for long periods in cold and wet positions pinned down by enemy fire. At sea, survivors from sunken ships may sit for days or weeks huddled in lifeboats with their

limbs immersed in icy waters and their bodies exposed to winter weather. Besides the immediate loss of effective manpower, the victims of cold injuries may have sequelae that render them permanently unfit for service or incapacitate them for long periods.

Physiology and Pathology

The effect of cold on the tissues was described by Lake and most extensively studied by Lewis. Numerous workers since then have made substantial contributions. When an extremity is exposed to severe cold, vasoconstriction occurs, evidenced by blanching of the skin. This may be followed by a bright pink color due to vasodilatation and lack of dissociation of oxyhemoglobin. Vasoconstriction then occurs and is permanent in the chilled state. All of the vascular tree is involved, even to the major vessels. This appears to be a protective mechanism to conserve body heat, but it is at the expense of the exposed limb. The phenomenon of supercooling may prevent solidification until the temperature is considerably below that of the normal freezing point. As the temperature falls, the chilled or supercooled tissue becomes solidified, although brittleness is not evident in the ordinary clinical cases.

The damage to the tissue depends on the severity of the cold and the duration of exposure. There may be injury from anoxia due to the vasoconstriction and actual damage to the cells from the freezing process. The anoxic state is augmented by capillary stasis, especially as seen in chilled limbs without actual freezing where there is escape of the plasma, leaving the red blood cells in a state of "silting" or "sludging," as described by Kreyberg and Greene. In frozen tissue considerable damage may be caused by the formation of ice crystals that continue to grow, disrupting the cells. This is not as evident in "quick freezing" as in a slower process. Experimentally, it has been shown that some tissues such as red blood cells may be frozen in glycerol and survive after thawing.

The thawing process is followed by a marked inflammatory reaction. Lewis suggested it resembled the "triple response" to histamine and thought the reaction might be due to the liberation of histamine like products from the

cellular injury. Thawing occurs either from without, in, or along the course of the vessels as circulation is re-established. Edema soon forms, with escape of plasma through the damaged capillary walls. This usually reaches its maximum in 48 hours. Stasis in the capillaries is apparently due to simple loss of plasma, possibly to the action of cold agglutinins or abnormal adhesiveness of the red blood cells. The surface temperature of the part is raised considerably, probably due to the opening of arteriovenous channels. These channels may rob the injured part of adequate circulation, as gangrene will sometimes develop in a part apparently well supplied with blood.

As edema increases, areas of cyanosis may appear, and there is always danger of infection in the devitalized areas.

There are many factors that modify the actual injury, such as the degree of cold, the duration of exposure, the surrounding medium, the resistance of the individual, and the effects of fatigue, emotion, and malnutrition.

Hypothermia*

Persons exposed to cold, who have had severe injuries, have been rendered unconscious, or are suffering from oligemic shock lose the protection of their heat-regulating mechanism, which results in a continuous fall of body temperature. This may be quite rapid in the range of 5°-15° an hour in an adult and accelerated in children. The fall in body temperature is associated with increasing stupor, and when the low 80's are reached there is complete loss of consciousness. If the exposure continues, respiration becomes slower and shallow, the heart goes into ventricular fibrillation, and contractions finally cease.

In addition to the general hypothermia, exposure of an injured person to this environment will frequently result in frostbite of the limb or face. It may be difficult in some cases, because of the slow heartbeat and shallow respiration, to be certain that death has not occurred, and such persons should not be presumed dead until electrocardiographic evidence is available. Such errors have occasionally been reported in civilian and military practice.

*See Chapter 8 for the use of hypothermia in surgical operations.

Treatment.—Most cases of hypothermia will be of a mild variety and will respond to ordinary restorative measures. Severer cases must have the rectal temperature recorded by a special thermometer, blood pressure and respiration rate charted frequently, and if the pulse is difficult to obtain, the heart action should be followed by electrocardiography. The patient should be warmed by radiant heat and given oxygen. Positive pressure may be necessary. Care must be exercised not to interpret the signs of hypothermia as being due to hemorrhagic shock and the patient transfused unnecessarily, thus overloading the circulatory system. Respiratory depressant sedatives should be avoided. Electric defibrillation may be indicated if the facilities are available.

Emergency surgical procedure may be carried out if the temperature is over 80°, with minimal anesthesia.

Many terms, such as frostbite, immersion foot, trench foot, chilblains, have been used to describe the syndrome produced by exposure to cold.

Frostbite

Frostbite is the term used to describe the condition resulting from exposure to cold of sufficient severity and duration to produce apparent solidification in the affected parts. In frostbite there is vasoconstriction that can be relieved occasionally by vigorous rubbing as is often seen in the ears. This will frequently prevent further injury, as the resultant hyperemia will enable the tissues to maintain an adequate temperature. When the defenses are overcome, freezing occurs. Anesthesia is limited to the frozen area, thus differing from that found in immersion foot, and edema does not occur until the part is thawed. The sequence of events is then similar to that of injury due to chilling or wet cold.

Immersion Foot (Trench Foot, Water Bite, Lifeboat Leg, Seaboot Leg)

Immersion foot is the name given to a condition resulting from long exposure of the limbs in icy water or to a cold moist environment. The term is inadequate as the limb need not be immersed, and the condition also occurs in other members.

The severity of the cold is not sufficient to cause freezing, but the conduction of heat from the tissues sets up a syndrome that has immediate and disabling sequelae. It resembles and is probably identical with the trench foot of World War I. It occurs chiefly in survivors from shipwrecks who are forced to sit in lifeboats, rafts, or floats with their feet dangling in icy water. Cases have been reported occurring in warmer water, but these were associated with poor nutrition and long exposure to brilliant sunlight in cramped positions. After some hours of exposure, the feet, which are at first painful, become swollen and numb, the edema involving almost all the exposed part. They are first a livid color, which later changes to a pallid waxy appearance, and if the temperature rises slightly, they become mottled with blue and green areas. After rescue and exposure to room temperature the affected limbs pass through three stages: prehyperemia, hyperemia, and posthyperemia.

Prehyperemic Stage.—At first the feet are numb, the patient feels as though he were walking on blocks of wood, and he should always be assisted. The color begins to change from pallor to lividity, and the legs show various color patterns. Vesicles and even large bullae appear. There is anesthesia of a stocking type, with occasional hyperalgesic areas. The posterior tibial and dorsalis pedis arterial pulsations cannot be detected.

Hyperemic Stage.—At ordinary room temperature the feet quickly pass into the hyperemic stage. The limbs become hot to the touch and the color a livid red. The dorsalis pedis artery becomes palpable and bounding in character. The circulation is unstable. Congestion appears when the legs are in a dependent position and blanching when elevated. The pain at this period is intense and of a throbbing, burning character. This may last for several days and then change to a shooting, stabbing pain in the dorsum of the foot, radiating to the toes. This may last for several weeks. The edema increases, and large blisters are common, especially over the dorsum and malleoli. In severe cases, portions of the foot, usually the toes, remain discolored and cold, heralding the onset of gangrene.

Posthyperemic Stage.—The hyperemic stage soon merges in a few days into the posthyper-

emic stage. The burning shooting pains subside, but the feet are subject to intermittent aching pain, and edema readily appears when the feet are in a dependent position. The circulation is unstable, as evidenced by color

sist for a long time and present a difficult problem in rehabilitation.

Treatment of Frostbite and Immersion Foot.—The aim of the treatment of frostbite or immersion foot is to prevent or minimize



FIG. 17.—Appearance of feet of patient 2 days after suffering immersion for 24 hours

changes in different positions. Excess sweating common, and the patients complain of cold mmy feet. Walking is often difficult as proprioceptive sense appears deficient. The sensitivity to cold, hyperhidrosis, and pain may per

necrosis. This has been the subject of a large amount of experimental work and the interest of many military surgeons. It was thought for a long time that slow thawing or warming was essential to allow the vessels time to recover

their tone and to limit the resulting hyperemia so that the increased metabolic demands of the injured tissues could be met by an adequate blood supply. To accomplish this, the limbs were rubbed with snow, packed in icebags, and cooled with fans or special cabinets. Experimental work in the last few years has indicated that less tissue is lost by rapid thawing than by slow warming and that stasis in the vessels is delayed. There is evidence that the larger vessels are in spasm during the chilling period and only relax when the tissue temperature is nearly that of body temperature. While the results of animal experiments cannot always be applied to man, it is probable that no harm is done by rapid rewarming of the frozen part to body temperature.

The blisters that have formed may be left intact, but if ruptured an occlusive dressing should be applied. Antibiotic drugs, antitetanic serum, and, when necessary, sedation for pain should be given. There is some evidence that anticoagulants may be useful in preventing thrombosis in the injured vessels, but they should only be used when adequate control is available. Vasodilating drugs, lumbar blockade, or lumbar sympathectomy has not been proved of value in the acute phase of frostbite nor in immersion foot where the sympathetic fibers seem to be inactive because of cold injury. They may be tried, however, if properly supervised, as an occasional case is apparently benefited by the procedure. However, in the late sequelae of painful, sweating hands or feet, where the digits become tapering and shiny and the movements restricted, sympathetic interruption is of considerable value. ACTH and cortisone have not been found useful.

Treatment must also be directed to keeping the injured parts scrupulously clean, to prevent any maceration between the toes and to protect them from infection and injury. Necrotic tissue should be carefully débrided, but amputation in the noninfected cases should be delayed as long as possible. It is surprising how often one may be deceived in mistaking superficial necrosis for gangrene of a large part. Following definitive treatment, physiotherapy is a most valuable aid to rehabilitation.

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Chapter 7

Radioactive Isotopes in Diagnosis and Treatment

Carleton B. Peirce, M.D., and Phoebe L. Cox, M.D.

INTRODUCTION

Disease of all or any part of the human body commonly is the result of some disturbance of the normal physiologic chemical processes or the physical state of the whole structure or one of its tissues. Heretofore, in the diagnosis of selected disease process, or the treatment of it, the physician has had to rely on what could be observed or determined from gross changes or end products by some means which has stopped the abnormal chemical reaction, altered the physical aberration, or diverted the irregular or abnormal physiologic process back into normal channels.

If any one of the several chemical elements and their compounds used throughout the body, or in a given tissue, can be labelled or "tagged," perhaps to microchemical levels, the true normal or abnormal physiologic process might become known. The discovery of the phenomenon of natural radioactivity and its sequel, the production of radioactive isotopes, has provided the tools.

Curiously, near the end of the last century (1895), almost simultaneously Becquerel discovered that pitchblende gave off some sort of penetrating "rays" and Roentgen found that γ could induce a similar radiation by prodding an electric charge against a relatively hard or dense metal target. Not long after, Pierre and Marie Curie isolated radium (one

of the "natural" radioactive elements) from the pitchblende ore, demonstrating that this element was the source of Becquerel's rays.

The more than 60 subsequent years have seen, almost tumbling one on top of the other, verification of Dalton's hypothesis of the structure of matter and its component chemical elements; Rutherford's identification (at McGill, 1904) of the alpha particle (α) by using atomic bombardment; his further studies here and in the Cavendish Laboratory at Cambridge, which were a foundation for Bohr's atomic theory (1912), Rutherford's proof of the existence of the proton (p) in atomic nuclear structure (1919); demonstration of the neutron (n) by his successor, Chadwick (1932). Experimental evidence has amply supported these broad principles which underlie the development of present-day knowledge of atomic and nuclear structure and radioactivity.

Further exploration of theoretic and applied physics, aided by the invention of the cyclotron (Lawrence, 1932), the discovery of artificial radioactivity (Curie and Joliot, 1934), and, during World War II, the concentration of knowledge, research, and effort on atomic energy, resulted in the control of "chain reactions" to split atoms into various pieces. The development of the nuclear reactor ("atomic pile") has made possible production of many "artificial" radioactive isotopes in usable quantity and in a variety which had been anticipated

For example, in the early work on the rate of entry of potassium from plasma into rabbit red blood cells, Hahn et al. (1939) bombarded a target of KCl with deuterons in a cyclotron. With the extracted K^{42} , an exchange rate of only 3% was determined. Mullins et al. (1941) later repeated this work and obtained a 50% exchange. In the preparation of their material, the cyclotron-produced radiopotassium was chemically purified further so as to remove the 0.02% NaCl present as a contaminant of the KCl target material. In the earlier work, the chemically insignificant sodium, when rendered radioactive (Na^{24}) in the cyclotron, had been a physical contaminant whose discharges were not distinguished by the instrument. Hence, as determined from counting its nuclear or physical discharges, there appeared to be an erroneously high concentration of isotope as predominantly an extracellular ion in the plasma.

Another type of radiochemical impurity may be due to either a different valence state or a chemical combination of the desired isotope other than the one believed to be present. For example, in the preparation of $P^{32}O_4$ by neutron bombardment of $K_2HP^{31}O_4$, a variable fraction of the P^{32} so produced (for a still unknown reason) assumes some form other than the orthophosphate. This was first brought to light in the observation that recoveries of P^{32} from such material (used as a tracer) were impossibly high in comparison to the amount of radioactivity present in aliquots of the original material precipitated as the orthophosphate of $MgNH_4PO_4$. Jacob Sacks suggests that the energy liberated in the nuclear reactions forming the P^{32} from P^{31} is sufficient to break chemical bonds, thus leading to a chemical form different from that of the target material. (A similar change may happen as a result of ionization in tissues subjected to radiation of any kind and may account for the considerable latent effects of apparently insignificant radiation.)

A third type of radiochemical impurity is the presence of two or more radioisotopes of the same element in a given preparation. For example, in the bombardment of tellurium with deuterons in the cyclotron, both Te^{130} and Te^{131} are formed. The former has a half life of 12 hours, the latter one of 8 days; both emit

negative beta particles and gamma rays. After a short bombardment period, the relative radioactivity of the Te^{130} is about 10 times that of the Te^{131} . Such a combination of isotopes of the same element can lead to results that would be difficult to interpret.

Exchange.—Another phenomenon which may cause trouble in certain types of isotope tracer investigations is exchange, especially in determination of rates of synthesis of a given compound. For example, if one wishes to study the rate of deposition of new phosphate crystals in bone, replacement of the $P^{33}O_4$ of previously deposited crystals by exchange with recently administered $P^{32}O_4$ will give an erroneously high value for neoformation of such crystals.

Similarly, it has been found that use of radioactive water (HTO) to determine by dilution the amount of body water is fraught with an inherent error due to exchange of the tritium for the H of the OH radical of carbohydrates and of peptide bonds on proteins.

Determination of Quantity of Isotope Required for Diagnostic Purposes.—A second special and practical consideration in tracer studies is the actual quantity of an isotope to be administered. Since counting apparatuses can detect the amount of isotope present in a given place in a given time, only dependent upon the character and frequency of its emitted radiation, a sufficient number of atoms must be administered to obtain a substantial count over the desired location with the apparatus available within a time period which will tire neither the patient nor the investigator. This limits the minimum amount of the isotope to be given.

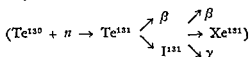
The maximum quantity should not exceed the number of atoms which may be tolerated without disturbance of the normal dynamics of the element or substance under study. Two factors affect this amount—the half-life of the isotope and the amount of "carrier" (the nonradioactive isotope of the element present in the sample of radioactive isotope to be used).

Te^{131} and Te^{132} exemplify these two characteristics. Te^{131} has a physical half-life (disintegration of 50% of the atoms present) of 8 days. Therefore, in a given location in any given period of time, such as 100 seconds—in order to have a "count" of 100 (disintegration of

100 atoms)—actually there have to be 100×10^3 atoms of the isotope present. P^{32} has a half-life of 14.3 days (a slower rate of decay). Therefore, to have the same count with P^{32} , there would have to be present 178×10^3 atoms. In both cases, the amount required will depend also upon the apparatus, which will be able to respond to only a fraction (usually known) of the total number of atoms decaying at any one moment.

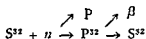
Lest the reader be staggered by the magnitude of these figures, it must be remembered that one microgram of I^{127} contains 4.75×10^{15} atoms, a similar amount of P^{32} contains 1.89×10^{15} atoms. Furthermore, the presumed normal daily dietary of stable iodine (I^{127}) is of the order of 100 μg , and that of stable phosphorus (P^{31}) is probably measurable in grams rather than in micrograms.

Since I^{131} is produced by slow neutron bombardment of tellurium¹³⁰ and decays to xenon,



it is available as "carrier-free" isotope. That is, no I^{127} should be present in the material used for tracer study.

P^{32} , on the other hand, is usually prepared by the bombardment of S^{32} with neutrons, by proton emission becomes P^{32} , and that in turn decays to S^{32} .



In the extraction of the phosphorus isotope from the sulfur, large loss of the P^{32} is inhibited by the addition of chemically small amounts of P^{31} . As a result, stable isotope is mixed with the radioactive one, so that the latter is not "carrier-free," and the administration of the P^{32} usually will include a chemically small but numerically large quantity of P^{31} atoms.

Other Factors in Section of Tracer Substance.—If a substance is made up containing an isotope whose radioactivity is to be used to detect, "tag," or "label" the normal behavior of that substance in the body, the isotope must be so incorporated into the

molecule that it will not be exchanged or broken off in the normal metabolic use of that compound.

For example, P^{32} in a phosphate group attached to a given substance by an ester linkage is a most unsatisfactory label, because such an ester linkage is unstable. As a result, the $P^{32}O_4$ radical rapidly parts company with its parent substance. On the other hand, I^{131} incorporated into a tyrosyl radical is a satisfactory tag, because the bond between the iodine and carbon is as strong as the carbon to carbon bonds of the tyrosyl radical. Loss of the iodine from such a radical would be equivalent, therefore, to disruption of the ring, hence degradation of the compound concerned.

Another example of a good label for a complete molecule is the linkage of radioactive hydrogen, tritium (T), to carbon in place of the hydrogen in the C-H bond, for this is strong, and the tritium will stay with that particular carbon atom under most circumstances. But linkage of tritium to oxygen, as in an OH radical, is an unsatisfactory tag, due to exchangeability of the H as already pointed out, a situation similar to the P^{32} of a PO_4 radical in ester linkage.

It is evident then that, if an isotope is to be used as a label for a complex molecule which is to be traced, the isotope selected must be incorporated into the molecule under study so as not to be separated easily from the labelled molecule.

Character of Tracer Investigations.—The applicability of radioactive isotopes to diagnostic problems will be determined by the type of problem to be studied.

Whether or not capillary and fixed tissue cell membranes of traumatized areas have become more permeable and, if so, to what substances; the mode of synthesis of the hemoglobin and the duration of red cell life; and the extent of the extracellular space in congestive heart failure and nephrosis can be elucidated by use of isotopes.

Determinations of circulation time and, in some degree, distribution to the lower limb as an aid to when and where to amputate in cases of circulatory disorders, studies of thyroid function, of plasma and red cell volume, and of detection of malignant lesions

comprise the majority of diagnostic tracer studies at present.

It must be remembered that if one desires to investigate an abnormal situation, some concept of the normal must be established. Hence, insofar as possible, comparison must be made with a sufficient number of presumably normal individuals whose biologic and environmental conditions have been and, during the period of study, remain equivalent to those of persons who appear to be abnormal.

Physiologic and disease processes may be grouped as follows, dependent upon the type of chemical substance which may be most useful as an analytic label:

1. Distribution and disposition of body fluids and electrolytes, or utilization of specific simple ions, such as Na^+ , K^+ , or Cl^-

2. Similarly with simple or specific radicals, such as PO_4^{3-} or I^-

3. Intermediate metabolism of biologically important organic compounds or specific cellular elements, in which readily traceable elements such as iodine and phosphorus are normal components or may be used as labels

4. Synthesis, distribution, and metabolism of specific compounds such as hemoglobin or serum albumin

5. Aberrations in physiologic synthesis or normal breakdown of biologically important compounds such as thyroxine

6. Location, extent, and physiologic activity of specific organs, tissues, or masses with selective avidity for a traceable element, such as the thyroid gland, halide-secreting cells, or neoplasms

Diagnostically at present, the isotopes in appreciable use are limited to Na^{24} , P^{32} , I^{131} , and Cr^{51} . Ca^{45} , Sr^{90} and Sr^{90} , Fe^{59} and Fe^{59} , and K^{42} have been used more in pure investigation. Considerable evidence is accumulating as to an osteosarcomagenic hazard from strontium²³

The life span of red blood cells in various blood dyscrasias is currently under investigation by means of labelling the cell with one of the isotopes of chromium (Cr^{51}) and then determining the rate of removal from the circulation.

The extent of a traumatized area could be determined by use of Na^{24} . But that is a short-lived isotope (half life 14.8 hours); hence, a

careful clinical assessment is certainly simpler and presumably sufficiently precise.

The extent of increase of the extracellular space in congestive failure and nephrosis can be obtained fully as precisely by use of thiocyanate as with Na^{24} ; and, what is more, its decrease can then be followed quite satisfactorily by the simple expedient of daily weighing, provided, of course, that the scales used register 10 pounds as 10 pounds, day after day.

There are problems to which isotopes can give exact answers; but in many of these, answers adequately good for clinical practice can be obtained by far simpler means. This does not disparage at all those investigations which are in progress to determine more precisely the actual physiologic pattern—about so much of which so little is known.

Radioactive Sodium (Na^{24}).—The short half-life of this isotope precludes its clinical use, to any major extent, save when the investigation can be done close to the source of production.

Shortly after artificial radioactive isotopes were first produced (1934), some quite spectacular demonstrations of their presence were in vogue. One of these was the detection, with a Geiger-Müller counter and annunciator, of gamma rays in the finger tips of a person who had just imbibed a cocktail of Na^{24}Cl .

Smith and Quimby (1947) put the prestidigitator's technique to respectable diagnostic use by comparing the rate of isotope count increase (following intravenous injection of a known amount of Na^{24} into the antecubital vein) over the affected limbs of patients with peripheral vascular disease to that of normal individuals. In their hands, the build-up over the sole of the foot of the normal person reached a plateau in about half an hour, whereas in those affected with vascular impairment the plateau was attained after a considerably longer period. The rate of build-up could be interpreted also in terms of adequacy of the circulation in the involved extremity. Although elaborate technical procedures were necessary, the results justified the effort, for by this means certain patients with thromboangiitis obliterans, or tensesclerosis with diabetes, or chronic ulceration of the leg, which by ordinary clinical judgment would have been subjected to a high am-

The radioactive iodine diffuses uniformly through this so-called "iodide pool" of plasma and extracellular fluids, mixing with whatever ions of stable I^{127} happen to be present at the time. As a result the body's whole population of iodide becomes uniformly labelled. That is, if there were 1,000 ions of I^{127} and 10 ions of I^{131} (once mixing is complete), for every ion of I^{131} in any locus there will be 100 ions of I^{127} . This dilution or uniform diffusion is important, because the rates at which both the thyroid and the kidney will remove I^{131} from the circulation will depend upon the inherent avidity of these two organs for all iodide ions and the number of such ions available to them in any given volume of blood, hence in the extracellular fluid bathing their respective cells at any given time.

In congestive failure, the radioactive isotope, with its fellow stable iodide and chloride ions, may become sequestered in these extracellular fluid spaces for some time. This is of importance in the differentiation of cardiovascular disease complicated by abnormal thyroid function. The spleen, in its capacity as a blood reservoir, can sequester the isotope also, thereby upsetting expected physiologic time relationships.

When such sequestering aberration is present, a peripheral tissue area such as the thigh is relatively slow in reaching its normal peak concentration and considerably slower than normal in showing clearance of the isotope from the general "iodide pool." In fact, a persistently elevated peripheral tissue count may be used as a sensitive index of otherwise unrecognized occult edema.

Shortly after oral administration, when the plasma still has a relatively high radio-iodide content, the count observable over the submaxillary and sublingual glands erroneously can be thought due to aberrant thyroid tissue or malignant thyroid cells in cervical lymph nodes.

Whenever food is ingested after the isotope has left the stomach initially, a rise in count over the stomach indicates that this organ is using the iodide ion in the same way it uses a chloride ion in elaborating acid gastric juice. The stomach, as well as the salivary glands, may contain concentrations of iodide ion some 30 times that in the blood circulating through

them at a given time. The iodide ions secreted into either the saliva or the gastric juices will reach the small intestine again, where they rejoin or repeat the cycle. Furthermore, after the first 24 hours following ingestion of the radioactive iodide, the degree of rise in count over the stomach postprandially can be used also as an indication of the amount of isotope still present or newly entering the circulation.

The Metabolism of Iodine Within the Thyroid Gland.—Iodine is taken up by the thyroid gland as the iodide ion, in which form it finds its way from the extracellular fluid about the capillary, by which it was brought as a constituent of plasma, to the parenchyma of the gland, across the cell membrane of the thyroid follicle cells, and subsequently rapidly distributed throughout both cellular and colloid elements of the follicle.

As yet there is no completely satisfactory explanation of the specific mechanism by which iodine is concentrated in a thyroid follicle. All that can be said categorically is that this mechanism is highly efficient, for the gland can extract iodide ions from plasma even when the ratio of the concentration of iodine in the gland to that in the plasma is 1,000:1. This does not mean that the concentration of iodide ions is great within any gland follicle. In fact, it is probably small, and some ions may diffuse almost immediately to the circulation. Probably, the remainder are oxidized to a form capable of reacting with tyrosyl radicals and thus form first mono-, and, second, di-iodo-tyrosyl. The latter two iodine-containing radicals are constituent parts of the large protein molecule thyroglobulin.

The precise locus of this iodination, the necessary chemical conditions for which must exist within each follicle, is at present controversial. However, the possibilities seem to have been reduced to a hollow sphere whose perpendicular wall thickness, occupying the cell-colloid junction, is less than 1 μ .

Once the iodide is thus joined or incorporated into the tyrosyl radical, spatial relationships allow such radicals to condense, with elimination of the alanine fraction of one, and thereby to give rise to thyroglobulin side chains of thyroxine or tri-iodothyronine, still bound by peptide linkage to the parent molecule in the colloid. These linkages are severed

enzymatically along with those of uncoupled mono- and di-iodotyrosine from the parent protein. As free amino acids they are capable of diffusing from the colloid into the circulation. However, a dehalogenase within the thyroid cell itself attacks the tyrosine, but not the thyronine amino acids as they diffuse through, and removes the attached iodine which, in turn, is free either to diffuse out of the follicle as iodide ion or to be reinvolved in the iodination of similar radicals attached to other thyroglobulin molecules.

Newly formed iodinated tyrosyl radicals, newly formed iodinated thyroglobulin molecules, and newly proteolyzed thyronine derivatives in turn undergo uniform distribution in the iodide pool within the gland. It is assumed (there being no evidence against it) that any iodide entering the thyroid at any given moment is subjected immediately to random distribution in the intraglandular pool, and that subsequent newly entering ions will behave similarly. Presumably, the newness of entry in no way makes any one unit different from similar units which may have been present in a given pool for varying lengths of time. It is this fundamental assumption of the randomness of distribution which has allowed the clarification of these steps by application of the philosophy of precursor-product relationships (Zilversmit et al., 1943) within this gland. This assumption permits an explanation of the early appearance in the circulation of labelled hormone from a gland already practically bursting with previously stored hormone.

On ingestion of a tracer dose of I^{131} , these iodide-organic-iodine transformations within the gland take place between the initial uptake and the reappearance of I^{131} in the periphery. The individual steps are indistinguishable at present, for only the number and energy of the I^{131} atom disintegrations are measurable. This decay of the isotope is independent of any chemical process or other influence, save the necessity in each atom to sooner or later achieve physical balance by expulsion of its excess nuclear particle beyond the restraining short-distance-acting intra-atomic forces.

What can be determined is the minimum time required for the complete sequence of changes between entry of a tagged iodide ion and the release of similarly labelled thyroid

hormone into the circulation. Subsequent to this release, the amino acid, apparently loosely bound to an albumin fraction of the plasma proteins, is carried via the systemic circulation to the peripheral extracellular fluids whence it may become intracellular and effect its specific hormonal action.

The question of whether thyroxine or tri-iodothyronine, in actual fact, is the effective tissue hormone remains unanswered.* Certainly the mechanism of fixed-cell membrane penetration is unknown, to say nothing of the nature or control of its definite intracellular role. Elucidation of the latter should provide ultimately some clue to the riddle of the various thyroid states—perhaps the answer to the WHY of Graves' disease.

Therefore, in the still very limited knowledge of thyroid disorders, I^{131} tracer studies can at best provide, in any one case, definite answers to only certain specific questions, if the iodide ion has free access to the extracellular fluid between thyroid capillaries and thyroid follicles, namely:

1. What is the size, shape, and location of the thyroid gland?

2. Is the distribution general or of nodular hyperactivity, or of definite lack of iodine uptake as in malignant disease?

3. Are there other masses of iodine accumulating tissue present in the body? If so, where and how large are they? (Other than the thyroid, the polysaccharide capsule of the tubercle bacillus is the only iodine-concentrating material currently known. Intrathoracic cold abscesses have caused confusion.)

4. What proportion of iodide is taken up by the gland and in what interval of time? Does this fall within the range previously determined as normal for the given environment?

5. If not, how great is the "avidity" of this gland for iodide ion?

The thyroid gland in a normal "steady state" has a certain constant or normal requirement of iodine to maintain its hormone fabrication and reserve. This will be approximately 100 mg. of iodine per day for an adult male.

It is probable that with deficiency of iodide supply the individual's fabrication of hormone will be altered.

*Thibault and Pitt Rivers (Lancet, 1955) have shown that in vitro the acetic acid analogues of thyroxine and tri-iodothyronine immediately cause a rise in the oxygen consumption of rat kidney slices and suggest that these are the tissue active forms of the thyroid hormones.

Further, if the thyroid-stimulating factor of the pituitary whips the thyroid into greater activity or some other alteration of the metabolism, so far as undetermined, causes increased demand surpassing ordinary requirement as in thyrotoxicosis, the relative greed for iodide may be spoken of as "avidity."

6 How rapidly is the iodide ion being transformed into thyroid hormone? (This can be observed by continuing iodide entry.)

7 At what rate is hormone being released? This may require observation over a period of several days.

Technique of the I^{131} Tracer Study of Thyroid Function.—The method or technique adopted for an I^{131} tracer study should be such as to answer best all or the majority of the questions posed above with the smallest practicable amount of radioactive isotope, the best economy of time and effort on the part of the patient and the examiner, and the least amount of physiologic disturbance of the patient.

Some observers have devised various short cuts or have selected certain periods for observation from which they extrapolate "uptake" and "excretion" curves and by which they achieve an interpretive assessment. Such techniques are not as trustworthy as desired in cases other than those which are relatively obvious clinically.

In our opinion, a more detailed series of observations should be employed regularly in order adequately to assess the situation and particularly to analyze the more obscure considerations of masked thyroid dysfunction, such as those in association with cardiovascular disease and diabetes.

The following technique is preferred

1 A dose of 50 μ c of I^{131} as NaI in 3-8 oz of water is given by mouth after an overnight fast and before breakfast. A normal breakfast should follow in 1/2-1 hour. Pleasant surroundings are provided so that the patient is as much at ease as practicable, and the findings will be physiologic.

2 Beginning 2 hours later, mass counts are taken of the thigh, liver region, chest, and neck, with an opposing pair of Geiger tubes mounted 50 cm apart on an adjustable carriage.

3 Detailed local scanning and counts of selected points, with an end window Geiger-Müller tube, sensitive to beta particles as well as to gamma rays, is done routinely over the thigh, liver, epigastrium, splenic area, upper end of the sternum, manubrium, sternal notch, and at least 5 points over the thyroid at similar periods preferably at 2, 5, 8, (12), 21, and 36 hours after ingestion of the isotope.

4 Each voided urine specimen is assayed with a liquid counter for I^{131} excretion up to 32 hours.

5 Curves (corrected for physical decay) of the thyroid isotope uptake and of the cumulative periodic urinary discard, expressed as per cent of administered dose, are constructed from these data.

6 Iso-topographic drawings of the detailed neck-scannings, preferably to scale on a tracing of the patient, are readily made and very useful.

7 In the assessment of each patient, these collected data are used to check stomach emptying, patency of vascular beds, sequestration of the isotope in spleen and extracellular fluids, secondary concentration of hormonal isotope in the liver for detoxification of excess thyroxine and clearance from the blood.

Counts over the upper end of the body of the sternum indicate the concentration of the isotope within the great vessels of the middle mediastinum, while those over the lower half of the manubrium will assay, in large measure, the amount in the blood flowing through the aortic arch.

Gland counts should include at least those perpendicular to the center and long axis of the isthmus and of each lobe of the thyroid, and at angles of 45 and 90 degrees medially and laterally. Assessment of the activity, size, and position of the isthmus and each lobe must be accomplished, or the observer is not doing his job properly.

Measuring instruments must be initially and repeatedly calibrated against known amounts of isotope, preferably with an anatomically correct plastic model.

In each geographic area and for any peculiarly segregated environmental group, the normal average uptake and "range of normal" must be determined by tracers on presumably normal volunteers (interns, medical students, and patients ill with other than thyroid disorders) in whom there is no recognizable or even suspected thyroid element, all being endogenous to the area and environmental conditions.

In assessment of the state of the thyroid in any one patient, a careful history, the BMR, and blood cholesterol levels as well as other laboratory tests, such as ECG's and blood sugar studies, are indicated. Total and protein bound blood iodine determinations are valuable in certain borderline cases. Normally, free blood iodide equals about 0.3 μ g %; an amount too small to measure accurately in ordinary samples of plasma.

The particular times chosen for counting are recommended for the following reasons:

Absorption, thorough mixing, and uniform distribution throughout the iodide pool is not completed in less than 2 hours after ingestion of the NaI^{131} test dose. If, at that interval, a high count over the epigastrium indicates possible pylorospasm, simple reassurance will ordinarily induce relaxation, detectable as a sudden spurt in the counting rate over the duodenum.

Zero time, the time of administration, can be

advanced then to this observation moment. Normally, the 2-hour observations will indicate the degree of avidity of the gland for iodide, the proportionate dilution is rarely known. At that period also, the plasma has a relatively high radioiodine content. The observer may find an increased count over the submaxillary and sublingual glands. As comparative data, later observations will correct possible error in suspicion that such may be due to aberrant thyroid tissue or malignant thyroid cells in the cervical nodes.

The 5-hour count period falls just after lunch—a convenient time to check on the efficiency of the body's use of iodide as a halide. The epigastric or stomach count should be elevated, salivary gland count down, peripheral tissue counts falling. Thyroid counts should provide evidence as to whether or not the thyroid is capable of holding onto the iodine it has collected or is concentrating.

In cases of acute thyroiditis, either cell-membrane permeability or the ability to collect and use iodide, or both are affected. In this condition, at 2 hours after ingestion, when the plasma level of iodide ion is high, the thyroid level is comparably high. Thereafter, as the plasma iodide ion count falls, so does that of the thyroid. Splenic concentration of isotope should be assessed at this time and later. If blood without or with the isotope is stored in the spleen, its release subsequently would either suddenly dilute or increase the concentration of isotope in the circulating plasma and hence affect the count in all the peripheral fixed-tissue areas.

The 8-hour observation, before supper on the first day, affords comparison or confirmation of the trends suggested at the two earlier periods. The third set of periodic data in this first day permits curve constructions which give a truer sense of the tissue-isotope behavior. In really toxic hyperthyroid patients, the peak uptake is reached by the gland early, often by 6 hours. An 8-hour count will show whether the uptake is still rising, is constant, or has started to fall (a possible indication of rate of fabrication of iodide ion into circulating thyroid hormone).

By 24 hours, for normal patients, there has been a sufficient time lapse for the extrathyroidal tissues to have been cleared and for the

thyroid to have reached its peak concentration. However, this is not always true, for even at 32 hours or later, the thyroid concentration ("count") may be continuing to rise and the peripheral count to fall in some presumably normal individuals. This is common especially in patients with sluggish circulation. (Twenty-four hours is a check point chosen by some laboratories using a short-form tracer study. However, it must be remembered that a single measurement at a single time provides only meager information on how a complex organism may be handling a single element, and definitely indicates only the status at that particular moment.)

In the majority of patients, by 32 hours the periphery is free of isotope, or nearly so, and the gland is well filled with it. This is the time at which a definite concept can be formed of the size, shape, position, and intimate reaction of the gland to this element.

For some patients, especially those with cardiac disease, the tracer observations have to be continued into a third or even later day before the periphery will have cleared sufficiently.

Observations can be extended until sufficient data are available to assess the rate of hormone release from the gland, although this is complicated by re-use of isotope released by hormone degradation in peripheral cells. One is likely to forget that only the raw material for hormone synthesis, the iodide ion, has been provided. In no way can impatience for an answer and possible resultant diagnosis speed up the orderly time sequences of nature's processes. Furthermore, the time clocks in various human beings are not synchronized, and in each individual the rate may change from time to time, due to varying conditions. With increasing experience, determination of certain specific check points may be practicable. Albeit, in problem cases, for whose diagnosis a careful tracer study is of the greatest usefulness, it has been repeatedly evident that the most detailed examination is the most economic in the achievement of a positive diagnosis.

Tracer Use of I^{131} Attached to Organic Molecules.—

Radioiodinated serum albumin (RISA)
Since radioiodine can be chemically added to

the tyrosyl radicals of the long chain organic molecules, such as serum albumin, without altering their biologic activity or immunologic properties, such preparations can be used to label and hence trace the physiologic behavior of similar nonradioactive molecules in the body.

Plasma volume can be determined with radioactive iodinated human serum albumin (in known quantity) injected into the blood stream, and its dilution by the solvent (blood plasma) volume assayed (measured in terms of radioactivity per unit volume).

Thorough intravascular mixture of added serum albumin is completed in 10-15 minutes. By that time none of the newly added albumin will have left the vascular tree. However, by 1 hour 10%, by 5 hours 20%, and by 24 hours 45% of the added RISA will have left the circulation to become equilibrated with the total body albumin occupying the "albumin pool" which presumably corresponds to the extracellular body water—some 15% of the total body weight. In 1 hour, some of the intravenously injected RISA has been reported to have appeared in lymph obtained from the thoracic duct.

Calculation of plasma volume is of particular value in anemia, in cases for replacement of blood loss—operative or essentially hemorrhagic—and preoperatively in the chronically ill patient with a probably contracted blood volume.

RISA has certain advantages over the dyes used in the older and classic dilution methods previously employed, in that:

- 1 The material is a normal physiologic component of plasma.
- 2 The small amount required will not upset the normal dynamics of the albumin already present.
- 3 That amount (5 ml. of a sizable dilution of a commercially prepared sterile RISA) is sufficiently radioactive (10-15 μ c of 131 I) that its diluted fraction can be detected and measured in a small sample of blood drawn from the antecubital vein 10-15 minutes after injection of the original tracer quantity into the opposite antecubital vein.

After noting and recording the hematocrit reading, the sample is centrifuged, and a 1 ml. portion of the resultant plasma measured as to its radioactivity in a well type counter. Comparison with the radioactivity of a standard dilution of the original injection material (1 ml. of a 1,000 ml dilution of an equivalent

volume of the injection solution) permits ready calculation of that particular patient's plasma and total blood volume.

Correction factors will include the following:

- 1 Plasma trapped between red blood cells—considered equivalent to 4% of the height of the column of packed red blood cells in the hematocrit reading.
- 2 The venous hematocrit correction, known to be 10% higher than the total body hematocrit.

Together these give a combined correction factor of 0.87.

Furthermore, such plasma volume determinations, especially if coupled with concurrent calculation of red cell volume, are valuable in selection of the proper replacement or substitution medium—whole blood, plasma, or packed red cells.

The progress of substitution therapy, undertaken as a result of the first determination, may be closely checked by withdrawal of a similar blood sample, determination of its contained radioactivity, and then injection of a second test volume of R.I.S.A. with similar assay of its dilution corrected by the known residual activity in the second pre-injection sample.

Localization of malignant tumors in brain or liver has become possible with radioiodinated serum albumin. If the blood-brain barrier deters entry of small molecules to the cerebrospinal fluid—the true extracellular fluid of nervous tissue—and the brain tumor is nourished from the extracellular fluid, which diffuses out of the involved blood vessels directly supplying the mass, there may be a relatively specific detention of the radioactive albumin in the tumor, the isotopic nuclear decay of which may then be detected as in other external tracer studies (Radioarsenic [74 As] may be used with a scintillation counter but is not commonly employed).

The situation regarding metastases to the liver is less easily explained, there being no such barrier on which to call. Philosophically, there may be increased, slowly turning over extracellular fluid—in effect, an edematous response to the abnormal growth—or the serum albumin may be the precursor for the protein portion of the nucleoprotein which is being rapidly synthesized in such tumor masses.

Whatever the reason, the fact remains that given a sufficiently large mass, sufficiently different from its surrounding normal tissue, such a mass can be detected in either location by the difference in radioactivity count rate of the normal compared to the abnormal tissue. The time interval chosen for such determination should be sufficiently long after intravenous injection of RISA to allow thorough equilibration with the extracellular fluids but before the iodinated albumin has begun to be destroyed by normal metabolic processes. This would suggest not less than 2 hours and not more than 24 hours. Sensitive instruments and discriminatory survey of the area are required.

Chromium⁵¹ in Determination of Red Cell Volume and Survival.—Red blood cell volume determinations with Cr⁵¹ are done in a manner somewhat similar to that employed for plasma volume determinations with radioiodinated serum albumin, except that the patient's own red cells must be taken, processed, and reinjected.

If these red blood cells are incubated with sodium chromate ($\text{Na}_2\text{Cr}^{51}\text{O}_4$) which has been made radioactive in an atomic pile, 97% of the Cr⁵¹ taken up will bind strongly to the contained hemoglobin, while the remaining 3% is attached to the stroma. (Of 50 μc of Cr⁵¹ in the incubation mixture, 50-90% will be incorporated.) The incubated, now radioactive, red blood cells must be washed free of the residual unincorporated Cr⁵¹, avoiding, insofar as possible, lysis of the cells.

When survival studies are to be done, injury to the red blood cells must be avoided. For that purpose and to reduce the hazard of washing the red blood cells, ascorbic acid is added to the incubation mixture, in order to convert the trivalent reactive Cr⁵¹ into the unreactive hexavalent ion, which is rapidly removed upon injection into the circulation. This method has the possible advantage of using the patient's own red cells as the labelling agent but requires rather meticulous processing of the cells under aseptic conditions and their reinjection, followed by the third venipuncture for the dilution sample in the volumetric study.

In the survival studies, samples are drawn every second day after injection of the tagged cells, until a satisfactory curve can be constructed of the rate at which these cells are

leaving the circulation, presumably either by hemolysis or splenic destruction. The curves constructed are not corrected for either physical decay of the isotope or for the slow elution of the chromium from the cells themselves, which occurs at the rate of 1% day. This latter factor is also affected by the age of the cell and seems to increase with age. Rather crude or uncorrected curves result. To obtain absolute survival rates, incorporation of one of the radioactive isotopes of iron into newly synthesized hemoglobin would be preferred, but the isotopes of iron (Fe^{55} and Fe^{59}) are difficult to use due to their half-lives (respectively, of 4 years and of 47 days) and also due to the character of their disintegrations, namely, by K capture (Fe^{55}) and by emission of soft, hard-to-detect beta particles, and hard and also poorly detectable gamma rays (Fe^{59}).

Nonetheless, comparative studies of survival rates in the different kinds of anemia may in time shed much light on such questions as whether a particular anemia is due to failure of formation, increased fragility, or excessive destruction of the particular red blood cells, or to some combination of these factors.

Radioactive Phosphorus (P^{32}).— P^{32} can be produced in the cyclotron by deuteron bombardment of P^{31} plus proton emission, or in the atomic pile by slow neutron bombardment of S^{32} plus proton emission. As mentioned before, it is not obtainable "carrier-free." The energy spectrum of its emergent beta particles is fairly broad, with an average of 0.695 mev (million electron volts). At most, these beta particles can travel 0.8 cm, and on the average only 0.27 cm through tissue. In transit, they may produce *bremsstrahlung*, a type of secondary electromagnetic radiation (like x-rays). The probability of their doing so is 1,000:1. The *bremsstrahlung* have an energy spectrum similar to that of the emitted beta particles, and like the beta particles lose momentum as they pass through matter. The geometry of the end-window Geiger-Müller tube used for their detection, i.e., the portion of total detectable rays actually "seen" by a given instrument, is at best only 50% and usually considerably less. Such a counter is responsive to about 1% of the *bremsstrahlung* that actually passes through it. When all these factors are taken into account, detection of P^{32} concentrations any dis-

tance below the surface of the skin in tracer studies *in vivo* is physically difficult.

In man, phosphorus may be considered as a universal cell constituent, for it is involved in intermediary metabolism reactions. Since there is so much of it normally present, and since the body cannot distinguish between P^{31} and P^{32} , the body tissues and individual cells will use either one indiscriminately. Administration of a chemically small amount of P^{31} along with P^{32} affects the various phosphorus pools less than a similar amount of stable isotope would affect those of P^{31} . Were it as surely and easily detectable as P^{31} , a larger dose would be required to get numerically comparable counts, because P^{32} has a longer half-life (14.3 days). It is, however, easily producible, transportable, and a physiologic element.

Much study has been devoted to the chemistry of phosphorus and its compounds, especially the phosphate, the form it so commonly takes within the human body. This radical (PO_4), stable enough within itself, flits from compound to compound, thereby transferring the necessary energy for anabolic and catabolic processes. Phosphate is involved in carbohydrate, fat, and protein metabolism, in the creation of new nucleoprotein required for cell division, in the chemistry underlying muscular contraction, and in the solidification of the skeleton by deposition of calcium phosphate, as well as many other physiologic processes. Attention has already been called to the problem of exchange in bone crystals. This extends to other phosphate ions as in the circulating fluids. In intermediary metabolism it is joined to one complex after another by ester linkage, and in the elaboration of new nucleoprotein all of the phosphate of any given nucleus is wholly replaced each time a cell divides. Even its absorption from the gastrointestinal tract, as well as its transport across tissue cell membranes, is dependent upon an active process that involves linkage of it to something else—probably glucose or one of its derivatives.

Theoretically, phosphorus should be concerned wherever cells are dividing, and undoubtedly it is, but there are so many places in the body where cells are normally dividing all the time. To recognize special regions, such as malignant tumors that are rapidly growing,

requires that the obscuring background of all the other processes be allowed to run their course and exchange back to the circulation the $P^{32}O_4$ for new $P^{31}O_4$, taking the place of the now excreted $P^{32}O_4$. Since such tumor cells are dividing at a greater than normal rate, they should lose the concentrated phosphorus more rapidly than do the normal sites, because all, not just part, of the phosphorus is renewed during division.

If P^{32} is to be used as a "tracer" for detection of malignant lesions, it is necessary that the suspected lesions be close to the surface, that sufficiently large amounts of isotope be given to overcome in some degree the technical difficulties of measurement, and that the optimum time at which definitive information will be obtainable be determined in every case.

THERAPEUTIC CONSIDERATIONS

Biologic alteration of human and other animal tissues by exposure to x-rays and the radiations from naturally radioactive radium was recognized shortly after the discovery of each. One of the earliest reports appeared in the Canadian literature within less than a year following Roentgen's first paper in December, 1895.

The isolation of radium by the Curies shortly thereafter was accompanied by the realization that it gave off somewhat similar radiations (called for some time, "Becquerel rays"), which induced much the same biologic changes on accidental or purposeful exposure.

Clinical observations and some research into relief of pain from local and particularly from confined inflammation, the modification, if not complete disappearance, of certain skin disorders, and the destruction or control of some malignant neoplasms by therapeutic use of these newly discovered physical phenomena were frequently reported in the medical journals at the turn of the century.

Considerable investigation of local effects and also whole body irradiation was pursued by Elihu Thomson, Codman, Panthelemy, Kienbock, and others before the end of the first decade (1895-1905). Many of these early observations formed the basis for fundamental concepts of the biologic effects of irradiation. Although some debate continues as to the selection of cases, in the curable or control

the use of the experienced radiation therapist, who, associated with an able radiation physicist, will continue to critically select suitable cases for such treatment and equally critically assess the results of his ministrations.

Europium^{152, 154} and cesium¹³⁷ may prove to be useful radioactive isotopes for similar use. At this time, cobalt⁶⁰ appears superior when all factors are considered.

Interstitial Irradiation

Tantalum¹⁸² has to have its beta radiation screened with 0.1 mm. of platinum. In wire form it is quite effective in treatment of carcinoma of the bladder and can be used in place of radium, interstitially, in the tongue.

Gold¹⁹⁸ grains, sheathed in stable gold¹⁹⁷ is supplanting the formerly used radon in gold tubing as "seeds" for implantation of small neoplasms. The short half-life, however, requires a short-time-loss relationship to an atomic pile source.

The biologic effect of the gamma radiation from these is the same as that from radium.

Interstitial infiltration of a colloidal suspension of gold¹⁹⁸ into prostatic tissue has been investigated by Kerr, Flocks, Elkins, and Culp in a dose ratio of 2 millicuries/Gm of tissue. Destruction of an appreciable quantity of tumor cells without an undue amount of damage to the tumor bed has been observed. This is still properly in the realm of clinical research, as are some studies by others on parametrial injection.

Intracavity Irradiation

Radioactive isotopes may be used in solid form in the cervix and uterus or in the maxillary antrum for the treatment of carcinoma, as has been done with radium. Here also, cobalt⁶⁰, properly fabricated as discussed regarding interstitial applicators, seems the most satisfactory of the isotopes. It has no advantage over radium, if the latter is available.

Another possible use for a cobalt⁶⁰ source would be as suspended mechanically in the center (or perhaps eccentrically) of a fluid-filled balloon in the urinary bladder for treatment of an extensive vesical carcinoma. Some technical problems of insertion, maintenance

of position, prevention of leakage, and comfort of the patient will arise. Cones and Gregory have found that the tumor dose required is about the same as would be expected with other agents, namely, some 6,500-7,000 roentgens equivalent physical.

Solutions or colloidal suspensions may be used for the treatment of diffuse primary or metastatic neoplasm, within limits:

1. In a *hollow viscus*, such as the urinary bladder, instead of using the balloon as a means of suspending a gamma radiation source discussed above, the balloon could be filled with a solution of a radioactive isotope salt containing sodium²⁴ (half-life 14.8 hours; beta 1.4 mev; gamma 2.76, 1.38 mev) in sodium chloride, or bromine⁸² (half-life 35 hours; beta 0.46 mev; gamma 1.35, 0.79, 0.53 mev) in calcium bromide.

Technical problems of rapid decay and possible leakage, with contamination of the patient and his environment, must be considered carefully.

2. In the *serous cavities* (pleura and peritoneum), disseminate implants of metastatic carcinoma (from the breast, ovary, etc.) and the pouring out of serosanguineous effusion, which they induce, can be modified, if not markedly controlled, by the introduction of a colloidal suspension of radioactive gold (Au¹⁹⁸). Our experience has not been as favorable as that reported by some investigators. Some of our relative disappointment has been offset by the appreciation that most of our patients have been rather desperate terminal cases. We have, however, combined the intracavity colloidal gold¹⁹⁸ method with external radiation therapy for postoperative patients with carcinoma of the ovary and for others with bronchogenic carcinoma, in which contamination of the space with neoplastic cells during the operation was probable. We are not prepared to comment on the results at this early stage. Theoretically, the method should be useful. The colloidal particles in these suspensions must be too large to pass into the lymphatic drainage channels.

Adjustment of colloid particle size or other means, such as insufflation techniques, or injection of radioactive material via cardiac and pulmonary catheterization, may evolve from

certain research which is in process in various centers, in order to effect local irradiation of selected regional lymph nodes, primary malignant tumors of the lung, or other relatively inaccessible malignancies not amenable to surgical ablation.

Parenteral Administration for Systemic Effect

"Whole-body irradiation" with high voltage x-rays has been used for a long time with considerable success in the management of the chronic leukemias and with less success in polycythemia vera. Similar methods can be used sometimes in widespread multiple myeloma or in diffuse metastatic carcinomatosis (as from the breast) as a palliative measure.

A much more logical and useful attack, especially upon the neoplastic tissue, would be to feed the malignant cells or, as in polycythemia vera, the erythropoietic tissues, some chemical element which they require for life but which would be radioactive and hence irradiate each cell or the tissues as a whole.

The universal requirement for phosphorus on the part of actively reproducing cells, as in the hemopoietic system, would seem to make its radioactive isotope (P^{32}) an ideal element for this purpose.

This has been proved useful, especially in polycythemia vera and in multiple myelomatosis. However, as has been noted above, the rapid turnover of phosphorus in cell metabolism and the side effects of general irradiation of the whole organism present problems which are similar to those associated with the use of cytotoxic or cytodepressant drugs (urethane, nitrogen mustard, etc.) and general roentgen irradiation.

Such treatment should be reserved for the use of the experienced radiation therapist working closely with the hematologist. The dosage must be adjusted to the individual patient. No standard rules have yet been evolved as to either the minimum safe dose or the maximum tolerable dose.

At present, in the authors' opinion and experience, the maximum initial dose of P^{32} should not exceed six millicuries. This should be given in divided doses in multiple myeloma. In polycythemia vera, a second dose, of any

magnitude, should not be considered short of 3 months.

As one of us has previously pointed out, "further development of possible systemic therapeutic agents incorporating radioisotopes will require greater knowledge of physiologic chemistry and cell metabolism than we presently have."

Parenteral Administration for Specific Organic or Cellular Effect

"Specific internal irradiation," or the use of selected radioactive isotopes for potential specific effect upon an organ or cell type is best exemplified in the treatment of hyperthyroidism or of cancer of the thyroid with I^{131} . Specific internal irradiation is accomplished in polycythemia vera or multiple myeloma with P^{32} , as applied to widespread tissue which perhaps could be considered as one organ. However, the special and almost exclusive use of iodine by the thyroid gland affords unusual opportunity to affect the thyroid cells specifically with the local irradiation of the decaying radioactive iodine.

Criteria as to specificity of physiologic usefulness and nontoxicity of the selected clinical element have been stated earlier in this chapter.

The physiologic use of iodine by the thyroid as an iodide ion has been discussed in some detail above, in order that the reader may understand the physiologic cycle of iodine use by the thyroid and appreciate that it can effect local irradiation while in the extracellular fluids of the gland, especially in the thyroid cell and its colloid.

The indications for treatment with I^{131} are as follows.

- 1 Hyperthyroidism in
 - a. Patients over 40-45 years of age
 - b. Younger patients in whom antithyroid drugs have failed to effect control or have induced deleterious side effects, or surgical methods have been refused
 - c. Patients who have associated cardiovascular disease which does not respond to other means of treatment
 - d. Patients with recurrence after surgical intervention
- 2 Thyroiditis, acute
- 3 Carcinoma of the thyroid, if the malignant cells or their immediate tumor bed normal cells will take up adequate amounts of the radioactive isotope

The contraindications for I^{131} treatment of hyperthyroidism agreed upon at the present time in this hospital are as follows:

1. Acute hyperthyroidism in younger patients
2. Hyperthyroidism during pregnancy
3. Nodular goiter

Opinions vary as to the optimum therapeutic dose in nonmalignant thyroid disease. The individual patient's utilization of the iodide 131 as studied with tracer amounts must be known and considered. At present, we consider a standard initial dose preferable, with supplemental administration in 3-4 months, as required.

Careful assay initially and a thorough reassessment are essential before any additional treatment.

It must be emphasized that in the hands of the experienced radiation therapist, equally effective treatment (as to clinical results) can be accomplished with properly administered conventional x-radiation. Pregnancy and youth are not contraindications for x-ray therapy of the hyperactive thyroid.

In malignant disease of the thyroid, the frequently relative disinterest of cancerous thyroid tissue in the use of iodine has been an obstacle, preventing material initial uptake of the radioactive isotope and any appreciable retention by the thyroid gland and its cells. Little advance in surmounting this hurdle has been made since Hamilton's first observation and Seidlin's initial report on treatment.

About 10% of the malignant thyroid tumors will use appreciable iodine.

Large doses of I^{131} are required for any effective treatment of cancer of the thyroid. In the majority of such patients, the discard of the isotope in the urine will be of a high order and will require careful observance of isotope isolation and noncontamination regulations.

In many of such malignant cases, external roentgentherapy can be used as effectively as and probably more effectively than specific internal irradiation with I^{131} .

Until more is known of specific cell metabolism and its biochemistry, especially with reference to any particular factor vital to the life of the malignant cell, employment of radioactive isotopes for specific internal irradiation must be confined to the select few discussed above.

The use of direct neutron irradiation to affect previously injected boron in treatment of brain tumors is being investigated at the Brookhaven center. This may in time be found to be useful but is still in the realm of research.

It is to be hoped that as knowledge of the radioisotopes grows, combined with increased interest in basic and especially clinical physiology, the use of these tools will advance our understanding of the normal and the abnormal state, and in turn result in more intelligent diagnosis and control of disease.

Chapter 8

Anesthesia

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and Philip R Bromage, MB

INTRODUCTION

As in the case of all other branches of medicine and surgery, the practice of anesthesia must be based upon a thorough appreciation of the great principles laid down in the study of the basic sciences. Added to this, attention must be paid to the psychologic approach to and the handling of patients.

For successful anesthesia three things are essential: the patient must feel no pain, conditions must be satisfactory for the surgeon, and no harm must befall the patient. In other words, there must be analgesia and suppression of reflex responses to surgical trauma, and the *milieu interne* must be maintained constant in the face of modifications of normal anatomy and physiology arising out of the nature of the operation or the anesthetic. For example, during thoracic surgery problems of the open pneumothorax must be dealt with by rhythmic artificial inflation of the lungs in order to correct mediastinal shift and paradoxical respiration. During operations on the nose and throat the airway must be made watertight to prevent the patient drowning in his own blood. When complete muscular paralysis is induced by curare-like substances in order to provide a quiet surgical field, the accompanying apnea must be corrected by adequate artificial respiration, and so on.

During recent years there has been a growing realization that deep general anesthesia

is harmful. Enzyme systems throughout the body are depressed and the functions of various parenchymatous organs disturbed for long periods after deep anesthesia. Furthermore, cardiovascular depression occurs with the onset of a shocklike state.

For many years it was thought that anesthesia had to be carried to reasonable depths to avoid the occurrence of neurogenic shock; otherwise the continued bombardment of the central nervous system by fierce afferent stimuli would cause exhaustion of the central nervous system. This gave rise to Crile's theories of anoci-association—the blockade of afferent stimuli by local anesthesia, combined with light general anesthesia to prevent psychic disturbances. While techniques based on Crile's theories gave excellent results compared with cases carried out under deep general anesthesia, it was not for the reasons that Crile supposed. Since the introduction of the neuromuscular blocking agents, such as curare, concepts of neurogenic shock based on Crile's theories have had to be modified. It has become apparent that the most destructive procedures can be done without any afferent blockade and under extremely light general anesthesia (so light that the patient can respond to the spoken word) and yet, provided there is muscular paralysis and provided blood loss is replaced by blood, no shock appears. Almost anything can be done to the patient so long as he can make no effective response.

ANESTHESIA

The reasons for this are probably quite simple. The nonspecific pattern of response to trauma is an expiratory grunt or groan, as the sensitive organism tries to protect itself by constricting its thoracic and abdominal muscles as a shield against dangerous stimulation of thoracic compliance causes a diminution of thoracic compliance and a rise of venous pressure. The former increases the work of respiration, and the latter diminishes the perfusion pressure across tissue capillary beds, increasing any stagnant anoxia present and enhancing bleeding from cut surfaces. Thus, it is not the surgical trauma which

does the harm, but rather the patient's own instinctive, but, under the circumstances, inappropriate pattern of responses. If the patient can be protected from himself by muscular paralysis, if as St. Paul puts it, there is no kicking against the pricks, afferent bombardment appears to have little effect on the clinical condition of the patient.

Thus, with few exceptions, deep anesthesia is seldom necessary, provided suppression of reflexes is obtained by other methods, for the reflex arc can be broken in any part of its course by either sensory or efferent blockade

TABLE 8

I CORTEX — NUCLEI — MEDULLA			
Stimulant	— Picotoin—Metrazol—Amphetamine		
Depressant	— Barbiturates—Bromides—Chloral—Avertin—Paraldehyde		
Analgesic	— Morphine—Demerol—Methadon		
Anesthetic	— Ether—Cyclopropane—Nitrous Oxide (weak)		
Anticonvulsant	— Mesantoin		
Emetic	— Apomorphine		
Antiemetic	— Dramamine—Largactil (Chlorpromazine)		

II AUTONOMIC NERVOUS SYSTEM			
STIMULANT		Support Normal Mediator Substances	
Inhibit Mediator Destroying Enzymes			
Parasympathetic	Sympathetic	Parasympathetic	Sympathetic
(Anticholinesterase)	(Antiaminoxidase)		
Physostigmine	Epinephrine	Pilocarpine	Noradrenalin
Prostigmine	Acetylcholine		Adrenalin
Neostigmine	Nicotine		Neo-Synephrine
Tension	Carbachol		Vasoxyl
			Methedrine
DEPRESSANT		Anticholinergic	Antidrenergic
Ganglionic		Atropine	Ergotoxine
		Scopolamine	Dibamine
		Cocaine	Physoline
			Benzedoxane
			Regitine
Tetraethylammonium (Etamon)			
Hexamethonium and Pentamethonium			
Arfonad and Pendiomide			
Apresoline			
Curare in large doses			
III DEPRESS SPINAL CORD	IV DEPRESS NERVE TRUNKS	V DEPRESS MYONEURAL JUNCTION	VI DEPRESS SMOOTH MUSCLE
Mephnesin (Myanesin)	Procaine—Ponto	1 Competitive	Papaverine
Prendotel	caine	Curare	Nitrofur
Benztimidazole	Nupercaine	Metubine	Xanthines
	Mephcaine	2 Depolarization	Nicotinic acid
	Cyclaine	Acetylcholine excess	Histamine
	Xylocaine	Nicotine	Arfonad
		T.T.A. C.C.C.	
		(Decamethonium)	
		Succinylcholine	

Emancipation from the need to employ deep anesthesia carries many advantages. Depression of hepatic, renal, and cardiovascular function by high concentrations of anesthetic agents is avoided, and light anesthetic "sleep" can be carried on for many hours without detriment. Prompt arousal at the end of operation simplifies postoperative care, for the patient is in full possession of his protective swallowing and coughing reflexes, and he can control his own posture. Moreover, if postoperative coma is prolonged for any reason (e.g., electrolytic imbalance, intracranial lesions, etc.), the diagnosis is not confused by the added factor of prolonged recovery from deep anesthesia.

place. In addition to those actions shown in the scheme, some drugs affect the spinal reflexes, some the internuncial neurons, and some the reticular activating substance.

Secondary Action of Anesthetic Drugs

Respiratory System.—Drugs may depress or stimulate the nervous mechanism by which respiration is controlled either centrally or peripherally, in a manner which may interfere with ventilation to any degree from hyperventilation to apnea. In addition, the biochemic influence of the blood gases and pH by their action upon the respiratory centers and chemoreceptors may affect ventilation.

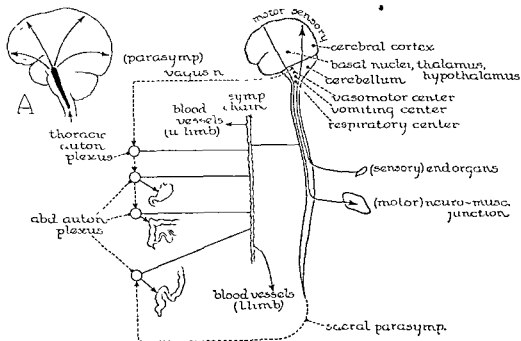


Fig. 48—Primary action of general anesthetic drugs. A, Depression of polysynaptic relays in reticular formation of mid- and hind brain blocks projection of afferent impulses to cortex. (Gradation in shading denotes degree of depression.)

BASIC CONSIDERATIONS

PHARMACOLOGIC OBSERVATIONS

Action of Drugs Upon the Nervous System

As all anesthetic drugs and those used in association with anesthesia act on the nervous system, it is appropriate to differentiate the effect of some graphically (Table 8) in an endeavor to show at what site this action takes

The bronchial muscle may be stimulated or depressed, resulting in bronchial constriction or bronchial dilatation.

The mucosal lining of the air passages may be irritated, resulting in the production of excessive secretions, unless an anticholinergic drug is given to depress the glandular activity.

Cardiovascular System.—Drugs used in anesthesia and anesthetic techniques may in-

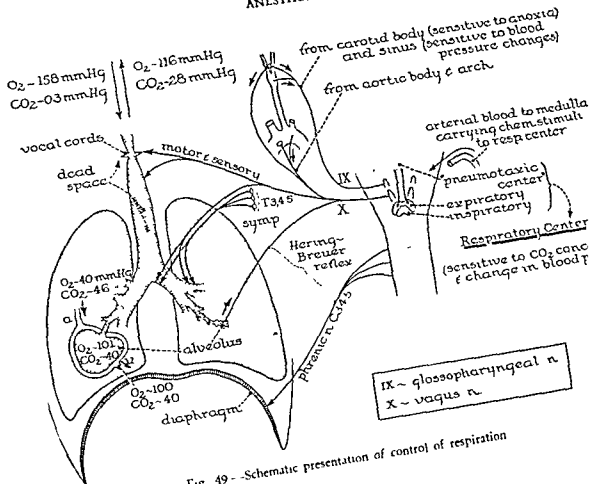


Fig 49 - Schematic presentation of control of respiration

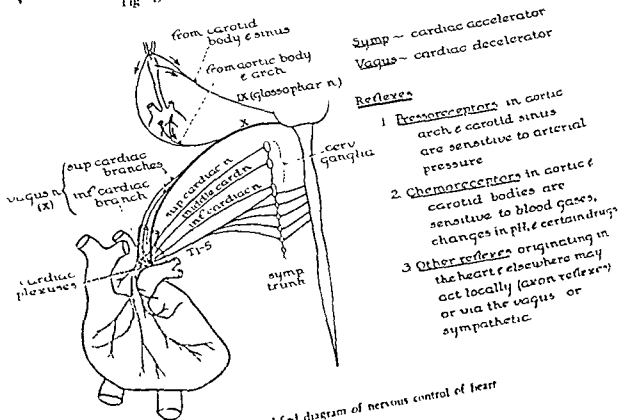


Fig 50 - Simplified diagram of nervous control of heart

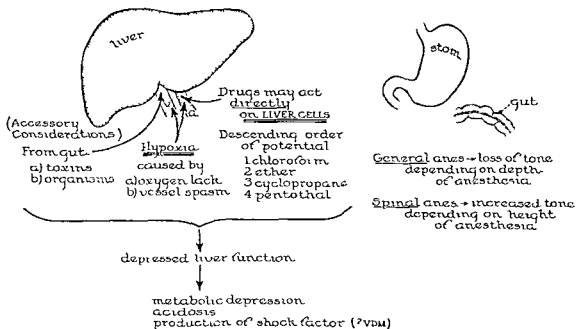


Fig 51—Effects of anesthesia on alimentary tract

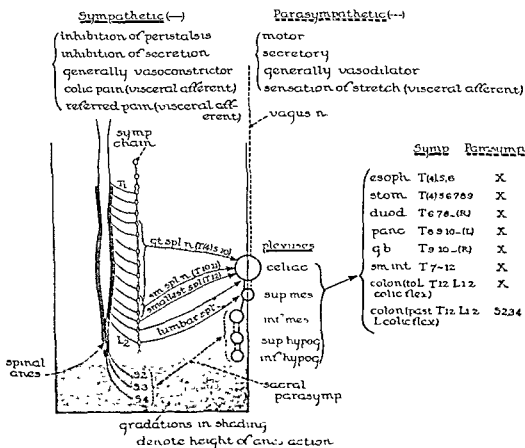


Fig 52—Innervation of the gastrointestinal tract

fluence the activity of the cardiovascular system. Such phenomena produced may be (1) brought about by a direct nervous mechanism, e.g., action of atropine on the vagal nerve endings in the heart muscle; (2) caused by oxygen lack or carbon dioxide excess, e.g., pressure changes, cardiac arrhythmias, or even cardiac standstill; (3) induced by the direct action of drugs on the heart muscle, e.g., sensitization by cyclopropane, (4) the result of the response of the coronary vessels, e.g., coronary constriction caused by Pitressin; (5) caused by responses to pressoreflexes or chemoreflexes, (6) produced by the action of drugs on the vasomotor center, e.g., peripheral vasodilatation and subsequent diminution of peripheral resistance; (7) due to action on the blood itself, e.g., hemoconcentration; (8) caused by temperature changes.

Alimentary System.—The commonest side effect in this system is the nausea and/or vomiting which may be caused by anesthesia. This may be produced by either central or local action.

The autonomic ganglia within the abdomen are extremely sensitive structures, the stimulation of which under inadequate anesthesia may result in unpleasant and even serious responses.

The numerous functions of the liver may be depressed by anesthetic drugs, by anoxia, or by hypotension. Among these functions is that of detoxifying or conjugating some of the agents used. Anoxia readily interferes with these functions, while some drugs may have a specific effect upon the liver. The adverse effect of drugs upon the liver is increased by oxygen lack (Figs 51 and 52).

Other Systems.—The effect of anesthetic drugs or techniques may be reflected in other systems. For instance, the secretion of urine is usually depressed by general anesthesia, the antidiuretic hormone being stimulated and renal blood flow diminished.

Spinal anesthesia is more commonly followed by retention of urine than is general anesthesia.

PHYSIOLOGIC OBSERVATIONS

Respiratory System. The change of conditions accompanying anesthesia and surgery may so alter the mechanics of respiration that oxygen uptake and carbon dioxide elimination may become inadequate to maintain the narrow

biochemical limits compatible with life. Therefore it is important to have a clear picture of the normal ventilatory requirements and the alterations of respiratory mechanics that may have to be corrected or allowed for during a surgical operation.

RESPIRATORY GAS EXCHANGE.—The normal adult utilizes about 250 ml. of oxygen each minute, and in the same period he excretes about 90 mEq of acid in the form of exhaled carbon dioxide (with a respiratory quotient of 1.0 this will be contained in 250 ml CO_2). Since expired air contains 4% carbon dioxide, elimination of this quantity of CO_2 will require a minute volume of at least: $250 \times 100/4 = 6,250$ ml. (i.e., 6.25 L.). Therefore, if the patient is breathing spontaneously, the minute volume should never be allowed to fall below this level, and the inflow of fresh anesthetic gases must be kept in excess of this in order to flush out exhaled CO_2 from the circuit. If a carbon-dioxide absorber of soda lime is in the circuit, the inflow of gases can be reduced to the basal requirements.

If respiration becomes depressed for any reason, so that the minute volume falls below about $6\frac{1}{4}$ L., ventilation must be augmented artificially by applying an intermittent inflation pressure to the rebreathing bag, the rhythm being timed to accord with the patient's own respiratory rate (this is called "assisted respiration"). Or, alternatively, the patient's own rhythm can be abolished by hyperventilation combined with respiratory depressant or paralyzant drugs, and an entirely different respiratory rate and depth can be imposed (this is called "controlled respiration"). Provided no noxious stimuli are entering the central nervous system, controlled respiration is surprisingly easy to impose under very light anesthesia, using hyperventilation alone. However, if painful stimuli are being received, the patient reacts by tightening his thoracic and abdominal muscles, and anesthesia must be deepened, or a paralyzant drug given, to overcome this reactive spasm of the expiratory muscles.

Once spontaneous respiration has been abolished, inflation of the lungs follows an abnormal pattern, since the alveolar tree is opened up by positive pressure from within instead of by the peripheral negative pressure and

ated with normal spontaneous respiration. This reversal of the normal inflationary pattern is accompanied by a considerable fall in pulmonary compliance. That is, a greater pressure has to be used to shift a given volume of tidal air, and an abnormally large minute volume has to be maintained in order to ensure adequate elimination of CO_2 . Thus, the anesthetist should err on the side of overventilation rather than underventilation when using controlled respiration, in order to avoid an insidious accumulation of carbon dioxide and an accompanying fall of pH of the blood to dangerously acidotic levels. For, under these circumstances, vagal tone is heightened and then cardiopressor reflexes may bring about cardiac arrest from relatively trivial stimuli, such as the removal of an endotracheal tube at the end of operation, which would normally produce no appreciable effect. Overventilation, on the other hand, seldom leads to trouble.

DEAD SPACE—Children require special care to ensure efficient respiratory exchange. In children the ratio between the volume of the air passages and the alveolar volume is large compared to that of adults, so that each breath contains a relatively large amount of "dead space air," that is, gas which does not take part in the respiratory exchange, since it occupies the air passages and does not reach the alveolar membrane. If rapid shallow breathing is allowed to develop in these patients, anoxia can quickly follow, since with shallow breathing the tidal volume approaches the dead space volume and all that happens is a shunting to and fro of functionless dead space air. For this reason, drugs such as trichloroethylene, which tend to cause rapid breathing, should be avoided in children.

It follows also that an apparatus which increases the dead space is harmful. Face masks should be designed to contain as small a volume as possible, and in long operations it is wise actually to reduce the normal dead space by inserting an endotracheal tube. In cases where severe respiratory embarrassment exists (e.g., copious bronchial secretions or partially paralyzed respiratory muscles), elective tracheostomy is an excellent way of reducing the dead space and improving respiratory efficiency.

RESISTANCE TO BREATHING.—Increased resistance to respiration not only increases the work necessary to breathe but also causes a rise of venous pressure which in turn increases surgical hemorrhage from cut tissues. Therefore, attention must be directed to keeping this resistance to a minimum at all times. Absence of respiratory resistance can contribute a great deal toward reducing blood loss during operation. Increased resistance to breathing can arise in the following sites:

Air Passages—Partial or complete obstruction to the airway has numerous causes, such as excessive secretions, laryngeal spasm, a relaxed tongue which has fallen backward, etc. Besides bearing in mind these common hazards, it is well to remember a few basic principles concerning the flow of gases in tubes, for their application provides a guide to good anesthetic technique.

Flow in tubes is proportional to the fourth power of the diameter; thus a small amount of laryngeal or bronchial spasm can have a profound effect on respiratory resistance and respiratory exchange; by halving the diameter of a bronchus the resistance will increase 16 times. Therefore every effort should be made to provide a relaxed bronchial tree. Ether, aminophylline, and topical anesthesia should be considered in cases of bronchospasm, while Pentothal should be used with caution, if at all, in such cases.

Apparatus—The foregoing remarks qualify the choice of endotracheal tubes which should therefore be as large as the larynx will comfortably admit. The lumen of such tubes should be as great as possible, concomitant with the rigidity of the wall.

In straight or curved tubes, flow is laminar and resistance is proportional to flow rate, but with turbulent flow, resistance is proportional to the square of the flow rate. Therefore, endotracheal connections should be curved and not angled, and any kinking of the breathing tubes should be rigorously avoided.

Most anesthetic machines have expiratory valves to permit the escape of excess anesthetic gases; these valves possess resistance. If perfect conditions are sought, these valves can be discarded in order to ensure complete lack of extrinsic resistance, and the patient may be

supplied with a high flow of anesthetic gases, the excess passing out into the room through an open-end tube

Lung and Chest Wall—The natural respiratory response to painful noxious stimulation is a forced expiratory grunt, as the thoracic and abdominal muscles tense into a shield over the underlying viscera. This same reflex is brought into action by stimulation of the tracheobronchial tree; for example, by the presence of an endotracheal tube. Too light anesthesia allows this protective reflex to take effect, and the patient then has to breathe against his own increased muscular resistance. Besides increasing the amount of work required for respiration, this also raises the venous pressure and increases surgical hemorrhage.

This adverse reflex can be abolished in exactly the same way as other reflexes, by attacking it centrally with deeper anesthesia or peripherally on the afferent or efferent side of the reflex arc with regional anesthesia or neuromuscular blockade.

The lung parenchyma, itself, may become abnormally stiff and resistant, as in pulmonary congestion from cardiac failure or from over-enthusiastic administration of blood and other intravenous fluids.

SIGNS OF RESPIRATORY INADEQUACY—Cyanosis is mentioned more fully elsewhere in this chapter. Except in polycythemia, it is a relatively late sign of anoxemia requiring urgent correction of the underlying cause.

Early anoxia causes a full bounding pulse which gives way later to a failing, thready beat. In infants, bradycardia may be the first sign of anoxia to appear, and this should be treated at once by inflating the child's lung with oxygen. A rising systolic pressure and widening pulse pressure may be warnings of carbon-dioxide accumulation due to underventilation or to an inadequate flow of gases to flush exhaled CO_2 from the anesthetic circuit.

Sweating is most commonly due to either CO_2 retention or to gross afferent stimulation in the presence of inadequate anesthesia or muscular relaxation. An increased minute volume and improved compliance will usually correct sweating.

Obstructive signs of respiratory obstruction may be audible or visible. Low-pitched snoring

sounds commonly originate above the larynx, due to the tongue or epiglottis falling back and blocking the glottis. High-pitched inspiratory stridor is usually due to partial laryngeal spasm, which should be corrected by deepening the anesthesia or by passing an endotracheal tube under direct vision. During laryngoscopic inspection the anesthetist can determine whether the obstruction lies at the cords or beyond, e.g., tracheal obstruction from a retrosternal goiter. Moist râles indicate the presence of bronchial secretions, and these should be removed by passing a bronchial aspiration catheter through the endotracheal tube. Lack of movement of the rebreathing bag spells respiratory paralysis, obstruction, or an endotracheal tube which lies in the esophagus. If obstruction is the cause, each abortive inspiration will be accompanied by indrawing of the supraclavicular fossae.

A continuous *electroencephalographic recording* can be useful for the early detection of cerebral anoxia in difficult cases. Cerebral cellular anoxia from anoxemia, arterial hypotension or hypoglycemia, and a deep general anesthetic itself, all produce a nonspecific pattern of electric depression in the EEG. The normal wave pattern of light anesthesia is replaced by slow waves of between 0.2-2.0 cycles/second, and later the pattern flattens out to complete electric silence. If very light levels of anesthesia are employed, then any depression of electric activity must be due to cerebral anoxia or hypoglycemia, and the cause can be quickly sought out and corrected.

Cardiovascular System.—The cardiac output depends upon a number of factors. Then must be considered the efficiency of the heart muscle, the valves, the coronary circulation, the conducting tissue, and the cardiac nerve. The hematologic, biochemical, and physiologic qualities of the blood must be within normal limits. The blood vessels should present no marked disease. The physiologic responses to vascular reflexes should be active, and finally, in some respects, there must be normal endocrine balance.

The integrity of the cardiovascular system presupposes a normal respiratory system, for there must be perfect oxygenation and adequate carbon dioxide elimination.

Blood pressure depends upon the cardiac output, the peripheral resistance, and the viscosity of the blood. During anesthesia, blood pressure fluctuations may occur. These reflect the basal fall due to vasodilatation and the compensating mechanisms trying to correct it. Observation of the pressure may detect a number of incidents, some associated with anesthesia, others not.

The *pulse* of an anesthetized patient may vary. These variations should be interpreted in conjunction with the blood pressure determinations. Specific types of rhythm may be noted.

From blood pressure and pulse observations such things as oxygen lack, carbon-dioxide accumulation, autonomic nerve stimulation, hemorrhage, shock, and responses to various other vascular reflexes may be deduced. Such observations are interpreted in the light of responses by the respiratory system, which give additional information.

Unless otherwise indicated, the blood pressure of a patient during anesthesia should be maintained in the neighborhood of its usual level. Factors responsible for a fall, such as hemorrhage, should be appropriately corrected.

Patients who are on cortisone therapy should have this continued during and after anesthesia and surgery. Those who have received it prior to surgery but to whom its administration has been discontinued should be carefully considered as possibly requiring intravenous cortisone therapy during anesthesia. Under these circumstances the previous treatment may have suppressed the patient's own cortical secretions. If this be the case, the stress of anesthesia and surgery may not be tolerated, and a persistent fall in blood pressure will follow which can be controlled only by cortisone.

Cyanosis, which is the visual index of the absolute quantity of reduced hemoglobin in the blood, is evident when there are at least 5 Gm/100 ml circulating. In the case of the individual with a normal blood count the hemoglobin will be approximately 80% saturated when cyanosis becomes apparent. It is not a sign to await in anesthesia, as anoxia may be present before cyanosis develops. In a polycythemic subject, however, it may not indicate hypoxia, while in the grossly anemic patient cyanosis cannot appear. Under certain

circumstances, cold for example, cyanosis may be seen locally; it is due to the narrowing and therefore stagnation of the vascular bed. This type of cyanosis is not indicative of anoxic anoxia.

Pallor may be observed in a patient who is known to be anemic, in one who has suffered severe blood loss, or in one who is in shock. In the latter two instances it is the result of compensatory vasoconstriction; this is also true of pallor occurring in that part of the body unaffected by a high spinal or epidural anesthetic.

The vessels in the nail bed should not only be watched for color changes but also tested for *capillary tone* by pinching. Capillary tone is diminished by deep anesthesia, as the vessels are paralyzed, while in shock there is vasoconstriction; both lead to a decrease in circulating blood.

Alimentary System.—The nerve supply of the gut is autonomic. The sympathetic portion influences the gastrointestinal tract in its entirety. The parasympathetic, however, although having an over-all influence, is divided into a sphere of vagal distribution, descending as far as the inferior mesenteric plexus and that of the sacral parasympathetic which, via the superior and inferior hypogastric plexus, innervates the lowest portion of the bowel.

A general anesthetic may, depending upon its depth, diminish the tone of the bowel and cause relaxation of the sphincters. A spinal anesthetic, on the other hand, will paralyze the sacral parasympathetic nerves and the sympathetic nerves up to the height to which it extends. The vagus nerve, however, will be unaffected and its action unopposed.

Other Systems.—In anesthesia for genito-urinary surgery it is important to know the segmental nerve distribution. Likewise in obstetric anesthesia it is of extreme importance to have an accurate knowledge of the motor and sensory innervation of the uterus and birth canal.

PRACTICAL CONSIDERATIONS

PREPARATION OF THE PATIENT FOR ANESTHESIA

The administration of an anesthetic should not be lightly undertaken. The induction

general anesthetic or the production of conduction analgesia is a major procedure.

The anesthetist should visit the patient prior to operation, elicit a history of previous illnesses and anesthetics, examine the patient, check urinalysis and hemoglobin, and then decide what preoperative medication and anesthetic management are most suitable for that patient undergoing that particular procedure.

In cases of election it is advisable to prescribe a barbiturate to ensure sleep the night before the operation. The combination of a quick-acting barbiturate with one of longer duration may be considered.

At the time of this examination the anesthetist prescribes certain drugs to be given before the patient goes to the operating room and leaves instructions concerning the general preparation, such as the withholding of food and fluid for 6-8 hours prior to anesthetic. He also takes this opportunity to reassure the patient. In certain cases such as diabetes, myasthenia gravis, or thyrotoxicosis, in patients being treated with cortisone, or in those whose hydration, acid-base balance, or blood volume is at fault, specific additional preoperative instructions may be necessary. Such may require consultation and additional help from other departments.

In the case of surgical emergencies, the premedication may have to be modified. The intravenous route for the administration of the premedicant drugs, with suitably modified dose, may be considered. Frequently the emptying time of the stomach is delayed in such patients. This factor may alter the choice of anesthetic technique and may affect the management of the case. It may even be necessary under certain circumstances to wash out the stomach prior to the induction of anesthesia. It can be emphasized here that vomiting during induction or emergence from anesthesia gives rise to greater mortality and morbidity than any other single factor.

Premedication Drugs.—Commonly, a narcotic such as morphine, Demerol, Pantopon, or Methadone or an hypnotic such as one of the short-acting barbiturates is given either alone or in combination to relieve anxiety, diminish pain, and produce a state of drowsy relaxation. Such a state permits the pleasant induction of anesthesia, lessens the quantity of toxic drugs

necessary to maintain anesthesia, and counteracts the undesirable side effects of the anesthetic. It should be noted that when pain is a feature of the preoperative condition, the use of a hypnotic alone will serve only to aggravate the condition by producing restlessness, for a hypnotic in the usual dosage has no analgesic action. It may also be noted that the use of some of these drugs may produce side effects which are undesirable; such an effect is the constipation which follows the use of morphine.

In addition to the sedative, either atropine or hyoscine is given. The anticholinergic action of these drugs depresses vagal activity, thereby diminishing bronchial secretions and lessening the vagal reflex responses of the heart. Hyoscine has yet another effect, that of producing amnesia.

The dose of these drugs is chosen according to the weight, age, and general condition of the patient, the presence of fever or an altered basal metabolism rate is also taken into account. Premedication drugs are usually given subcutaneously about one hour prior to the induction of anesthesia.

Avertin (2.5%, prepared according to instructions; 50-100 mg/kg.) or a solution of an ultrashort-acting barbiturate (Pentothal, 40 mg/kg in a 10% solution) may be given rectally, together with atropine or scopolamine subcutaneously, 30 minutes before anesthesia, to provide basal narcosis in very nervous subjects or in the young.

GENERAL ANESTHESIA

Analgesia means loss of sensation to pain without unconsciousness.

Anesthesia means loss of all sensation. Under general anesthesia there is also loss of consciousness. The types of anesthesia are described as (1) general and (2) conduction.

Discussion.—Generations of students have been brought up on the signs of anesthesia laid down by Guedel; these are described in terms of three stages, the third being that of surgical anesthesia, which in turn is subdivided into four planes depending on the depression of respiration and other reflex responses. It is not sufficiently appreciated that these signs were originally described for ethyl ether, and

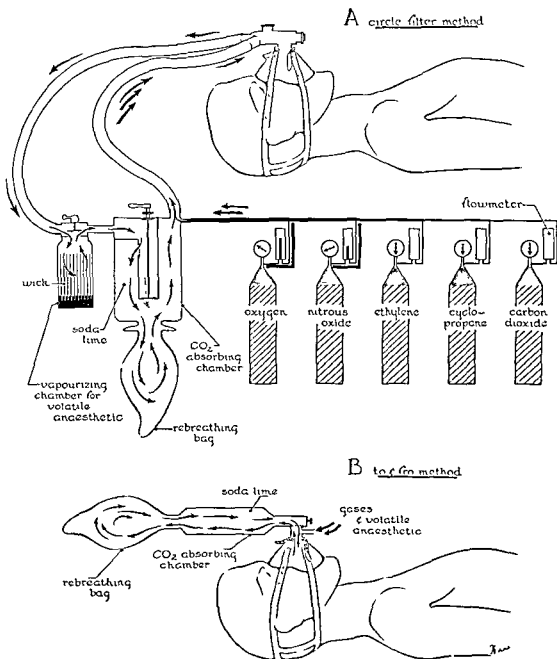


Fig 31—Anesthesia apparatus

TABLE 9 PROPERTIES OF INHALATION AGENTS

AGENT	SP. GR.	B.P.	PHYSICAL CHARACTERISTICS	POTENCY	MUSCULAR RELAXATION	INFLAMMABLE	REMARKS
Chloroform CHCl_3	Liquid 1.476 Vapor 1.12	61° C	Colorless oily fluid Sweet smell Irritates skin Vapor nonirritating	Very strong Blood conc 16 mg %	Extreme	No	Lowers pulse pressure. Diminishes cardiac output. Stimulates heart muscle. NEVER USE WITH EPINEPHRINE. May cause primary cardiac arrest. Toxic effect on liver. Causes nausea and vomiting. Causes acidosis.
Ether Diethyl ether $(\text{C}_2\text{H}_5)_2\text{O}$	Liquid .718 Vapor 2.6	34.5° C	Colorless fluid Pungent smell Irritating to skin and mucous membranes	Strong Blood conc 75 mg %	Good	Yes	Increases cardiac output (except when deep). Raises pulse pressure. Causes nausea and vomiting. Causes acidosis. Sympathomimetic.
Vinethene Divinyl ether $(\text{C}_2\text{H}_4)_2\text{O}$	Liquid .77 Vapor 2.2	28.3° C	Colorless fluid Musty smell Nonirritating	Strong Blood conc 40 mg %	Good	Yes	Suitable for induction, especially in children. Causes salivation.
Trichlorethylene $\text{CHCl}_2\text{CCl}_3$	Liquid 1.35 Vapor 1.5	87° C	Colorless fluid (blue added) Sweet smell Liquid irritating Vapor nonirritating	Use only at analgesic conc	Poor	No	Should only be used as an analgesic. Deeper stages cause tachypnea and arrhythmia. DO NOT USE WITH SODA LIME. Rarely causes nausea or vomiting.
Ethyl chloride $\text{C}_2\text{H}_5\text{Cl}$	Liquid .912 Vapor 2.28	12.3° C	Colorless fluid Disagreeable smell Very volatile Nonirritating	Strong Blood conc 25 mg %		Yes	Formerly used for induction. May cause arrhythmia. May cause laryngeal spasm. Causes nausea and vomiting.
Nitrous oxide N_2O	1.1	89° C	Colorless gas Sweet smell Nonirritating	Very weak Blood conc 25 vols %	Poor	No	Used for induction short cases or combined anesthesia.
Fiblenone C_8H_8	978	-103° C	Colorless gas Foul smell Nonirritating	Weak Blood conc 150 mg %	Poor	Yes	Not often used. More expensive than N_2O . Gives everyone in the operating room a headache.
Cyclopropane C_3H_6	1.1	-31° C	Colorless gas Sweet odor Slightly irritating	Strong Blood conc 15 mg %	Fair	Yes	Parasympathomimetic. Stimulates heart muscle. NEVER USE WITH EPINEPHRINE. Depresses respiration markedly. May cause cardiac arrhythmia. May cause nausea and vomiting. Rapid induction. Rapid elimination.

Pontocaine (Tetracaine, Amethocaine, Decicain).—Pontocaine, as procaine, is an amino benzoic acid derivative. The structure of its salt is dimethylaminoethyl-para-N-butylaminobenzoate hydrochloride. Its introduction in 1933 resulted from efforts to produce a local anesthetic drug with topical activity which is capable of producing more prolonged local or regional analgesia than that produced by procaine. It proved to be successful in both respects. The anesthetic potency of pontocaine is about 10 times that of procaine and, in concentrations commonly used for procaine, it is proportionately toxic. However, its potency allows for analgesic efficiency in much weaker dilutions. Bonica has assessed its corrected toxicity ratio (in mg/kg. body weight corrected for dilution) as 0.6 that of procaine, with an associated anesthetic index of 1.6 when compared with the same drug (See Table 10.)

TABLE 10*
PONTOCAINE VOLUME AND CONCENTRATION

USE	CONCENTRATION	VOL. ML	MG
Local infiltration	0.025%	200-300	75-100
Field block	0.05%	150-200	75-100
Small nerve block	0.1%	100-125	100-125
Block of larger nerves, plexus blocks, and epidural blocks	0.2%	50-60	100-125
Spinal block	0.3-0.5%	3-5	10-20
Topical application	1-2%	2-3	20-40

*From Bonica, J. J. *The Management of Pain*, Philadelphia, 1953, Lea & Febiger

Xylocaine (Lidocaine).—Xylocaine, an acetanilide derivative, represents the best addition to this type of drug in recent years.

The drug is extremely active when used either topically or by infiltration. It is non-irritant, soluble, and heat stable. Onset of analgesia is rapid and the degree is profound and prolonged. The systemic toxicity is little more than that of procaine.

For topical anesthesia of the larynx the optimum concentration is 4%. For nerve blocks and caudal and epidural blocks 1-2% solutions are used. Epinephrine may be added to produce the desired vasoconstriction with increasing duration of action, at the same time reducing the rate of absorption.

In comparison with other agents, most authorities report a higher incidence of successful blocks and a more profound analgesia when Xylocaine is used.

Nupercaine (Perthane).—This drug is a quinoline derivative and is the longest acting of all local anesthetic agents in common use. On a mg/kg body weight basis it is 20 times as toxic as procaine. This toxicity is compensated for by its anesthetic activity in high dilution. For topical use 0.5-1% may be used in small quantities. In the urethra, 1% is not safe because of the possibility of rapid absorption. For infiltration anesthesia, a solution of 1:2,000-1:4,000 is satisfactory. For nerve block procedures, a solution of 1:1,000-1:1,500 gives good results. For spinal anesthesia, a solution of 1:1,500 is made up in 20 ml. ampules, which has been used for many years according to the techniques described by Jones and Wilson. For low spinal anesthesia, a 1:400 solution in dextrose is available.

The relative safety of this drug results from its potency and subsequent anesthetic activity in high dilutions, which results in a lower corrected toxicity.

A hazard has arisen as a result of the similarity of the name with procaine. Pharmacists have, on occasion, confused 2% perthane with 2% procaine, a mistake which has produced fatalities.

Types

Topical.—The drug is applied to a mucosal surface where it anesthetizes nerve endings.

Local Infiltration.—The drug is injected and affects the nerves with which it comes in contact. This method is applicable to minor procedures, such as the excision or biopsy of small tumors, or for suturing lacerations. A vasoconstrictor is usually added to prevent rapid absorption of the anesthetic drug and to diminish hemorrhage.

Field Block.—The terminal branches of the nerves are blocked by injecting a wall of local anesthetic along the borders of the area they supply. A continuous intradermal wheal is followed by gradual infiltration of deeper tissue planes to any required extent.

Nerve Block.—The nerves are blocked at any convenient point along their course to the periphery of the body before they have divided.

PRACTICAL CONSIDERATIONS

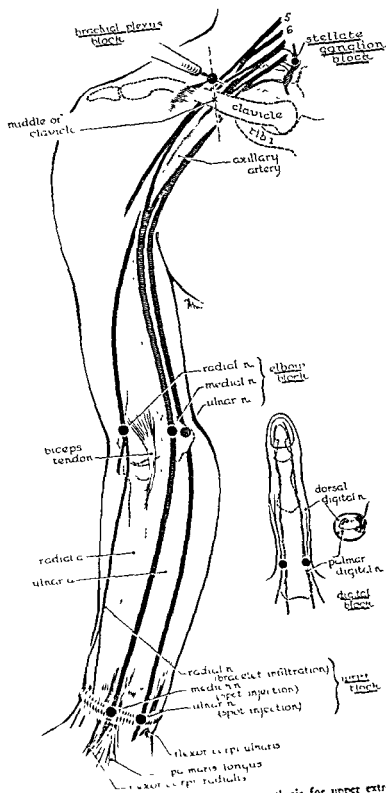


Fig. 57—Regional nerve block anesthesia for upper extremity

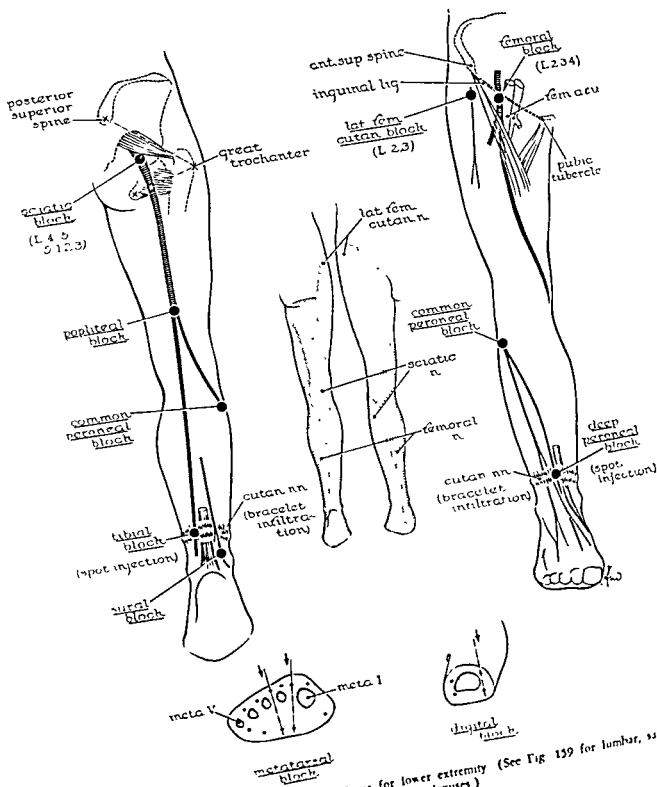


Fig 38--Regional nerve block anesthesia for lower extremity (See Fig 139 for lumbar, sacral, and pudendal plexuses)

into their terminal branches. Efficient nerve blocking requires accurate anatomic knowledge. Any sensory nerve may be blocked, but for many the landmarks are variable and difficult to define accurately. The addition of hyaluronidase to the local anesthetic drug is no excuse for an inaccurately placed injection. Although hyaluronidase destroys the cement substance in the tissue architecture, it will not diffuse between tissue compartments and it will weaken the local concentration of the drug by increased diffusion.

Paravertebral Block.—The spinal nerves are blocked as they emerge from the intervertebral foramina or in the vicinity of the vertebrae. A paravertebral block may be performed at any level of the spine and used for diagnostic or anesthetic purposes.

Epidural Analgesia.—The spinal nerves are blocked in the epidural space. This procedure may be carried out at any spinal level; most commonly L2-3 and L3-4. It provides analgesia similar to that produced by a spinal anesthetic but is slower of onset. Sympathetic paralysis may be complete, while sensory loss may be made to extend to the jaw; motor paralysis may be less profound.

Spinal Analgesia.—

PRINCIPLES.—The spinal nerves are blocked within the dural space. Both anterior and posterior roots are affected. The extent of the analgesia produced from a relatively small dose of analgesic drug is much greater than can be obtained by a similar dose using any other regional technique. Spinal analgesia can be used for operations anywhere below the clavicles but is commonly reserved for operations below the diaphragm. It provides ideal conditions for abdominal operations or operations on the lower extremities but should not be administered by an unskilled person without adequate supervision.

There is a great variety of drugs available for spinal analgesia. Those most frequently used are procaine (Novocain), Pontocaine, and Nupercaine. The drug selected may be used either as a hypobanic, hyperbanc, or isobanc solution. The type of solution depends on the technique that is to be employed, since most techniques of administration rely to a certain extent on gravity control of the range of

spread of the anesthetic effects and to a lesser degree on the volume injected and rate of injection. The duration of their effects varies with different drugs, but with the same drug the duration may be affected by the concentration, the total dose of the drug injected, or by the addition of a vasoconstrictor. The analgesic effect following the administration of procaine is approximately 1 hour, of Pontocaine 2 hours, and of Nupercaine 3 hours.

In order to be able to provide spinal analgesia of longer duration the *continuous* spinal technique was introduced by Lemmon, employing a malleable needle and a split mattress. Any of the local anesthetic drugs may be used in the continuous spinal technique, but procaine, Pontocaine, and Metycaine are those which are used most frequently.

The continuous technique may also be employed in a somewhat similar manner in epidural analgesia to provide prolonged analgesia. A popular form is the continuous caudal technique utilized in obstetrics.

Toohy modified this method, using a Huber tipped lumbar puncture needle, through which is passed a No. 3½ gauge ureteral catheter or vinyl plastic tube. This catheter or tube can, with care, be passed to any desired height. It can also be used in the epidural space, a method which carries less morbidity than that by the subarachnoid route.

TECHNIQUE.—

Ascertain that a spinal anesthetic is one of choice and premedicate accordingly.

Preparation of Equipment.—All instruments, ampules, files, etc., must be autoclaved. All drugs used in this procedure must be fresh preparations, and the ampules in which they are contained must be checked. It is advisable to use only those ampules on which the description is etched and not printed. The lumbar puncture needle should not be larger than No. 20, finer ones may be used when the technique has been mastered. The needle should be tested before the puncture by running through a piece of gauze. Full sterile technique should be observed by the anesthetist.

Preparation of the Patient.—The correct posture is imperative if lumbar puncture is to be

simple and atraumatic. The back is painted with iodine or suitable substitute and then draped. Anesthetic solution should then be infiltrated at the elected site. This should not be higher than L2-3, while a puncture can be satisfactorily performed as low as L5-S1; the commonest level is L3-4 which is the first space above the intercrural line. If a vasopressor is deemed advisable, as for instance in the case of a "high spinal" to counteract an anticipated fall in blood pressure, it should be given at this time, along with the infiltrating drug. Vasoxyl 10 mg is frequently considered

the vasopressor of choice as it does not stimulate the central nervous system as does ephedrine.

The Puncture—A lumbar puncture is the most made. A midline puncture is usually the simplest and the least traumatic. In performing this, the anesthetist should familiarize himself with the various resistances met by the needle as he pushes it inward: (1) the skin; (2) the interspinous ligaments; (3) the ligamentum flavum (the tip is then within the epidural space); (4) the dura. On passing through the dura, the bevel will lie within the subarachnoid

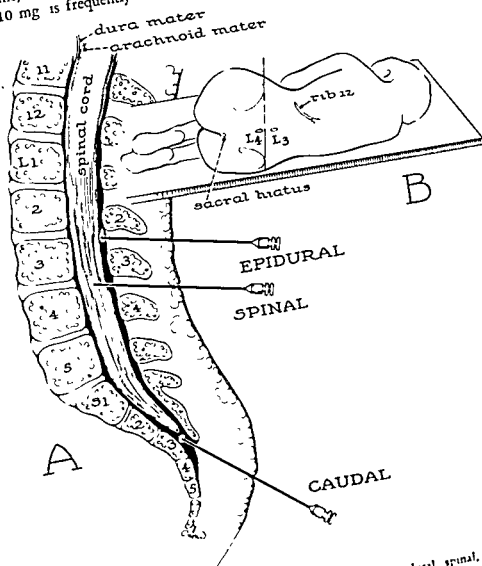


Fig. 59 Spinal anesthesia
A. Diagram showing position of needle for induction of epidural, spinal, and caudal analgesia.
B. Posture for spinal tap in lateral position. Note patient at edge of bed, with spine well flexed and pelvis and shoulders vertical.

space. It is important that the entire lumen of the bevel should be so placed that it should not lie halfway in and halfway out. The stylet is then withdrawn and when a free flow of cerebrospinal fluid ensues, the spinal analgesic is injected. The iodine is then removed with alcohol, the back dried, and the patient's position gently changed in order to attain the correct level of anesthesia. It is unwise to test for an anesthetic level too vigorously, if at all, as this makes the patient apprehensive. Thereafter, careful observations should be made of the patient's clinical condition until his recovery.

Oxygen should be administered if there is respiratory depression, while a sleeping dose of some agent may be thought advisable.

Precautions.—At all times, during any form of conduction analgesia, an anesthetic machine, with which to administer oxygen, should be at hand. Likewise, endotracheal tubes and a laryngoscope should be readily available in order to efficiently oxygenate either a subject who has received a relative overdose of anesthetic agent, from too rapid absorption, or one in whom spinal or epidural analgesia has paralyzed the phrenic nerves. Pentothal should be prepared and ready for use in order to control the convulsions which may result from a relative overdose.

Advantages of Spinal Analgesia:

- 1 Excellent muscular relaxation is provided
- 2 There is little disturbance of metabolism
- 3 The inhalation of irritating drugs is unnecessary so that the patient does not suffer from postanesthetic nausea and prolonged sleep
- 4 The use of the electrocautery and other electric appliances is permissible

Disadvantages of Spinal Analgesia

- 1 It is difficult to control
- 2 Its duration is variable and uncertain
- 3 Technical difficulties may result in absence of analgesia
- 4 Motor analgesia may ascend to high levels. If this occurs, respiration can and should be aided and adequate oxygen provided
- 5 Severe circulatory collapse may follow the administration of a spinal analgesic

6. Consciousness during operation is not desirable in some cases.

7. Retching may follow traction on the viscera, since the vagal pathways are not blocked.

8. Postoperative neurologic complications may follow spinal analgesia. The most common of these is headache, the most serious ones, though uncommon, are of disastrous nature.

Regional analgesia is becoming more popular as techniques improve. This is true not only in providing analgesia for surgery but, perhaps of more importance, in the various diagnostic and therapeutic procedures related to vasospasm, causalgia, etc.

Reference should be made to standard texts for further details about conduction analgesia.

ANESTHESIA AND SURGICAL BLOOD LOSS

Anesthesia can have a profound effect on surgical hemorrhage. Poor anesthetic technique may double the amount of blood lost during operation, whereas good tranquil anesthesia, with attention to a few simple rules, can effect great economy in blood as well as the surgeon's time and effort during a difficult dissection. "A pint of blood saved is worth two in the bank," is nearly true, quantitatively as well as qualitatively, when one remembers that every 500 ml. of bank blood is made up of 150 ml. of dextrose citrate solution and only 350 ml. of whole donor blood.

Blood loss from a surgical incision is dependent on two factors, the size of the cut vessels in the incision and the pressure of the blood in those vessels. Measures to diminish blood loss are based on reducing one or the other of these factors. (See Chapter 4)

Controlled Hypotension

There are many situations where injection of vasoconstrictor solutions is undesirable or dangerous; for example, in the neighborhood of malignant tissue, where injection techniques may spread the disease further; in these circumstances, attention is directed to reducing the hydrostatic level of pressure in the vessels of the operative site. An obvious and simple method is depression of local pressure by elevation of the part; for example, the use of a

head-down tilt during operations for varicose veins or the placement of the arm on a raised support in operations on the upper limb. Lord Lister pointed this out many years ago, but succeeding generations forget and have to re-discover this simple maneuver.

In recent years a great many papers have appeared on the subject of controlled arterial hypotension.

Before inducing hypotension as an aid to any particular operation, the morbidity of this measure should be considered. In patients in whom there is pre-existing coronary disease, cerebral arteriosclerosis, or renal inefficiency, the marked fall in blood pressure may prove fatal.

It is argued that whatever the initial mean arterial pressure, by the time the blood reaches

the vascular tone is in a state of vasodilatation. In oligemic shock, when compensatory vasoconstriction exists, blood supply to vital organs may be inadequate at these levels.

Curiously enough, while so much attention has been focused on the arterial pressure, little thought appears to have been given to the venous side of the circulation. This omission is the cause of some inconsistency in the published work on arterial hypotension, because from the standpoint of blood loss, the venous pressure is probably much more important than the arterial pressure, since it is a more labile factor and more directly affected by changes in respiratory resistance.

Changes of intrathoracic pressure are transmitted directly to the great veins and from there via the intervertebral foramina to the valveless extradural plexuses, which equalize

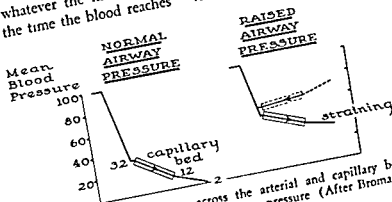


Fig. 60—Changes of mean blood pressure across the arterial and capillary beds under normal conditions (on left) and with raised airway pressure (After Bromage)

the capillary bed, peripheral resistance has reduced the mean pressure to about 32 mm Hg, and this pressure of 32 mm Hg is the only one of importance; higher pressures are merely resting supplies in hand for the changing needs of a conscious dynamic organism. Therefore, provided peripheral resistance is abolished by removal of sympathetic tone, and provided the blood volume remains constant, a mean arterial pressure slightly in excess of 32 mm Hg should be sufficient. However, this argument fails to consider the needs of secondary capillary circulations such as are found in the liver and pituitary (pituitary-hypophyseal portal system), and for these systems mean pressures somewhat in excess of 32 mm Hg are probably necessary. In practice, mean pressures in the region of 50-60 mm Hg appear to be safe (i.e., 75/40-90/50 mm Hg), provided

venous pressures all over the body. Thus, during coughing or straining or partial obstruction of the airway, the venous pressure throughout the body rises, and in extreme cases blood flow in the capillary bed may be halted or actually reversed. In the conscious subject, venous pressures follow closely upon changes of airway pressure. But, under anesthesia, venomotor reflexes appear to be exaggerated, so that in stead of returning to normal after a bout of coughing, a raised venous pressure persists for 20-30 minutes, and during this time blood loss from capillaries and venules is greatly increased.

Translated into practical terms, this means that for optimal surgical conditions anesthesia must be entirely tranquil at all times. There must be no coughing or straining or increased airway resistance from the moment of induction onward.

Completely tranquil anesthesia, with the patient postured so that the operative site is at the highest level, goes far to provide a relatively dry surgical field. Where an extra reduction of bleeding is required, as in neurosurgery, arterial hypotension is added to this background of tranquility.

Arterial Hypotension

Arteriotomy.—The circulating blood volume is temporarily reduced by removing up to 1,200 ml. of blood from a major artery, usually the radial, and the blood is reinjected at the end of operation. This procedure induces a state of temporary oligemic shock with arterial hypotension and vasoconstriction. Although very effective for neurosurgery, the accompanying vasoconstriction jeopardizes the blood supply to parenchymatous organs and makes it a dangerous method.

Spinal Blockade.—High subarachnoid or extradural spinal block produces a temporary preganglionic sympathectomy, with accompanying vasodilatation and fall of arterial pressure. The systolic level is controlled by posture and raised by vasopressor drugs if necessary.

Ganglionic Blockade.—Some drugs of the methonium group, pentamethonium and hexamethonium, produce ganglionic blockade which lasts about 45 minutes when given intravenously. A thiophanium derivative, Arfonad, is shorter acting and more controllable, and this is administered by continuous intravenous infusion. Drugs with less clear-cut actions may also be used. For example, chlorpromazine has a combined adrenolytic and ganglion-blocking effect, as well as producing depression of the central nervous system, this may be useful for intraocular operations where a low intraocular tension combined with prolonged postoperative sedation is desirable.

HYPOTHERMIA

The state of hypothermia appears to have been introduced by Simpson and Hering of Edinburgh, who in 1905 lowered the temperature of cats for experimental purposes. They described the condition produced as being one of artificial hibernation and realized that in this state metabolism was depressed.

This theory was not utilized clinically until Temple Fay incorporated it in the treatment of malignant tumors. It is interesting to note that since then, cooling has been shown to depress hypophyseal function. The regime of cooling patients was later tried in the treatment of schizophrenia.

The first really useful clinical application of hypothermia was its introduction to combat the hyperthermia that may follow major surgery in children and infants. Since then research work has been undertaken in many centers, some of the most important contributions coming from Bigelow of Toronto.

Universal employment of hypothermia has followed the postulation that in this state metabolism is reduced; the oxygen consumption of the brain is diminished by approximately 50% at a temperature of 30° C. The temperature of 30° C. is regarded in many centers as being the optimum for cooling purposes.

Advantage can be taken of hypothermia in certain types of surgery in which the blood supply, and therefore oxygenation, of the brain may be interfered with for periods which previously would have been considered impossible, without being either lethal or grossly morbid. Use of this principle can also be made under other circumstances, such as closed head injuries, when it may be thought beneficial to reduce brain metabolism and incidentally to relieve brain edema.

Physiologic Responses to Hypothermia

The natural response to cold is the shiver reflex. If shivering is allowed to occur when the body is cooled, the initial responses are entirely different from those found in a subject who is prevented from shivering by anesthesia or a muscle-depressant drug. If shivering is allowed to occur, oxygen consumption is greatly increased, the blood pressure and pulse rate rise, and the cardiac output is grossly augmented. In similar fashion, respiratory rate, depth, and the minute volume are all elevated.

Once a temperature of 30° C. is reached, the subject is almost completely anesthetized by the cold; he lies as if in a trance, requiring only nitrous oxide and a bare minimum of other anesthetic agents before surgery is undertaken. Further discussion of the body's re-

sponses refers to those conditions that occur when shivering has been prevented or controlled.

Cardiovascular System.—There is a reduction in blood pressure and pulse pressure while at the same time the pulse rate is lowered. Below a temperature of 30°C . myocardial irritability is increased as demonstrated by the incidence of cardiac irregularity, the frequency of which increases with decreasing body temperature. The advent of auricular fibrillation, which is not uncommon, though innocuous in itself, should be regarded as a sign that the pacemaker is wandering and that keen observation and great care be exercised. When the temperature is reduced to between $28^{\circ}\text{--}26^{\circ}\text{C}$., irregularities may herald ventricular fibrillation.

However, at this temperature ventricular fibrillation may occur spontaneously, or it may follow a period of marked bradycardia accompanied by extrasystoles.

The activity of the myocardium in the cooled patient or experimental animal shows specific electric changes which are reversible. Ventricular fibrillation is most frequently encountered in operations that necessitate the handling of the heart. Many factors besides actual trauma have been held responsible for this ever-present hazard of hypothermia. Anoxia, acidosis, hyperkalemia, and acetylcholine metabolism have each in turn been incriminated. There is therefore no certain knowledge as to why this complication occurs.

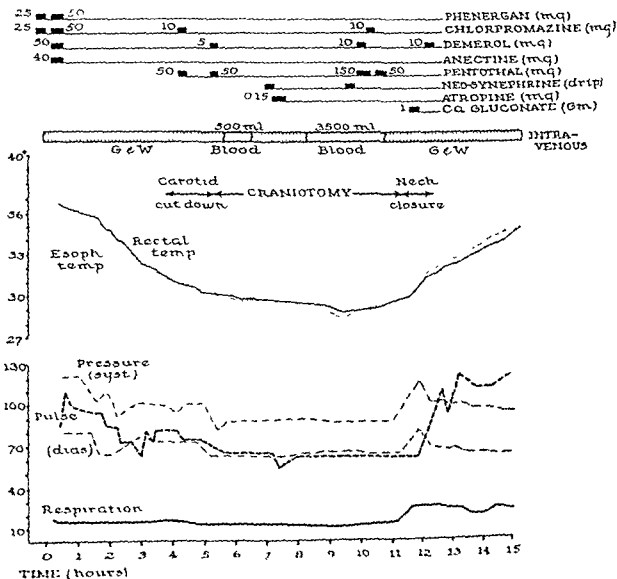


Fig. 61—Chart showing vital signs during a craniotomy under hypothermia

when cooling the mammal Burton and Edholm record that the heart of the European marmot can easily be made to fibrillate by cold during its active stage, whereas the heart of a similar animal when hibernating will not so respond

Respiratory System.—The rate and depth of respiration increase in the early stages of cooling and then they decrease, the oxygen consumption, after an initial rise, diminishes. This diminution in oxygen consumption succeeds the fall in minute volume. The carbon-dioxide content of the alveolar air is decreased, but the solubility of carbon dioxide is increased, and the arterial content rises. The increased plasma carbon-dioxide tension counterbalances the swing to the left of the oxygen dissociation curve brought about by cold. When the temperature is lowered, the respiratory center becomes less active and the carotid body relatively more so.

Blood Gases.—The arterial oxygen concentration is well maintained with normal ventilation. There is no evidence that breathing an atmosphere enriched with oxygen is of advantage. The percentage of dissolved oxygen increases with the lowering of temperature. The plasma carbon-dioxide level tends to rise and the pH to fall very gradually but progressively.

Blood.—The hematocrit is elevated, and the viscosity of the blood is increased. There is therefore a loss of plasma for which as yet there is no accounting. The bleeding time may be increased, and the coagulation process may in some way be affected.

Kidneys.—The renal output varies with the blood pressure and the effect of the antidiuretic hormone. No pathologic changes have been reported except those by Knocker. In cooled dogs she found changes in the kidneys analogous to those found in death from anoxia.

Liver.—During the induction of hypothermia, glycogen is synthesized. Thereafter, the blood sugar level rises. This fact seems first to have been pointed out by Claude Bernard and rediscovered in the Dachau concentration camp experiments. Knocker found changes in the liver similar to those she found in the kidney. When hypothermia is established, the many functions of the liver are slowed down;

it is therefore important to modify the dose of all drugs given under these circumstances.

Biochemical Changes.—When patients are kept cool for a considerable period, the levels of potassium, sodium, and chloride tend to vary. They therefore need careful watching and correction by appropriate infusions. Enzyme activity is greatly modified during hypothermia.

Clinical Hypothermia

The Use of Drugs in Hypothermia.—Drugs such as chlorpromazine or Hydergine, by virtue of their antiadrenergic action, cause vasodilatation and therefore facilitate the production of hypothermia. Some of the other actions of the drugs may also be of advantage during the induction and maintenance of hypothermia. The anticholinergic and antihistaminic efforts of drugs such as Phenergan and the sedative effect of Demerol may be incorporated in the medication which precedes hypothermia. Such a mixture prevents shivering, induces anesthesia, and expedites the cooling by the vasodilatation produced. Should shivering occur under these circumstances it can readily be controlled by minimal doses of Pentothal. The mixture of chlorpromazine, Phenergan, and Demerol or similar drugs was introduced by Laborit and Huguenard as the lytic cocktail. This mixture alone is capable of producing a type of anesthesia, this procedure being termed potentiater anesthesia. These drugs alone do not produce hypothermia as was originally thought, neither is there valid evidence to support the claim that their use prevents the occurrence of cardiac irregularities when they are used in association with physical cooling.

By virtue of the fact that these drugs depress autonomic nerve transmission and therefore autonomic nerve responses, it has been claimed that suitable mixtures may be used advantageously in the management of shock. This theory has been given trial, but as yet it has not been universally accepted.

Indications for the Use of Hypothermia

Short Term.—This has been advocated for cardiac surgery when the circulation will be interrupted, also for certain neurosurgical operations such as cerebral aneurysm or vascular

tumor, when the circulation of the brain may be temporarily cut off. It may find a place in surgery of the major ablative or extirpative type.

Long Term.—This is carried out in some centers in the treatment of certain cases of closed head injury, ruptured cerebral aneurysm, encephalitis, and hyperthermic poliomyelitis. There are some who advocate its use in the treatment of tetanus and of certain hyperthermic conditions.

Methods of Cooling

There are the following methods: (1) surface cooling, (2) extracorporeal cooling, and (3) cavity irrigation. The details of these methods must be studied elsewhere. Surface cooling appears suited to neurosurgical cases, while the extracorporeal method, in which blood is artificially by-passed through a cooling and possibly also oxygenating apparatus and then returned to another vessel, is more adaptable to heart surgery.

Once a patient has been cooled, great care has to be taken to make accurate observations. The temperature should be recorded in the esophagus distal to the bifurcation of the trachea and also in the rectum. It should be noted that when a subject is cooled, there is a continuous drift downward of a further 2° C. after active measures of cooling have been discontinued. This is because the muscle bed of the body acts as a reservoir of cold. It should also be noted that there may be a difference of up to about 4° C. between the temperatures recorded in the esophagus and rectum. The former is that taken as being indicative of the heart temperature. Electrocardiographic studies must constantly be made and the blood pH checked from time to time.

When hypothermia procedures are used, there must always be at hand a defibrillator which has been previously checked and found in working order.

During the long term cases, monitoring must also include daily studies of hematology and biochemical importance as well as urinary secretion. Dependent upon these is the quantity and quality of the intravenous therapy which

also must contain additional vitamins. Great care in these long-term cases must be taken to preserve a perfect airway, avoid infection, and prevent shivering.

CHOICE OF ANESTHETIC

The anesthetic selected should provide the patient with a comfortable and not too unpleasant interlude and the surgeon with nearly ideal operating conditions as possible without increasing the risk to the patient.

The patient may have prejudices regarding anesthesia, and his wishes must be given consideration; however, his physical status and the nature of the contemplated surgery are the more important factors. The anesthetic drugs selected should be such that their action does not adversely affect other complicating diseases. For example, patients with respiratory infections are usually safer if not given an inhalation agent, and patients in shock should not receive a spinal anesthetic or straight Pentothal anesthesia. Shocked patients should, if possible, not be anesthetized until the state of shock has been brought under control. If, owing to the nature of the condition, the patient must be anesthetized at once, this should not be undertaken in a manner that might interfere with the patient's compensatory reflexes.

The anesthetist should know the surgeon's preferences, how much relaxation he will require, and how long he is likely to take to perform the operation. For most intra-abdominal operations, it is necessary to provide a marked degree of relaxation, whereas, for example, in the case of a mastectomy, a less potent anesthetic agent or a lighter level of anesthesia suffices.

COMPLICATIONS FOLLOWING ANESTHESIA

It is not possible here to give a detailed account of all the complications which may follow anesthesia. As in the former paragraph, the aim of this brief outline is to convey fundamental ideas. It may be stated that the vast majority of the sequelae referable to anesthesia are preventable. Thought, therefore, must be given to possible deleterious effects

when agents are selected and procedures performed. Each system must be mentally reviewed: will this cause a fall in blood pressure? If so, will it be harmful? Is this likely to cause atelectasis? Will this ensure that the cough reflex is regained soon after the operation? Will this produce respiratory acidosis? If such and such be done, certain complications might occur; are the benefits that it confers worth a possible risk? Can the operative position produce ill aftereffects? Has due caution been paid to the prevention of nerve palsies, cautery burns, pressure sores, and explosions?

RESUSCITATION AND OXYGEN THERAPY

Reference is here made to the sections elsewhere in this book on shock, transfusions, infusions, and cortisone, as the anesthetist must be as familiar as any one with these subjects. Details concerning the care and treatment of a patient in shock, the optimum time for operation, the anesthetic to be given in these circumstances, and the postoperative care are integral parts of his work. Indeed, in time of war or emergency, he may be solely responsible for them. At all times the treatment must be based upon what is known of the underlying physiopathology. Incorrect treatment may aggravate the condition.

The use of oxygen may be of benefit, it may produce no response or if not used correctly, it may have ill effects which might even prove fatal. The methods of administration should be carefully considered before oxygen is used, and its use should be supervised. It should not be left to someone without knowledge, for under such circumstances administration might be dangerous, while at the best it would be wasteful.

Normal blood of a patient breathing room air contains 20 ml oxygen per 100 ml. Of this, 19.7 ml % is carried as oxyhemoglobin, producing 95% saturation, and 0.3 ml % is in solution in the plasma. When 100% oxygen is given, the hemoglobin becomes 100% saturated, an increase of 5%, and the concentration in the plasma rises to 2.3 ml %. This gives a total content of 23.5 ml /100 ml blood, or a percentage increase of over 15%.

Advantage may at times be taken of the physical and pharmacologic properties of helium and carbon dioxide. The former, though inert in itself, lowers the density of inhaled gases; the latter is the most powerful stimulant to the respiratory center. Oxygen under a pressure of 4 ml water during expiration may be valuable in re-establishing the normal transalveolar tensions in a case of pulmonary edema.

The necessary humidity of gases for inhalation should be remembered and the benefits of high humidity under certain circumstances known, while familiarity with the nebulization of drugs, their mucolytic and bronchodilator nature, should be gained.

THE POSTOPERATIVE RECOVERY ROOM

There are certain principles of importance to both anesthetist and surgeon during the stage of early postoperative care.

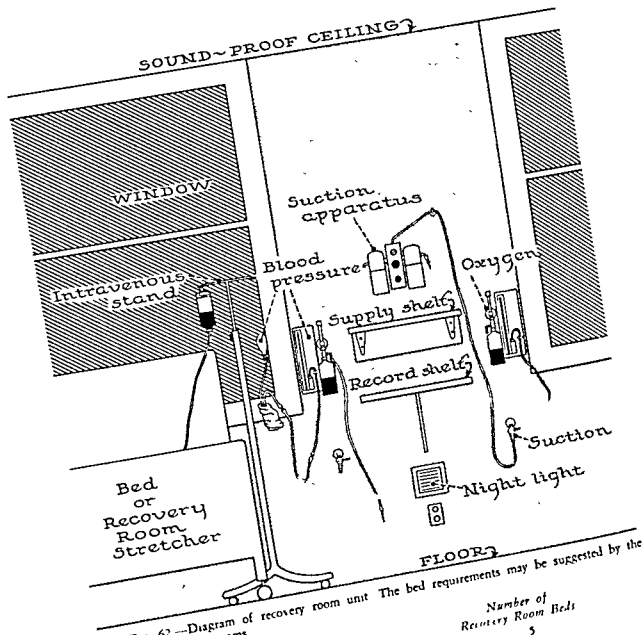
1. When patients leave the operating room, with few exceptions, they should have regained their cough reflex.

2. Patients may have to be handled; this should be done in the most gentle manner possible. The sympathetic control of blood vessels and the automatic vascular reflexes may still be depressed; hence roughness at this stage may precipitate a fall in blood pressure. Such a fall has been known to be fatal.

3. An unobstructed airway must be obtained. To ensure this, suction must be at hand; if possible, there should be alternative facilities to secure it, lest one method break down.

4. Posture is of considerable importance. The unconscious patient should be placed in the lateral or post-tonsillectomy position, so that ingress of foreign material into the trachea is avoided. Thereafter, the patient can be turned every hour to ensure equal ventilation of both lungs. This position is feasible after most operations. The beds should be of a type that can rapidly have the foot elevated. Specially designed recovery-room stretchers and beds are now available.

5. Equipment for oxygen therapy should be readily available.



number of operating rooms

Number of
Operating Rooms

3
5
7
9-10
12
14-16
18-20

Number of
Recovery Room Beds

5
7
9-10
11-13
14
17-20
22-25

6. Intravenous therapy will require special supervision, while the facilities of the blood bank should be readily available

7 Gastric and other suction, drainage tube and dressings, bladder irrigations, casts and such like must constantly be watched.

8 Pain is relieved in the recovery room according to the individual's needs and not by routine prescription of drugs that might prove ill suited

9 All therapy, observations, and records of the vital functions should be checked. The ideal system would be to have a chart continuous with the anesthetic chart, so that the whole procedure can readily be reviewed

10 Physiotherapy directed toward the minimization of pulmonary and venous complications may be initiated

11 There should be readily available all equipment that might possibly be needed, including the following

a Mouth gag, mouth props, a variety of airways to suit all ages, metal suction tips and rubber or plastic suction catheter, a laryngoscope with a variety of blades, endotracheal catheter, and bronchoscopes with spare bulbs

b All equipment necessary to support the circulation, such as intravenous equipment and solutions, cutdown sets, and intravenous stands

c Equipment for assisting or controlling respiration, such as an anesthetic machine with which to give oxygen, and some form of resuscitator

d Consideration given to the equipment necessary for looking after special cases, such as laryngectomies and hypothermic procedures

e Emergency equipment and personnel in the event of cardiac arrest.

f Emergency bell and electric clock with sweep hand

12 A variety of drugs should be readily available, such as (a) narcotics and their antagonists, (b) atropine and hyoscine, (c) barbiturates and their antagonists, (d) vasopressors, (e) analeptics, (f) anticoagulant drugs, (g) antihemorrhagic drugs, (h) antihistaminic drugs, (i) antibiotics, (j) ATS, insulin, and hydrocortisone

RECORDS

The charting of information should never replace the care and attention needed to give an anesthetic properly, but there should be time to record the reactions of the vital functions and the use of various agents, drugs, and infusions. Such observations may be of help during the postoperative period, and, if accurate, their value for analytical purposes may be great. In this day and age their medicolegal significance has greatly increased

EXPLOSIVE HAZARDS

The anesthetist should always be aware of the possibility of an explosion, which necessitates a source of ignition and an inflammable mixture. The former may come from (a) a spark from electric equipment as exemplified by endoscopic instruments, electric cautery, switches, plugs, light lamps, or faulty wiring, (b) an open flame or burning instrument; (c) static electricity

It has been stated that switches and plugs in an operating room should be of the explosion-proof variety, unless they are placed 5 feet above the ground. All endoscopic equipment should be carefully checked and wiring faults eliminated. Only under special circumstances should the cautery or similar instrument be used in the presence of inflammable gases or vapors; such circumstances include proper ventilation, the use of a high humidity atmosphere, and the aspiration of the exhaled gas mixtures. Every effort should be used to reduce the chance of static sparks occurring. These include the use of cotton clothes and coverings for patient and personnel, the use of conducting floors, and the grounding of all equipment and personnel.

In order entirely to eliminate the possibility of explosion, those techniques and agents must be used in which inflammable gases, vapors, or liquids are avoided. Such include all forms of conduction analgesia, barbiturate anesthesia, nitrous oxide and oxygen mixtures, trichlorethylene analgesia, intravenous narcotics, and relaxing agents or combinations of them. To this list may be added one of the oldest anesthetic agents, chloroform, and one of the most recent, fluothane.

FINAL REMARKS

There has been little discussion of anesthesia for specific operations, for patients suffering from various disorders, and for those of different age groups. The choice of anesthetic does not depend so much upon the agents as upon the manner of their administration. There are exceptions to this statement. The part-time or learning anesthetist should perfect one technique at a time. He should avoid using agents with which he is not familiar even if they are theoretically the agents of choice.

In dealing with children the scaled-down anatomy and modified physiology must be considered. With the aged and with patients who are suffering from such conditions as heart disease, pulmonary insufficiency, and glandular disorders, different factors must be noted and appropriate action taken.

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Film References

	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Intravenous Anesthesia With Barbiturates (1953) (By Mary Karp, M D, D Earl Remlinger, Jr, M D, Charles D Anderson, M D, Lewis C Hitchner, M D, and William O McQuiston, M D)	34 min	Sound Color	Abbott Laboratories North Chicago, Ill
The Dynamics of Respiration (1944) (Medical School, University of Wisconsin Departments of Anesthesia, Radiology and Photography)	40 min	Silent Color	American Medical Association Committee on Medical Motion Pictures 535 N. Dearborn St Chicago 10, Ill
Endotracheal Anesthesia (1944) (Prepared for use in training anesthetists in Great Britain, supervised by Dr I W Gilt and the anesthetics staff of Westminster Hospital, London)	25 min	Sound	International Film Bureau 57 E Jackson Blvd Chicago 4, Ill
Regional Analgesia (Presents the most important clinical details of regional analgesia) (1952) (By Daniel C Moore, M D, and John J Bonica, M D, Seattle)	36 min	Sound Color	Winthrop-Stearns, Inc. 1450 Broadway New York 18, N Y

Chapter 9

Surgical Technique

David W. MacKenzie, Jr, MD, and Mary G Warnock, RN.

PRINCIPLES OF WOUND HEALING

Problems relative to the healing of wounds, acquired because of trauma or surgical intervention, are of fundamental importance. A surgeon's interest in and ability to cope with such problems are an accurate measure of his technical skill. Failure to appreciate the principles involved, which are few in number and entirely self-evident, produces disastrous results. An understanding of the natural forces of healing, of the sequence and timing of events in the process of repair, and of factors which, on the one hand, expedite and, on the other, retard this process is essential to good surgery.

Wounds are classified as follows:

1. Abrasions
2. Contusions
3. Lacerations, with or without contusion, abrasion, or loss of substance
4. Burns
5. Incised wounds, clean, contaminated, or infected

The first two categories will not be considered at present. The third, concerning traumatic lacerations and associated complications, will be covered under wound excision and débridement later in this section.

The healing process in surgically incised wounds is precisely the same as that in traumatic lacerations, except that it is usually

quicker, with less deformity and loss of function. The rate of healing and the ultimate anatomic and physiologic results obtained depend to a very great extent upon the technique of the operator. Other contributing factors will be dealt with subsequently.

Surgical dissection may be *sharp* or *blunt*. Sharp dissection is carried out with the blade of the knife, blunt dissection, with scissors, the handle of the knife, any suitable instrument, a gauze sponge, or the surgeon's finger. For the most part, sharp dissection is preferable. It effects clean exposure and creates relatively little trauma. Blunt dissection is employed as a minor complement to sharp dissection. Cleavage planes yield nicely to strokes of the blade, together with an occasional gentle push by the handle of the knife. Forceful blunt dissection is never to be condoned. It tears and contuses the tissues, opens up potential dead spaces far beyond the required field, and predisposes to hemorrhage and infection.

Control of hemorrhage is essential. A surgeon's efficiency may be judged by the dryness and neatness of his wound. It is impossible to operate effectively, and dangerous to attempt to do so, in the presence of uncontrolled bleeding. Whenever possible, vessels should be seen and accurately clamped with finely pointed hemostats before being cut. Together with the vessel, the clamp should crush little or none of the adjacent tissues. Ligation of large bites of tissue is to be de-

plored. Ligature by transfixion is recommended for all large vessels and for some small ones, when it is difficult to stabilize the knot. Transfixion also makes possible the use of more delicate suture material than would be required otherwise. Within reason, the fewer and less bulky the ligatures, the better the healing of the wound. Experience teaches which smaller vessels must be tied and which may be left to contract spontaneously and thrombose. Hematoma formation in a clean incision, besides delaying wound healing and predisposing to infection, indicates poor surgical technique.

various types of catgut, plain or chromicized; the latter, such materials as silk, cotton, stainless steel, silver, and numerous plastics. They are available in various sizes, from the extremely fine to the very coarse.

The choice of suture material is governed by the nature of the operation and the presence of contamination or infection. It is also influenced by the individual preference of the surgeon and by the facilities which are available to him.

Operations are classified as clean, contaminated, and infected. Elective hernia repairs,

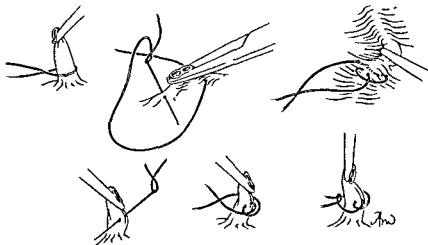


Fig. 63—Methods of vascular ligation. The first diagram represents the common manner of simple ligation. The others show various stages and types of ligation by transfixion. These last have been modified from those of Halsted which appear in his monograph on *The Use of Silk in Surgery*, published by the Welch Bibliophilic Society in 1939.

Adequate reconstruction of a surgical incision implies accurate apposition of the various fascial planes without tension, obliteration of dead spaces, and absolute hemostasis. The tensile strength of the sutures need be no greater than that of the tissues they are designed to secure. The sutures should take small bites and must not be tied so tightly as to cause necrosis of the tissues which they compress. Bulky suture material of any sort complicates wound healing. The less foreign material present in an incision, the better. Each suture, therefore, should be cut as close to its knot as is compatible with security.

Suture materials are classified as *absorbable* or *nonabsorbable*. The former group includes

mastectomies, and thyroidectomies are examples of clean operations. Interval appendectomies and cholecystectomies may be minimally contaminated. Stomach and bowel resections are contaminated. Cases dealing with acute or subacute inflammatory lesions are said to be infected.

A clean wound may be repaired with either absorbable or nonabsorbable sutures. In a contaminated case, which becomes infected, nonabsorbable sutures predispose to sinus formation, except in situations where they are rapidly sealed off as occurs in the seromuscular layer of intestinal anastomosis. Stainless steel wire is the nonabsorbable suture material least likely to cause sinus formation complicating wound

infection. However, nonabsorbable sutures of any sort must not be buried in the presence of infection.

The first significant report on the use of silk in surgery was published in 1888 by Theodor Kocher, who noted that wounds repaired with silk became infected much less frequently than those repaired with catgut. Kocher's influence and his own ambition to perfect a technique of clean wound repair led Halsted to adopt silk. His meticulous personal use of this medium and his instruction of others soon developed, at the Johns Hopkins Hospital, a school of surgery which, on this continent, remained for many years unique. George Heuer's monograph* on Halsted's life and work, which was published on the occasion of the centennial of the birth of this great teacher-surgeon, contains the comment here quoted, to inform the student of the qualities of the man whose influence on North American surgery may never be surpassed: "During his Baltimore period he was the exemplification of the slow, careful, painstaking surgeon who is concerned with the patient's safety, not with his own brilliancy. He was aware then, as most of us now are, that safe and successful surgery is the result, not of a rapid and dexterously performed operation, but of a combination of factors which includes the routine preliminary preparation of the patient, selected and well-administered anesthesia, perfect aseptic surgical technique, the gentle handling of all tissues exposed, the careful avoidance of injury to all structures not involved in the operative procedure, and the use of fine delicate instruments and suture material of fine silk in the control of hemorrhage and the closure of the wound. No one appreciated more keenly than he that the safety of the patient and the success of an operation can be jeopardized by a faulty anesthetic, by infection, by injury to an important structure, or by failure of proper wound healing."

Yet silk has never received general acclaim, despite the experimental and clinical evidence

in its favor provided by many distinguished surgeons since Halsted's time. This is not difficult to explain. Silk demands the utmost in aseptic technique and in painstaking surgical judgment and skill. The requirements of ideal wound healing must be appreciated and fulfilled with precision: minimal trauma, rigorous hemostasis, and anatomic reconstruction without tension. As Halsted remarked, "In the hand of a bad technician silk is disastrous."

While there is no doubt that, under identical conditions, the healing process in a "silk" wound exhibits considerably less inflammatory reaction than in a "catgut" wound, the gross difference is not nearly so marked when catgut of comparable delicacy is used with the same precautions against trauma, tension, and hemorrhage as those required by the use of silk.

The work of Howes and others has dissipated the illusion of catgut allergy, which was formerly blamed for the occasional disruption of wounds repaired with absorbable sutures. If the contributory factors of tissue devitalization and increased tension are present in sufficient degree, a wound will disrupt as readily through sutures of catgut, silk, or steel. It seems reasonable to conclude that a careful and well-trained surgeon, working under suitable conditions, will use silk (or other equally inert and delicate material) in clean wounds; but that, when in any doubt regarding the presence of contamination or infection, he will rely on fine catgut handled in the same manner.

WOUND EXCISION

Wound excision consists in the systematic removal of all devitalized or grossly contaminated tissue from a recently inflicted wound. It attempts to convert a contaminated and potentially infected lesion into one that is relatively clean. Depending upon the nature and site of the wound and the degree of its contamination, the time factor is somewhat elastic. Early, adequate excision of a contaminated wound is usually followed by uncomplicated healing. Wound excision following bacterial proliferation is dangerous, because it removes natural barriers to spreading infection.

*Heuer, G. W.: Bull. Johns Hopkins Hosp. (suppl.) 90, 83, 1932.

Generally speaking, it is safe to perform wound excision within 12 hours of injury. In selected cases, minimally contaminated and *presenting no sign of infection*, the safe period may be extended a few hours.

The excision of a wound is performed in layers from without inward. Dead spaces are eliminated and hemostasis secured. Adequate drainage is established. The wound is enlarged, depending upon the extent of its penetration, by suitably placed incisions, which may increase its greatest diameter four to six times. Contused skin edges are cut away with a scalpel. As little healthy skin as possible is sacrificed in this procedure. Subcutaneous fat that is flecked with foreign material, severely contused, or infiltrated with blood is then removed. Deep fascial planes, which might prevent adequate drainage, are incised, preferably parallel to their fibers, but, when necessary, transversely. The viability of traumatized muscle is determined by its ability to contract when compressed by forceps. When in doubt, it is safer to excise more than less, for ischemic muscle is an ideal medium for gas gangrene infection. In compound fractures, only those bone fragments which are completely separated from periosteum and muscle should be removed.

When practical, it is advisable to change gloves and dissection instruments after the skin layer has been excised. Some advocate voluminous saline irrigation of the wound with each succeeding step of excision. We feel that irrigation may at times be helpful, but that, in excess, it obscures the extent of contamination and may, in fact, spread potentially infected material beyond the natural confines of the wound. We prefer not to irrigate wounds that are excisable.

The excised wound is lightly filled, not packed, with dry, fluffed dressing gauze, after its walls have been covered with a single layer of finely meshed (bandage), gauze or silk, of which the interstices are small enough to prevent the inward growth of granulation tissue. This dressing is secured by an elastic bandage, and the lesion is immobilized by splinting or a plaster cast.

In excisable wounds, we feel that topical chemotherapy, applied as a thin film of finely particulate powder, is indicated. A wide-spectrum antibiotic is recommended for this purpose.

The term *débridement*, as applied to wounds, is by some considered synonymous with excision. We prefer, however, to confine its use to describe the management of wounds which are unsuitable for excision. In such cases, the lapse of time between injury and operation is commonly much longer than the safe period for excision. There is gross tissue devitalization, and suppuration is evident. Infection is established to a moderate or marked degree.

The aims of *débridement* are to relieve tension, to remove such necrotic tissue and foreign material as is readily accessible, without disrupting natural barriers to spreading infection, and to effect adequate drainage of all regions of the wound. Tension is diminished by incision of confining fascial planes. Loosely adherent slough is clipped away. Foreign bodies are removed, when this may be accomplished without extensive exploration. Dead spaces are unroofed and the defect is filled with dry, fluffed gauze dressings. Counter-drainage is established when indicated. It is doubtful that topical chemotherapy of any sort is effective in such wounds or that their irrigation with antiseptic solutions speeds the healing process. Most important is their complete immobilization, preferably in highly absorbent plaster-of-Paris casts. Infrequent dressings are definitely indicated. Each change of dressing inflicts trauma, delays healing, and increases the incidence of cross-infection. These facts were established by Trueta during the Spanish Civil War and have stood the test of time since then.

CLOSURE OF ACCIDENTAL WOUNDS

Small, superficial, cleanly incised wounds within 12 hours of infliction and following excision of grossly contaminated fat, fascia, and devitalized skin may be closed safely by *primary suture*. Larger, more deeply penetrating wounds, though recent, are better treated by

excision and closed by *delayed primary suture* after 48-72 hours or by *secondary suture* after 7-14 days or longer. In such cases the optimum time for closure occurs when the entire defect is lined by healthy granulation tissue, when no dead space remains, and when all slough has separated.

Wounds that, for the reasons mentioned, require débridement rather than excision may be closed by secondary suture after an interval of several weeks. Because of excessive loss of substance, however, they often require one or the other type of definitive plastic repair.

SUPERVISION OF WOUND HEALING

The incidence of wound healing complications is inversely proportional to the surgeon's technical ability and the efficiency of his operating team. It is obvious, therefore, that such complications should be scrupulously recorded and subjected to frequent review. By means of this information, errors in surgical judgment and technique, in sterilization procedures, etc. are detected and eliminated without delay. Infection, nutritional deficiencies, metabolic disorders, blood dyscrasias, and numerous other factors impede the normal process of repair to such an extent that the more serious complications of wound healing are, at times, inevitable. To record only such major catastrophes is of little value. The picture must be complete: either a surgical incision heals cleanly by primary union or it does not. If the slightest complication occurs, it should be noted in the progress report and discharge summary of the case.

To properly assess the quality of wound healing, the type of case—clean, contaminated, or infected—must be stated, suture materials and drainage devices noted, and healing summarized as follows: (1) healed cleanly by primary union; (2) stitch abscess; (3) hematoma; (4) trivial infection; (5) serious infection; (6) wound disruption.

An accurate statistical record of wound healing, presented as an annual report, will clearly demonstrate technical improvement or the reverse.

SURGICAL STERILIZATION

Surgical sterilization implies absolute destruction of bacteria. It is accomplished by physical or chemical processes: by immersion in boiling water for at least 10 minutes; by autoclaving at 18 pounds pressure (25 $^{\circ}$ F.) for varying periods, depending upon the article to be sterilized; by dry heat in the hot air oven, or by chemical reagents, such as ethyl alcohol (70%), oxycyanide of mercury (1:1,000), biniodide of mercury, or numerous other antiseptics.

Routine procedures are devised for the maintenance and sterilization of all types of surgical equipment. A few of the commoner methods should be mentioned. Packaged dry goods, granite ware, rubber goods (including drainage material), silk sutures, brushes, rubber glassware are autoclaved for 30 minutes, rubber gloves for 15 minutes, and instruments for 10-20 minutes. Articles that may be damaged by intense heat are immersed in appropriate antiseptic solutions for varying periods.

SCRUBBING FOR OPERATIONS

It is impossible to sterilize completely the hands of the operating team. Numerous reagents, because of their bactericidal and detergent properties, reduce bacterial flora to a minimum, but surgical sterilization is never accomplished. Systematic scrubbing for 10 minutes with a bland, superfatted soap, and brush of not too coarse texture, followed by 1 minute's immersion of the rinsed hands and forearms in a trough of mild antiseptic (aqueous Zephiran, 1:1,000) is an effective routine. Frequent scrubbing facilitates cleaning. Adjuvant detergents and antiseptics may reduce scrubbing time to a minimum of 3 minutes.

The following points concerning scrub-up must be emphasized:

1. There must not be any infection of the skin or break in skin continuity (abrasion or laceration) of the hands or forearms.
2. The nails should be clean, smooth, and pared short.
3. The scrub-up zone should include the elbows.

SCRUBBING FOR OPERATIONS

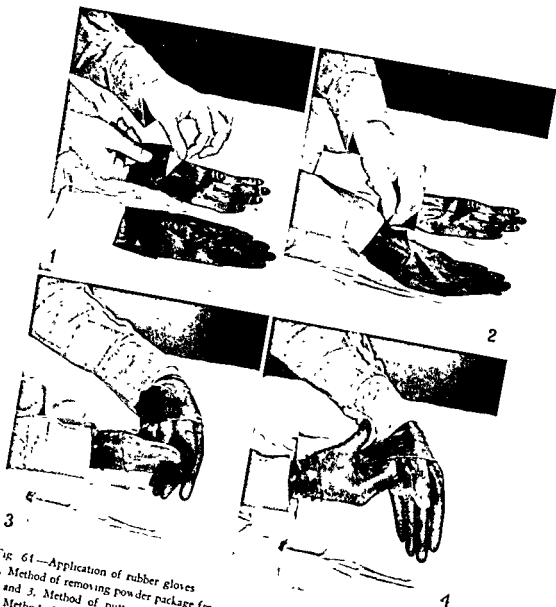


Fig. 61—Application of rubber gloves
1, Method of removing powder package from glove cuff
2 and 3, Method of putting on gloves, hand to hand and glove to glove technique.
4, Method of rolling glove cuff over cuff of gown

PREPARATION OF THE OPERATIVE FIELD

The night before operation, the area is cleansed with green soap and carefully shaved far beyond the limits of incision. In the operating room, further cleansing is carried out with alcohol and ether, and an antiseptic solution is applied in systematic strokes from the center of the field to its periphery. Contaminated zones are painted last

SURGICAL INCISIONS

An incision is designed primarily to afford the best possible exposure of the lesion in question, with minimal retraction of adjacent structures. It should, for cosmetic reasons, parallel the natural creases of the skin. Its course should be at a right angle to the minimal distracting forces in the area. It should avoid compromising major nerves and blood vessels and, when practical, divide fascial apo-



Fig. 65—Commoner breaks in aseptic technique 1, Second assistant's left arm outside sterile field 2, Inadequately draped anesthesia screen 3, First assistant improperly masked 4, Instrument nurse's hair uncovered by cap 5, First assistant reaching behind instrument nurse 6, Hustle nurse's uniform touching sterile hand basin 7, Surgeon's hand below level of sterile field 8, Surgeon's cuff outside glove 9, Second assistant's retractor outside sterile field

For orthopedic operations it is customary to supplement the above procedure by one or more similar treatments on the ward during a period of 24-48 hours prior to operation. The limb is wrapped in sterile towels following each treatment. In such cases of repeated application, it is particularly important that the antiseptic used should be nonirritating

neuroses in the direction of their fibers rather than transversely. In cases of emergency, the speed with which the incision may be opened and closed is important. The closure of the wound is often a conservative procedure, its importance may be important than its insofar

meticulous reconstruction is never wasted. Accurate approximation of serosal surfaces, fascial planes, and skin edges, with delicate sutures under minimal tension, rigorous hemostasis, and obliteration of dead spaces are basic to adequate surgery

DRAINAGE OF WOUNDS

In clean operations drainage is seldom required and should be avoided, because the drain itself creates a dead space, acts as a foreign body, and affords access to infection from without. In many cases, drainage of clean wounds indicates indolence or ineptness on the part of the surgeon, who has failed to effect hemostasis. Occasionally, when oozing is difficult to control or when removal of a large tumor leaves a dead space which may fill with serosanguineous fluid, drainage for a period of 24-48 hours is advisable.

1. Localized abscess
 2. Residual necrotic tissue
 3. Bleeding such as is controllable only by packing
 4. Operations involving the gall bladder, bile ducts, or liver parenchyma
 5. Unsatisfactory inversion of the duodenal stump
 6. Operations on the pancreas
 7. Grossly contaminated colon anastomoses
- Drainage of the abdomen in the presence of acute diffuse peritonitis is ineffectual, because whatever device is used will be walled off from the major cavity within a few hours. Though the peritoneum may be closed safely in many cases of contamination or infection, it is advisable, by suitable drainage, to anticipate involvement of the less resistant extraperitoneal areolar tissue and subcutaneous fat. Delayed primary closure of the skin is an effective safeguard when nonabsorbable sutures are used to reconstruct a potentially contaminated incision.

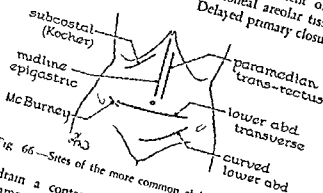


Fig. 66.—Sites of the more common abdominal incisions

Whether or not to drain a contaminated wound is decided by the amount and character of the contaminant and the natural resistance of the tissues immediately involved. When in doubt, it is safer to drain such wounds until it is apparent that no infection has taken place.

Infected wounds must be left open and drainage maintained by tamponade or tube. If precautions are taken to protect the subcutaneous fat from contact with deeply seated infected material, it is usually safe to close the angles of the wound up to the drainage tract and thereby shorten the time of healing.

Indications for drainage of abdominal incisions merit special consideration. Normal peritoneum is amazingly resistant to infection. Once inflamed, however, it rapidly loses this happy faculty. The following conditions necessitate drainage of the peritoneal cavity:

INSTRUMENTS

In many teaching hospitals, surgical interns serve for several months as "instrument nurses," becoming thoroughly familiar with the proper names and applications of the tools of their trade. Though this luxury is impractical for the student, and the consensus that details of surgical technique should not complicate undergraduate teaching is probably correct, we feel that the student should be encouraged to learn the names and purposes of instruments in common use. Figs 67-75 display instrument setups routinely associated with various operative procedures at the Royal Victoria Hospital.

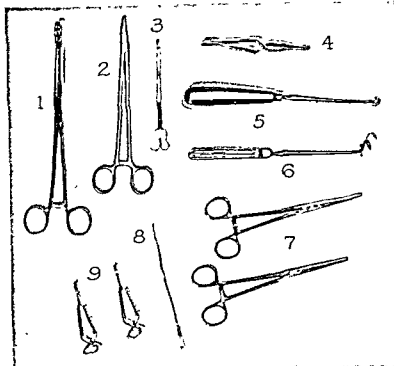


Fig 69—Instruments supplementary to basic dissection 1, Foerster sponge forceps 2, Mayo needle holder 3, Grooved director 4, Hagenbarth-Michel clip holder 5, Curette 6, MacEwen needle holder 7, Ochsner clamps 8, Probe 9, Jones towel clip

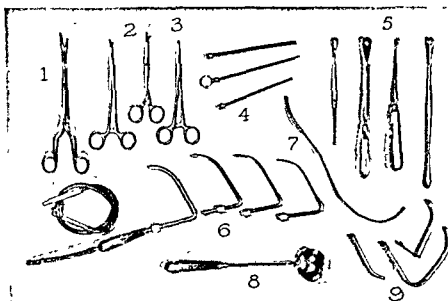


Fig 70—Gall bladder and common duct instruments 1, Common duct stone forceps 2, Lahey forceps and right angled duct forceps 3, Artery forceps (7 1/2 inch) 4, Set of suction tips with universal handle (R.V.H.) 5, Gallstone scoops and curette 6, Common duct probes and irrigators (Archibald) with universal handle (R.V.H.) 7, Common duct metal catheter 8, Moore gall bladder spoon 9, Common duct suction and T-tube inserter (R.V.H.)

SURGICAL TECHNIQUE

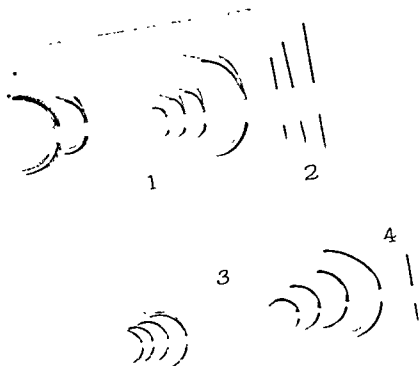


Fig 67—Needles 1, Heavy fistula and surgeon's cutting edge needles 2, Keith's abdominal needles 3, Set of Ferguson and Mayo round bodied, curved needles 4, Straight, round needles (TGH)

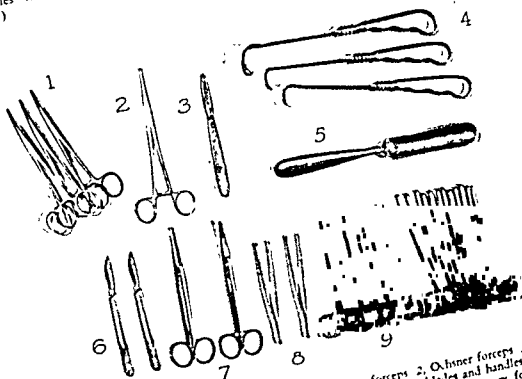


Fig 68—Basic dissection instruments 1, Curved Péan forceps 2, Kocher dissector 3, Richardson retractors 4, Bird Parker blades and handles 5, Mayo scissors, straight and curved 6, Toothed and plain tissue forceps 7, Straight artery forceps 8, Allis forceps 9, Mayo scissors

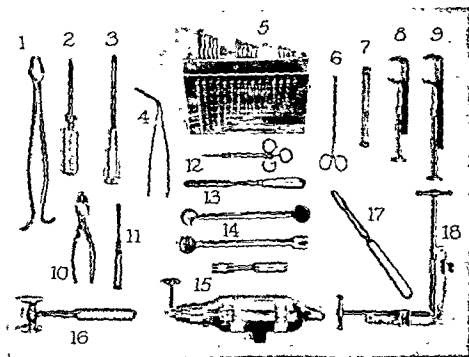


Fig 73—Fracture instruments: 1. Lane bone-holding forceps 2. Screw driver (Zimmer) 3. Screw holder and driver (Down Bros) 4. Plate-holding forceps 5. Set of Vitallium plates and screws 6. Screw holding forceps 7. Osteotome 8, 9 and 18. Set of Lewman bone holding forceps. 10. Wire-cutting forceps 11. Lane periosteal elevator 12. Depth finder (Down Bros) 13. Bone file 14. Zimmer plate bending forceps 15. Wire twister (Down Bros) and electric motor for drills and circular saws (Luck) 16. Metal mallet 17. Lane elevator and metal rule

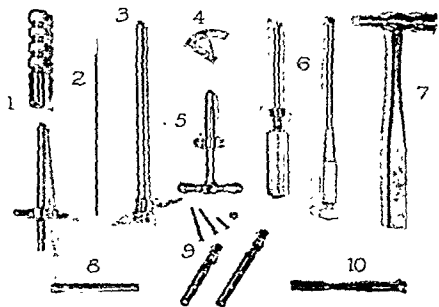


Fig 74—Hip pinning—Smith Petersen instruments 1. Nail extractor 2. Guide wire 3. Nail inserter 4. Pin extractor 5. Pin extractor 6. Screw driver and screw holder (Down Bros) 7. Metal mallet 8. Nail 9. Side plates and screws 10. Nail starter

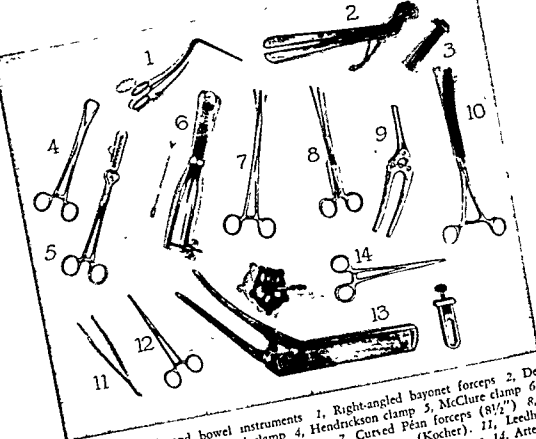


Fig 71—Stomach and bowel instruments 1, Right-angled bayonet forceps 2, De Martel clamp 3, Set of blades for De Martel clamp 4, Hendrickson clamp 5, McClure clamp 6, Furniss clamp and special needle for intestinal anastomosis 7, Curved Péan forceps (8 1/2") 8, Bayonet forceps 9, Payr's clamp 10, Rubber-covered intestinal forceps (Kocher). 11, Leedham Green forceps (7 1/2") 12, Miller-Allis forceps 13, Von Petz clamp for stomach and intestine 14, Artery forceps

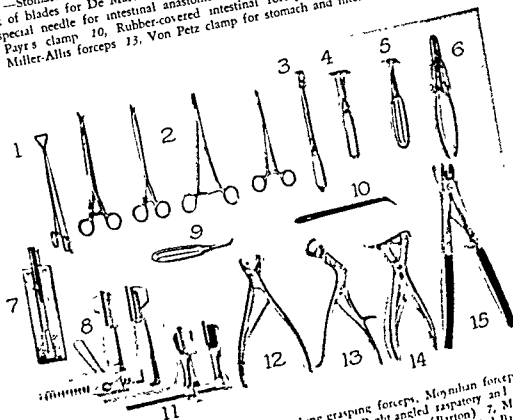


Fig 72 Chest instruments 1 Duval Crile lung grasping forceps, Moynihan forceps, Gray forceps 2 Mackenzie silver clip holder, Lahey forceps 3 Right angled raspatory and elevator 4 Doyen costal elevator 5 Bethune elevator 6 Double-action rongeur (Barton) 7 MacKenzie silver clip magazine 8 Finocchio retractor 9 Goose neck raspatory 10 Right angled Bard Parker blade handle 11 Fuller rib retractor 12 Right angled rib cutter 13 Giertz Shumaker rib shear (Stille) 14 Shumaker rib shears 15 Bethune rib cutter

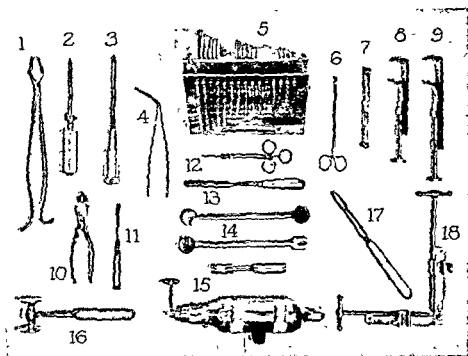


Fig. 73.—Fracture instruments. 1, Lane bone holding forceps. 2, Screw driver (Zimmer). 3, Screw holder and driver (Down Bros.). 4, Plate holding forceps. 5, Set of Vitallium plates and screws. 6, Screw holding forceps. 7, Osteotome. 8, 9, and 18, Set of Lowman bone-holding forceps. 10, Wire cutting forceps. 11, Lane periosteal elevator. 12, Depth finder (Down Bros.). 13, Bone file. 14, Zimmer plate bending forceps. 15, Wire twister (Down Bros.) and electric motor for drifts and circular saws (Luck). 16, Metal mallet. 17, Lane elevator and metal rule.

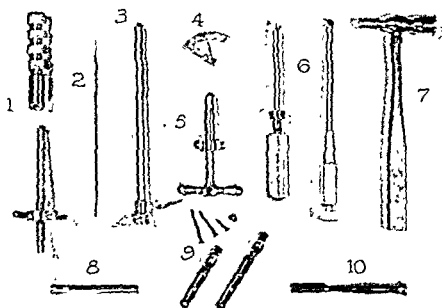


Fig. 74.—Hip pinning—Smith-Petersen instruments. 1, Nail extractor. 2, Guide wire. 3, Nail inserter. 4, Protractor. 5, Pin extractor. 6, Screw driver and screw holder (Down Bros.). 7, Metal mallet. 8, Nail. 9, Side plates and screws. 10, Nail starter.

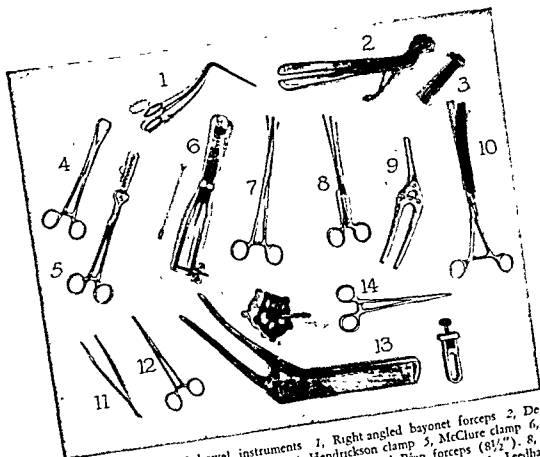


Fig 71—Stomach and bowel instruments 1, Right angled bayonet forceps 2, De Martel clamp 3, Set of blades for De Martel clamp 4, Hendrickson clamp 5, McClure clamp 6, Furniss clamp and special needle for intestinal anastomosis 7, Curved Pén forceps (8 1/2"). 8, Bayonet forceps 9, Payr's clamp 10, Rubber-covered intestinal forceps (Kocher) 11, Leedham Green forceps (7 1/2") 12, Miller-Allis forceps 13, Von Petz clamp for stomach and intestine 14, Artery forceps

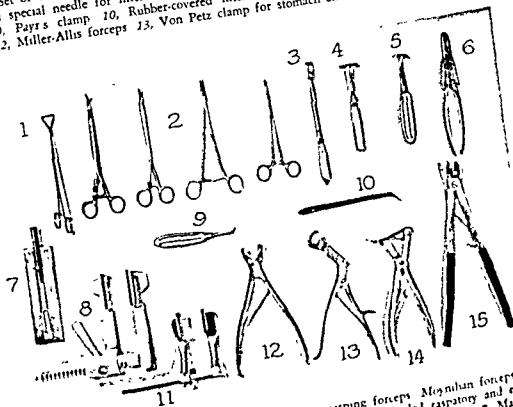


Fig 72—Chest instruments 1, Duval Crile lung grasping forceps 2, Magnan forceps, Gras forceps 3, Right angled raspatory and elevator 4, Doyen costal elevator 5, Bethune elevator 6, Double action rongeur (Barton) 7, MacKenzie silver clip magazine 8, Finocchetto retractor 9, Goose neck raspatory 10, Right angled Bard Parker blade handle 11, Tuffier rib retractor 12, Right angled rib cutter (Stille) 13, Shumaker rib shears 14, Bethune rib cutter 15, Large pair of forceps

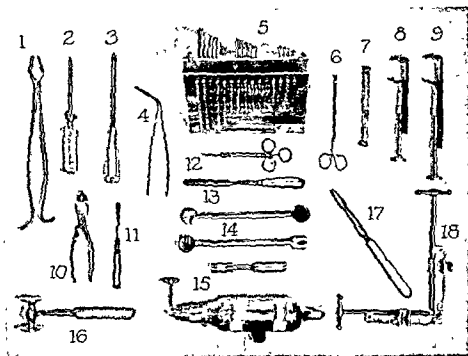


Fig. 73—Fracture instruments. 1, Lane bone-holding forceps. 2, Screw driver (Zimmer). 3, Screw holder and driver (Down Bros). 4, Plate-holding forceps. 5, Set of Vitalium plates and screws. 6, Screw holding forceps. 7, Osteotome. 8, 9, and 18, Set of Lowman bone holding forceps. 10, Wire-cutting forceps. 11, Lane periosteal elevator. 12, Depth finder (Down Bros). 13, Bone file. 14, Zimmer plate-bending forceps. 15, Wire twister (Down Bros) and electric motor for drills and circular saws (Luck). 16, Metal mallet. 17, Lane elevator and metal rule.

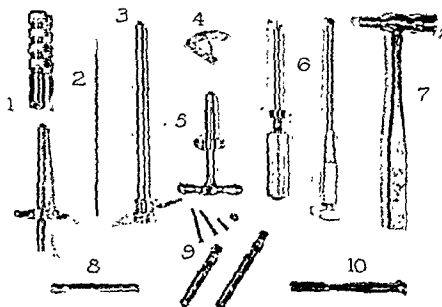


Fig. 74—Hip pinning—Smith Petersen instruments: 1, Nail extractor. 2, Guide wire. 3, Nail inserter. 4, Protractor. 5, Pin extractor. 6, Screw driver and screw holder (Down Bros). 7, Metal mallet. 8, Nail. 9, Side plates and screws. 10, Nail starter.

SUTURES

Methods of wound reconstruction are governed by many factors. The site of incision, the nature of the lesion, the nutritional state of the patient, the possibility of contamination or infection, and the requirements of drainage—all must be considered in the evolution of a suitably balanced technique.

Continuous sutures should be avoided, except in the mucosal layer of intestinal anastomosis and the closure of peritoneum or synovia. A continuous, removable, subcuticular stitch is useful in some cases. Interrupted sutures are described as plain, mattress, or figure-of-eight. Numerous additional modifications

may be made without tissue strangulation. In debilitated patients the Jones vertical, figure-of-eight interrupted suture, which includes all layers of the abdominal wall except subcutaneous fat and skin, is most effective in closing vertical paramedian incisions. When properly tied, these stitches create a smooth peritoneal surface and do not tear out. The small loop through the anterior sheath of the rectus helps to prevent excessive constriction of the larger bite of peritoneum, muscle, and fascia.

SURGICAL DRESSINGS

Clean wounds, when not drained, require no more than a few layers of plain gauze,

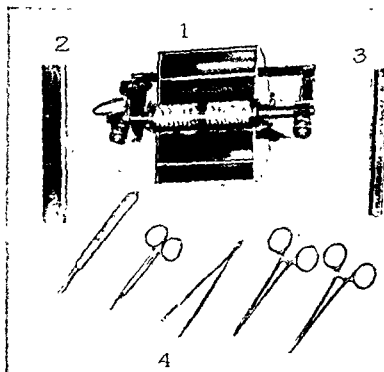


Fig 75.—Skin-grafting instruments: 1, Paget dermatome. 2, Blade for dermatome. 3, Blade holder. 4, Bard Parker blade handle, plastic scissors (Stille), Adson forceps, curved mosquito forceps, and straight mosquito forceps.

need not complicate this text. Mattress sutures more securely grip the tissues they approximate and can be placed so as to effect either eversion or inversion of the apposed edges. Plain, interrupted stitches suffice for flat or convex skin surfaces and most fascial planes. In concavities, such as the groin, vertical mattress sutures are essential to prevent inversion of the skin. The far-and-near interrupted suture, recommended by Whipple for appropriate fascial planes, accomplishes accurate approxi-

mation without tissue strangulation. In the presence of drainage, a small absorbent pad is added.

When pressure is required, to obliterate dead spaces or prevent oozing, stuffed gauze and cotton waste are spread evenly over the innermost dressing and its environment and compressed by elastic adhesive tape.

To prevent it from slipping beneath the surface of the skin, the end of a drainage tube

must be transfixed by a safety pin. If drainage is intended to be of brief duration, a thread, attached to the safety pin and brought out at the margin of the dressing, permits withdrawal of the drain without exposure of the incision.

The dressing of accidental wounds is dealt with in the section on excision and débridement.

technique is usually excellent. Adhesive tape has, to a great extent, replaced the bandage.

Bandages designed to fit conical or cylindrical surfaces or a combination of these, requiring figure-of-eight turns, are referred to as spica bandages. If the surface is cylindrical, spiral convolutions with uniform (2/3) overlapping are sufficient. If the region is conical, a spiral bandage is started at the narrower

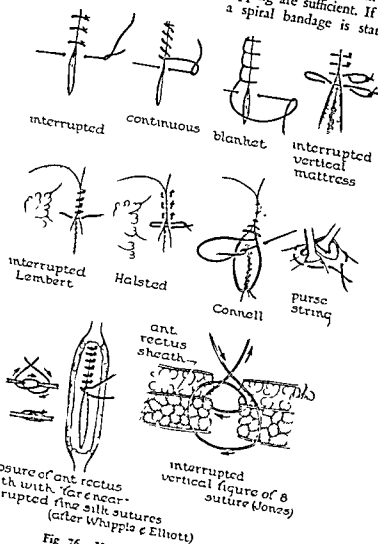


Fig 76—Various types of sutures

BANDAGES

The art of bandaging, so meticulously fostered by the French school of a century ago, is lost to many contemporary surgeons. As a rule, the student and intern receive most of their practical instruction in bandaging from nurses in charge of outpatient clinics, whose

extremity and reversed, when necessary, to accommodate the increasing diameter of the part. A spica bandage, secured distally and proximally by two circular turns, consists of a series of overlapping figure-of-eight convolutions and affords best support to dressings at the junction of one or another extremity with the trunk.

Bandages which envelop dry dressings are best maintained by spiral strips of adhesive tape. When moist dressings are prescribed, commonly in minor infections of fingers or toes, the bandage is first made fast by two or three turns at wrist or ankle, carried obliquely across the dorsum of hand or foot to secure the dressing of the affected digit, and then back again in the opposite oblique to be tied at its proximal origin.

and drained of excess fluid, is ready for use. In hospital and office practice, proprietary plaster bandages and slabs have largely replaced the homemade variety. Their neatness, delicate texture, uniform impregnation, and rapid setting are distinct advantages, which more than compensate for diminished absorptivity.

The requirements of a plaster cast are accurate coaptation, in no greater thickness than

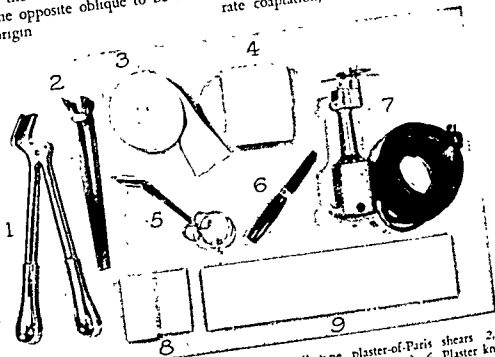


Fig 77—Plaster-of-Paris cast instruments 1, Stille type plaster-of-Paris shears 2, Cast spreader 3, Stockinet 4, Sheet wadding 5, Plaster scissors (Lister bandage) 6, Plaster knife 7, Stryker cast cutter 8, Plaster bandage 9, Plaster slab

Bandages should afford uniform, firm support to dressings. They must never be so tight as to impede circulation or exert excess pressure on bony prominences or superficial nerves. The resiliency of an elastic bandage is infinitely preferable to the rigidity of plain gauze. Inelastic adhesive strapping must never encircle any part of an extremity, nor may circular bandages of any rigid material be applied beneath plaster casts.

PLASTER CASTS

Plaster of Paris is still the medium of choice for immobilizing injured tissues. It is rubbed into strips of coarse muslin which are then rolled into bandages or folded into slabs of various lengths and widths. The bandage or slab, having been dipped in tepid water

the situation demands, and complete immobilization of the part, by inclusion of joints proximal and distal to the lesion, in a position of optimal function. When open wounds are thus immobilized, the absorptivity of the plaster is of great importance. If this quality is impaired, tissue fluid accumulates beneath the cast with consequent maceration of skin, edema of the granulating surface, and an evitable infection. The stain of serosanguineous drainage should appear on the surface of a suitably absorbent cast within a few hours of its application.

Strategic use of plaster slabs greatly diminishes the bulk of a cast without lessening its mechanical efficiency. The slab, of any required thickness, is molded to the under (weight bearing) surface of the extremity and secured by a circular bandage of which 2-4

layers generally suffice. Such a cast is easily removed by a longitudinal cut through its thinner surface and eversion of the cut edges. A thick, cumbersome, irregular cast demonstrates the inexperience of the operator. If the individual who applies a cast is also responsible for its removal, he will soon take steps to improve his technique.

When applied during the acute phase of injury, plaster casts must be carefully padded to accommodate increased swelling and avoid pressure on traumatized soft tissues. For this purpose, a few layers of sheet wadding are effective. Bony prominences, superficial nerves, and weight-bearing areas are additionally protected by pads of felt. At this stage, continual elevation lessens edema and improves circulation. If constant supervision of the case is impossible, as when battle casualties are evacuated from forward areas, it is customary to split the freshly applied cast longitudinally through its thinner surface. This is accomplished most simply when a flattened roll of paper (newsprint is satisfactory), comprising several layers about 2 cm wide, is placed for protection of the skin in the long axis of the limb opposite the slab and incorporated in the circular bandage. While the plaster is still soft, it is easily incised by a sharp blade down to the protective layer of paper. When the split is complete, its edges will spring several millimeters apart, but further separation is prevented and the function of the cast fully maintained by 2-3 circular strips of gauze bandage. If, during transportation of the patient, swelling should obstruct circulation or infection necessitate further treatment of the

wound, the properly split cast may be opened in a matter of seconds by the fingers alone.

When the acute reaction to injury has subsided, muscle atrophy and decreased swelling require that the cast be changed for one that snugly fits the contours of the shrunken limb. The new cast needs relatively little padding. In simple fractures, it is molded over stockinet plus a turn or two of sheet wadding in pressure areas. In compound injuries with persistent drainage, it is applied preferably over sheet wadding which, however, should not be interposed between the absorbent wound dressing and the cast.

The quality of a cast is directly proportional to the operator's experience and personal interest in its composition. Plaster technique, like formal sculpture, is not acquired overnight. Numerous devices in padding, smoothing, and molding greatly lessen the patient's discomfort and expedite restoration of function. Finally, it is axiomatic that when a patient complains of localized pressure beneath a cast he is always right until the affected area has been unroofed for inspection. The pressure lesions caused by ill-fitting casts are often more serious than the initial injury.

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Plastic Surgery

Hamilton Baxter, M.D.

GENERAL CONSIDERATIONS

Ancient records of the Egyptians and Indians, written many centuries ago, reveal that they had developed methods of repairing mutilated and deformed features; thus, the origin of this specialty is deeply rooted in the past. Great advances, however, have been achieved in this branch of surgery since the beginning of the 20th century. This revival of interest has been stimulated by World Wars I and II, the invention of new instruments, and recently discovered therapeutic agents.

Plastic surgery deals with the repair of congenital or acquired defects, with a view to restoration of function, improvement of appearance, and resolution of any psychologic disturbances caused by the defect. In recent years, more emphasis has been placed upon the latter aspect, because of the increasing number of handicapped persons injured by warfare and civilian accidents. A facial injury or malformation should be corrected early when possible to prevent the development of deep psychologic wounds. If only partial or no improvement can be accomplished by surgery, psychotherapy should be instituted to aid the individual to become adjusted to the deformity.

Plastic surgery extends into some aspects of general surgery and various surgical specialties. Collaboration is often required between other specialists and the plastic surgeon. In many cases the special experience of the latter in planning incisions, designing flaps,

and obtaining free grafts of various tissues contributes greatly to successful results. This is particularly true, for example, when extensive trauma of skin and subcutaneous tissues has occurred.

A basic sense of artistic imagination is required in which the various stages, in their proper sequence, of a given reconstructive problem can be visualized. The most suitable method of treatment of a specific condition must be selected, due consideration having been given to the physical condition and status of the patient. It is important to diagnose whether tissues have been lost or only misplaced and how they may be restored.

The general principles of wound healing and of the rate of healing of different tissues must be known. The type and rate of healing of a wound is dependent upon the treatment given and the presence or absence of infection. In the absence of infection, healing is firm and strong. During the first four days, termed the lag period, the tensile strength is due only to the slight adhesion of the wound margins by fibrin and to the sutures. Serum, fibrin, leukocytes, and red blood cells fill in the spaces within the wound surfaces, and this initiates the first stage of wound healing. Proliferation of epithelium and connective tissue cells begins early. Subsequently, regeneration of capillaries penetrates the exudate and there is active fibroblastic proliferation. This reparative tissue is termed granulation tissue. After four days, in the absence of infection, the healing of the

wound, indicated by its tensile strength, increases rapidly, reaching a maximum on the 12th-14th day. This is due to proliferation of fibroblasts. Later, the young connective tissue cells shrink, become spindle shaped, and the intercellular collagen fibers contract. Gentle handling of tissues, employing sharp knives, scissors, skin hooks, and atraumatic needles with finest suture materials will aid healing. The blood supply of flaps or tissues being operated upon should be carefully preserved, or the best-planned operation will fail. Positive hemostasis should be obtained by fine ligatures since the use of pressure alone or hot towels may permit hematoma formation which may mar what might have been an excellent result. The type of dressing and amount of pressure applied vary with different procedures and should be considered as the last stage of the operation. The dressing should immobilize the tissues, approximate raw surfaces, minimize edema and oozing of blood or plasma, but should not interfere with adequate blood supply to the area. When elective plastic operations are being performed, no local pustules or general infective process should be present.

TISSUE GRAFTS

Skin Graft.—A free skin graft may be defined as a portion of skin which has been completely severed from its position and has been transferred to another area of the body. The technique of skin grafting has been known for centuries, and it was not an uncommon procedure for gluteal skin to be grafted in order to restore nasal defects. Through the years, surgeons such as Reverdin, Thiersch, Wolfe, Blair and others have described various types of skin grafts and methods of obtaining them. The methods of obtaining split-skin grafts were revolutionized by Padgett in 1939 when he introduced the dermatome. Three main types of skin grafts are used.

Full-Thickness Grafts.—These are dissected from an area of the body which provides skin of suitable color, thickness, and hair distribution to that of the recipient area. This type of graft possesses the advantages of having a good cosmetic appearance, contracting little, and providing a good weight-bearing and

friction-tolerating surface. It is now most frequently used about the face, hands, and weight-bearing areas of the body. The most obvious disadvantages are that these grafts will not "take" on infected areas; cannot be obtained in large quantities, and require extremely careful dressing.

Technique—A pattern of the defect to be covered is made with sterile polyethylene, celluloid, or thin lead sheet. This is then placed on the donor area and the outline traced with a scalpel. One margin of the graft is then raised and, using a small cylindrical object as a roller, the graft is held on tension, which facilitates removal, and is gradually dissected from its bed at the junction of the dermis and subcutaneous fat, none of which should remain on the graft. The donor site is then closed by undermining and suturing the margins of the wound, or, if this is not feasible, by the application of a split skin graft.

Dermatome Skin Graft.—This type of graft may be taken by any of the commonly used types of dermatomes now on the market which permit the cutting of a graft in accurately calibrated thicknesses from thin to almost full-thickness grafts. The *thin* grafts vary from 0.010-0.014 inch in thickness and are particularly suitable for grafting in children where the skin is relatively thin, and in granulating wounds where the take of a thick graft would be doubtful due to the paucity of blood supply or heavy contamination with bacteria. On the other hand, the *thick* dermatome grafts, which usually vary from 0.018-0.030 inch in thickness, provide, in considerable measure, the advantages of the full-thickness graft, without the necessity for covering or closing the large defect which results from the removal of a full-thickness graft.

Technique—When the dermatome has been set to the required thickness of the graft to be cut, the drum of the dermatome and the skin of the donor area are painted with rubber cement which is allowed to dry until the initial gloss of the cement has disappeared. Padgett dermatome cement may be applied and the dermatome drum painted with Plastikrim. The cement adheres to the drum when the skin graft is removed and thus facilitates handling and suturing of the graft. The leading portion

Scarpa's Tumor of the Scapula of the Forearm

Fig. 7

Fig. 8



Fig. 9



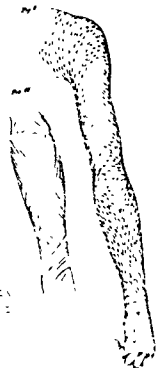
Fig. 10



Fig. 11



Fig. 12



Scarpa's Tumor of the Scapula of the Forearm

Fig. 13

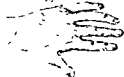


Fig. 14



Fig. 15



Fig. 16



Fig. 17



FIG. 78.—(See opposite page for legend)

of the drum is then pressed firmly against the donor area, and when the adherence between the two is firm, the drum is rotated upward slightly, and the knife handle is then slid backward and forward until the desired amount of skin is removed. At the end of the drum the skin may be severed with scissors or by pulling the drum away from the skin and cutting through with the dermatome blade. The common reasons for unsuccessful operations of the dermatome are a dull knife,

dressing is changed, any necrotic tissue or exuberant granulations may be scraped away before applying the next dressing. Exudates should be washed away with saline or mild antiseptic solutions. When known pathogens are present, with an increased amount of purulent exudate, they may be controlled by the application of dressings, soaked in a solution of some effective antibiotic for a day or two before the graft is applied. When the skin graft is applied to the raw area, it may be



Fig 78 (cont d) — Langer's conception of the lines of normal skin tension as determined by multiple puncture wounds with a round pointed metal awl in the skin of cadavers

cement that is too thick or too thin, and moisture on the drum or on the skin. In emaciated patients saline may be injected into concavities, such as between the ribs, to aid in obtaining a complete drum of skin.

Granulating areas may be prepared for skin grafting by covering the region with Xeroform or petrolatum gauze and a firm pressure dressing which is changed at intervals of several days for a short period. When the

laid on healthy granulations, or, if these are not healthy, they may be shaved off with a scalpel or an electrodermatome, carefully, so that the tendons and other important structures are not exposed. The grafts are sutured in place with fine Dermalon and are dressed with Xeroform fine-mesh gauze, several layers of dressings, followed by cotton waste and rubber sponge. The whole dressing is firmly secured with even, firm pressure by band-

and adhesive. Nearby joints should be immobilized by a padded splint.

Failure of skin grafts to live may be due to a variety of causes, both systemic and local. In the first category it may be noted that anemia is most detrimental to successful growth. It has been stated that a hemoglobin below 65% markedly decreases the chance of take of a skin graft. It is good practice not to perform an extensive skin graft if the hemoglobin is below 80%. In patients with severe burns, an effort is made to raise the hemoglobin to at least 85% by transfusions before operating. Other general causes are a

until a later date when the operative procedure may be completed with safety.

Autografts (self) are those taken from and applied to the same person and survive permanently. Grafts between identical twins may be included in this category since they are monozygotic. *Homografts* (same) are those between individuals other than identical twins. They survive for a few weeks and then slough. Homografts are useful as a temporary cover for extensively burned patients while autografts are being applied in patchwork fashion between the homografts to obtain the maximum marginal growth. It may be necessary to obtain

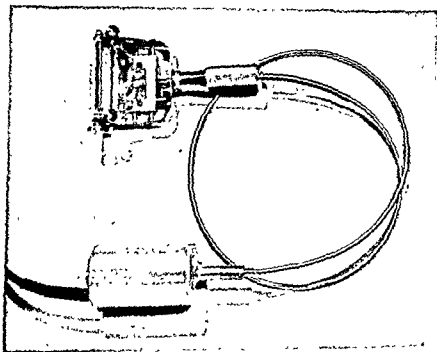


Fig. 79.—Dermatome. The electrodermatome may be used to obtain long skin grafts from any part of the body and may also be employed to remove burn eschar or exuberant granulations.

high postoperative fever, hypoproteïnemia, and hypovitaminosis. The most important local factors interfering with successful healing are an improper preparation of the granulating bed, the presence of virulent pathogenic organisms, insufficient or uneven pressure dressings, and inadequate fixation of joints.

Skin grafts folded with the raw surface approximated and wrapped in moist gauze may be refrigerated at or slightly above 0° C. for several weeks and then be transplanted successfully. If for some reason the patient's condition deteriorates, the operation may be terminated, and the remaining skin grafts preserved

several crops from the same donor area to eke out the supply of autografts. *Heterografts* (other) are portions of living tissue taken from one animal and grafted onto another of a different species. A *zoograft* is a heterograft in which the recipient is a human being; this is no longer used. The term *isograft* should not be used synonymously with *homograft* since *iso-* means equal.

Small Island Grafts.—Large dermatome skin grafts occasionally fail either completely or partially when they are placed on areas which are grossly infected, where the blood supply is inadequate, or in cavities with over-

hanging walls. Successful regrafting may be accomplished by cutting a thin, split dermatome skin graft into small pieces $\frac{1}{4}$ "- $\frac{1}{2}$ " square, and applying these, slightly separated from each other, upon the remaining surfaces of granulation tissue. Wide-mesh Jelonet gauze is secured firmly over the skin grafts, which are dressed every 4 hours with fluffy gauze moistened with physiologic saline, until firm healing has occurred.

Corneal Homografts.—These grafts can be performed successfully, possibly because this tissue is nourished only by lymph. Technical success in many cases was often reduced by clouding of the graft which gradually became opaque because of vascular ingrowth. The local application of aqueous suspension of cortisone to the eye postoperatively appears to be useful in reducing the incidence of cloudy grafts.

Mucous Membrane Grafts.—These grafts may be removed from the lip or cheek and used to restore conjunctival lining of the eye or vermillion border of the lip. Skin grafts when used to replace conjunctiva produce an objectionable discharge.

Dermis.—Dermis consists of the deeper elements of skin, the epithelium having been removed either with a dermatome or a razor. It may be inserted beneath the skin to fill depressions, it shrinks less than fat. Sheets of dermis have been used to reinforce large hernial defects. Thick dermatome skin grafts may be resplit on the dermatome by setting the blade at half the previous thickness and removing the deeper layer of the graft in a single sheet, thus doubling the surface area of the graft. The dermis takes well and regenerates a new epithelium rapidly. The surface is paler than that of an ordinary skin graft because few pigment cells are present.

Fat.—Fat may be used to fill depressions, particularly those about the face. Rigid asepsis and atraumatic technique are essential in obtaining the graft. Shrinkage after grafting amounts to about 40%, so that a defect should be overcorrected. It is less extensive if large pieces are grafted. About half the cells die, but those which survive form the fat graft that remains. Abdominal fat grafts will increase in size, if the patient deposits additional abdominal fat. Fat may be combined with

dermis or fascia, which provides a firmer tissue with less tendency to shrink, due either to degeneration or absorption.

Fascia.—Fascia is most frequently used to form slings which are employed to support the drooping cheek and mouth following facial paralysis or to raise the eyelid in ptosis. Fascia is also used in arthroplasty, to replace tendons, or to prevent adhesions between tendons and adjacent structures. It is abundantly available from the fascia lata of the thigh, from which it may be removed, by direct exposure or through a small incision with a fascial stripper. Autogenous grafts are preferable since it has been shown that the cells remain viable after transfer in human beings.

Muscle.—Muscle is rarely used as free grafts, but flaps of masseter or temporal muscle with blood and nerve supply preserved are employed to provide correction of facial paralysis.

Artery and Vein Grafts.—These grafts are discussed in Chapter 33, *Peripheral Vascular Diseases*.

Cartilage.—Cartilage has many uses as a free graft, chiefly to fill in bony depressions about the face. It has many advantages since it does not shrink, may be carved easily, and provides a resilient support for soft tissue. Cartilage is probably most frequently used in the reconstruction of the saddle nose. It should be noted that in children, cartilage grafts must be repeated, since the graft does not participate in the general growth of the area. Most surgeons prefer to use fresh autotransplants, the cells of which survive and do not absorb. However, either fresh or preserved homografts may be used, although marked absorption usually occurs in time. Cadaver cartilage may be sterilized by placing it in a glass jar containing physiologic saline solution and exposing it to 4,000,000 r from a powerful cobalt⁶⁰ source. The cartilage is then stored at room temperature until required.

Tendon Grafts.—These grafts are almost exclusively employed to replace missing or damaged tendons. The palmaris longus tendon or the long extensor tendons of the toes, removed with paratenon, are those usually chosen for tendon grafting in the hand. Tendon grafts have been advocated instead of fascial slings, in facial paralysis, in the belief that fewer ad-

hesions which limit motion would form about a tendon with its paratenon. They may also be inserted as a pad in retruded upper lips or other parts of the face following congenital or traumatic deformities, to restore normal contour.

bone grafts appear to survive. However, in dense cortical bone grafts where the cells die because of lack of nutrition, the transplant is replaced by ingrowth of cells from the host bone and periosteum. Autografts are preferable in plastic surgery, since healing is more rapid,

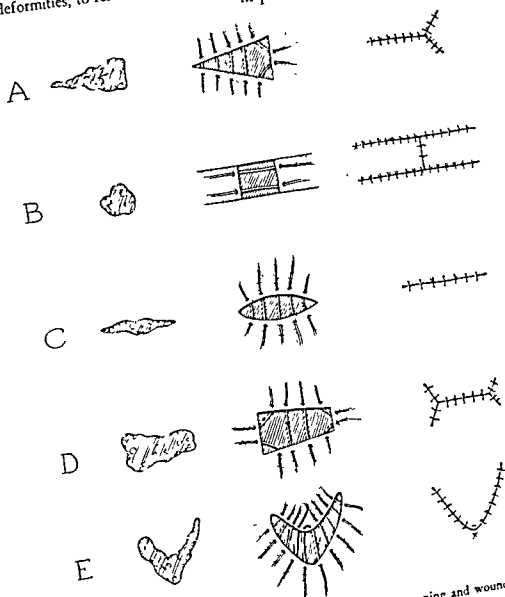


Fig. 80.—Various methods for excising defects with free undermining and wound closure

Bone Grafts.—Bone grafts are indicated when a rigid supporting tissue is necessary and are used to repair mandibular, cranial, and sometimes malar and nasal defects. Pedicle grafts of bone and osteoperiosteal grafts are rarely used. Functional use in contact with living bone is essential for retention of the calcified matrix of autogenous rib, tibial, and iliac bone grafts. Most of the cells in cancellous

although bone bank material may be used if circumstances contraindicate the use of autogenous bone. The usual source is the crest of the ilium, but the ribs, tibia, or fibula may be used. Cancellous bone chips from the ilium have been employed successfully in forming molded bone grafts for reconstruction of contour bone defects of the face. Two advantages of these grafts are that they can survive and

consolidate even in the presence of mild infection and tedious carving is not required.

Nerve Grafts.—These grafts are occasionally required following loss of substance of the facial nerve or nerves in the hand. Autografts are required and have been described by Bunnell, Ballance, and Duel.

Composite Graft.—First described by Koenig, these grafts are obtained from the helix of the ear and consist of two skin-covered surfaces and intervening cartilage. These may be used to repair defects of the ala, tip, and columella of the nose, with excellent results.

if rigid support is required, a strip of cartilage must be added later.

Skin Defects and Pedicle Flaps.—Removal of a scar, area of granulation tissue, or a tumor may be accomplished by elliptical excision and immediate closure of the defect by suture. If the wound is not large, this may be accomplished by undermining the adjacent skin and approximating the skin edges by suture. The resulting straight scar is most inconspicuous, particularly if it is parallel to Langer's lines of skin tension. Larger defects may be eliminated by converting the defect into a

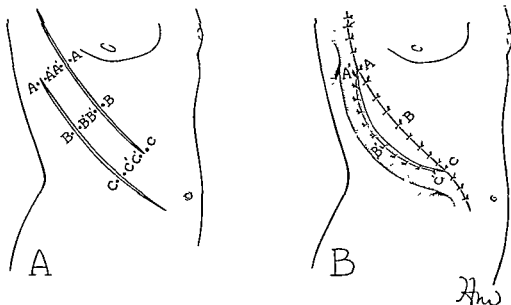


Fig. 81—Method of formation of tubed pedicle flap

A, Outline of staggered skin incisions to facilitate closure of the wound at each end of the tube. A number of points are marked opposite each other on the flaps to indicate the correct site for insertion of sutures to form the tube.

B, Following free undermining, the wound left by formation of the pedicle is closed by sliding flaps of skin beneath the tube and suturing securely. If the defect is too wide, it should be covered with a split skin graft.

Full-thickness loss of the eyelid has also been replaced with this type of two surface graft with a cartilaginous support to maintain its shape. The exact curvature desired may be obtained by selecting an appropriate site on the helix. The defect in the helix may be repaired by attaching a skin flap from the postauricular region which is severed at its base about two weeks later, folded, and sutured to the posterior edge of the defect. A full-thickness section of the lobule of the ear may be grafted in the same way to restore nasal defects, but

square, triangular, or other geometric shape; then by undermining and freely shifting local skin flaps, the deficiency may be overcome. Those procedures, which are least complicated as to rotation of tissue and in which the length of the scar is minimal, are usually the most satisfactory.

A skin flap may be defined as a given amount of skin and subcutaneous tissue which is attached to the body at some part of its periphery, through which it receives its blood supply. This flap may be rotated from its

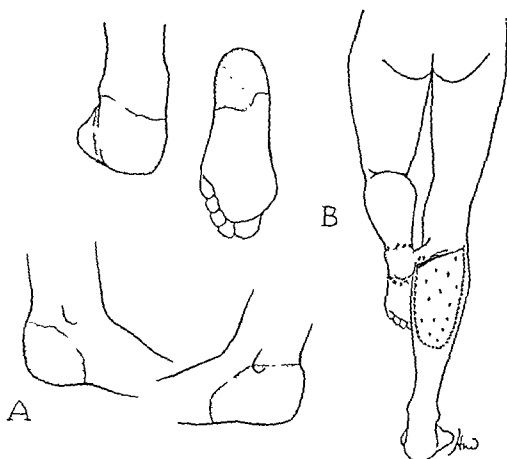


Fig 82.—A, Avulsion of soft tissues of heel when foot was run over by train

B, Cross leg pedicle flap after being delayed was swung over into defect on foot

C and D, The result one year postoperatively, showing flap. It is still protected by sponge rubber pad in shoe

C

D.

original site to fill an adjacent defect and is called a *simple* or *open flap*. The blood supply to a flap may be increased by raising part and then suturing it back in its bed. This is called a *delayed flap*.

A *tubed flap* eliminates raw areas and chronic infection present in open flaps. The desired width and length of the flap having been determined, parallel incisions are made through skin and fat, and the strip of skin is undermined. Points opposite each other on the flap, previously marked with dye, are sutured with the skin surface outward. The lateral skin edges are undermined and closed with sutures. If the

applying a skin graft to the raw undersurface of a flap. These are useful in repairing loss of two surface structures such as the cheek, lip, nose, or eyelid.

An *island flap* consists of an area of skin and subcutaneous tissue attached and nourished only by blood vessels and a small amount of subcutaneous tissue. This flap is often used to transplant an island of scalp to reconstruct an eyebrow.

Z-Plasty.—Contraction of scar tissue may cause limitation of motion of joints; in many cases a heavy ropelike cord of scar tissue forms in the line of tension of scar over the area

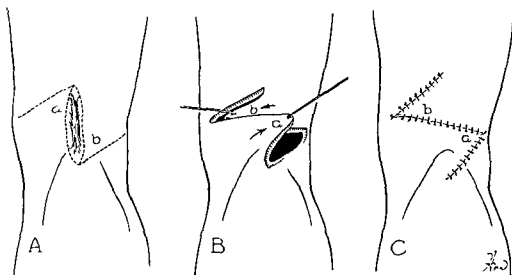


Fig. 83.—Use of Z-plasty to relieve cicatricial contracture

A, The Z incision with angles of about 60 degrees to obtain optimum increase in length

B, The flaps transposed after free undermining

C, Skin closure with correction of the linear contracture

tubed flap is too wide to permit closure of the raw wound by undermining, the raw area is covered with a skin graft. These flaps which form solid tubes of skin and fat attached at each end to the body may be transferred rapidly from one part to any other part or extremity, by attaching one end to the wrist or arm as a carrier. When the blood supply from the arm is adequate, the opposite end of the tube is severed and the whole flap is swung to the recipient site and sutured in place.

A *lined flap* is formed by folding together the raw surfaces of one end of the flap or by

affected. The heavy scar may be partially excised and by suitably placed Z-shaped incisions with transposition of the two flaps, the bow-string tension may be overcome. The area adjacent to the flaps should be extensively undermined. This procedure causes an increase in length of the contracted scar. Mathematically it has been shown that the most practical angle for outlining the two flaps in Z-plasty is 60 degrees. The percentage increase in length of the contracture is 75%. In the case of long bridle scars, double or triple "Z" incisions may be made in series with still greater relaxation

resulting than that obtained by a single incision. When broad scar bands have formed, for example, in chest and arm adhesions following severe burns, the shifting of local tissue flaps may be combined with free skin grafts.

Hypertrophic scar.—A hypertrophic scar is one in which there is excessive formation of fibrous tissue and is often seen following deep second and third degree burns or infected wounds. Many of these scars subside spontaneously in time, or their departure may be hastened by a course of x-radiation.

ischial, unilateral or bilateral. The incidence of pressure sores in civilian patients treated in paraplegic therapy centers is much lower.

Etiology

The development of decubitus ulcers in spinal paraplegics is due to intrinsic and extrinsic factors.

Intrinsic Factors.—The most important intrinsic defect is the lowering of tissue resistance to pressure which is pronounced during the



Fig 84--Multiple decubiti involving the sacral, ischial, and trochanteric regions. Necrosis of deeper tissues has resulted in extensive undermining of skin edges, with formation of sinuses and bursae.

Keloid.—A keloid may be easily recognized since the growth of fibrous tissue extends beyond the original limits of the wound and sometimes occurs following minimal trauma. Excision and skin grafting followed by early irradiation appear to be the most effective treatment.

DECUBITUS ULCERS

As a result of World War II, the numbers of paraplegic patients were greatly increased. Many of these developed large bed sores, which seriously complicated their condition. The majority of these individuals developed multiple ulcers, and the combinations most frequently seen were sacral, trochanteric, and

ischial. This is caused by interruption of spinal vasomotor control and flaccid paraplegia, not only of the skeletal muscles but also of the muscular wall of internal organs and vessels. Thus, paralytic vasodilatation is produced in these areas. The second most important intrinsic factor is sensory loss in the paralyzed regions which prevents the paraplegic from feeling the sensations of numbness or pain normally arising from an area exposed to undue pressure and consequent reduction in blood circulation. The stimulus to request a change of posture is lacking. The third factor is the thickness of fat and muscles between the bony prominences and skin of certain weight-bearing areas of the body. Other

intrinsic factors are spasticity of the paralyzed limbs, nutritional deficiency, anemia, and infection

Extrinsic Factors.—Exposure of the skin to moisture from excreta tends to cause maceration and should be avoided. The other factor of cardinal importance is pressure, which leads to ischemia and represents the immediate cause of development of ulcers. Decubitus ulcers occur over bony prominences, where the padding is thinnest and the pressure is greatest. The effect of shearing stress is more injurious than vertical pressure, since the former decreases the blood supply of larger areas

Pathology

The degree of ischemia, virulence, and direction of extension of infection along fascial planes distinguishes various stages in the development of decubitus ulcers. Transient circulatory damage has occurred when pressure has caused erythema of the skin with some edema but without death of the tissues. Relief of pressure and gentle massage will cause these symptoms to disappear. When permanent damage has developed, the epithelium may be separated from the dermis by exudation of plasma, and if the pressure continues, necrosis of the deeper layers of the skin will follow with formation of a black area of full-thickness skin destruction. Deep penetrating necrosis may involve fascia, muscle, and bone with undermining of skin edges, ramifying sinuses, infection of joint spaces, and osteomyelitis. The sores always become infected with mixed organisms, including staphylococci, *E. coli*, protei, and *Pseudomonas aeruginosa*. Sepsis and death most commonly result from infection of the urinary tract or from the decubitus ulcers

Treatment

General

The local treatment of pressure or bed sores in paraplegics must be correlated throughout all stages of treatment with maintenance of the optimum general physical condition. A high calorie, protein, and vitamin diet will prevent nutritional deficiency and help to maintain a positive nitrogen balance. Anemia may be treated by blood transfusions at regular inter-

vals. Daily administration of 25-50 mg of testosterone has been reported to accelerate healing of the sores

Local

During the period of spinal shock which occurs immediately after paraplegia has developed following trauma, the patient is most susceptible to the formation of sores. Prevention rather than treatment is the objective, and the cardinal method of prophylaxis is change of posture at least every 2 hours *day and night* to redistribute pressure. Later on, every 4-6 hours is sufficient. Oscillating beds and foam rubber or intermittent pressure mattresses are useful mechanical aids. Hygiene of the skin is important, and it should be kept dry and free from contamination by feces or urine. Once sores have developed, the principle of frequent change of position is enforced even more vigilantly. When necrotic tissue has demarcated, the slough should be excised to permit drainage of infected material and reduce toxic absorption. The wound is dressed daily with gauze moistened in an appropriate antibiotic solution or mild antiseptic and secured by Elastoplast. When granulations have formed, skin grafting will complete healing of the ulcer. Intractable spasticity of the legs not only delays healing and creates new sores, but may render plastic repair of an ulcer difficult if not impossible. This troublesome condition may be eliminated by intrathecal alcohol injections.

Plastic Repair.—In instances where very extensive or recurrent sores persist, rotation flaps or other reconstructive procedures may be required. Prior to operation it is essential that the general condition of the patient should be improved as much as possible and that renal infection and diarrhea must be overcome

Sacral Ulcers —

1 *Direct closure:* Excise all scar tissues and any bony prominences. Undermine and close without tension.

2 *Single lateral rotation flap:* A flap is rotated into the defect after removal of all sinuses and prominent bone. The secondary defect, caused by shifting the flap, is skin grafted.

3 *Double flaps:* This is a useful method, especially if the flaps from the buttocks are based inferiorly

4. Skin grafts are used only as a temporary cover, since they tend to break down.

Ischial Ulcers.—

Excision and direct closure: All scar tracts must be excised together with the gluteal bursa. The ischial tuberosity is removed if involved. Resection of the ischium is reserved for thin patients with a recurrent ulcer.

Trochanteric Ulcers.—

Following wide removal of the ulcer, bursa, and bony prominence, a rotation flap of skin based anteriorly on the thigh is swung over the defect. The secondary defect is skin grafted

Postoperative Care

Most sores are on the posterior aspect of the trunk, and the patient may be conveniently

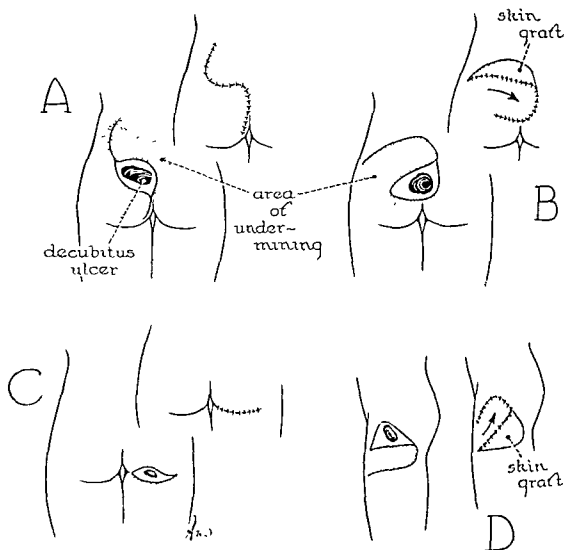


Fig. 85—A, Sacral decubitus ulcer showing tissue excised, undermining, and closure by two rotation flaps

B, Sacral decubitus ulcer showing a preliminary delaying procedure of a single pedicle flap, which was later rotated into the defect created by excision of the undermined ulcer and bony prominences. A thick split skin graft was used to close the defect created by rotation of the flap

C, Ischial decubitus ulcer deeply undermined and involving the tuberosity. Repair by complete excision of the undermined ulcer and tuberosity and suture of muscles and skin in several layers with stainless steel wire

D, Trochanteric decubitus ulcer with exposure of the greater trochanter. The trochanter is removed and the adjacent muscles swung into the defect

nursed in the prone position. The bowels are prepared with enemas preoperatively and then not opened, if possible, for a week. Blood transfusions are usually necessary to counteract surgical shock. If a hematoma forms beneath a flap in spite of insertion of a drain, it should be evacuated immediately. Flexion of the hips is not allowed for 3 weeks after healing of ischial and trochanteric sores. Finally, the patients should receive training in a rehabilitation center, where they will be instructed about the danger of pressure and how to avoid it.

CONGENITAL AND ACQUIRED DEFECTS

Some children have the misfortune to be born with congenital defects of various types. Cleft lip and palate will be discussed in a later section.

A rather common congenital defect in children is *lop ears*, quickly noted by playmates who ridicule the victim until an inferiority complex develops. Fortunately, this condition can be corrected by a simple operation which

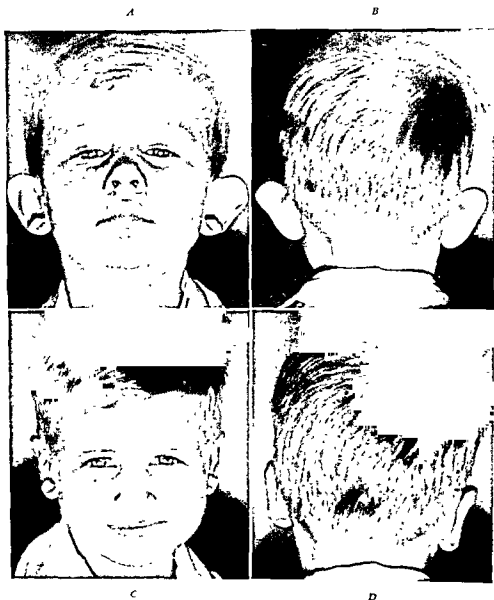


Fig. 86 - A and B Protruding ears which are large, show no antihelix, and stand out at right angles to the head

C and D Postoperative result achieved by the operative procedure described. Note the reconstruction of the missing antihelix.



FIG. 87—*A*, Marked saddle nose following childhood infection of septum.
B, Reconstruction with cartilage graft



FIG. 88—*A*, Long hump nose with a drooping tip
B, Six months after operation. The hump was removed, septum was shortened, and the alar cartilages were reduced in size

may be performed under local anesthesia. An ellipse of skin is excised with its long axis at the cephaloauricular junction, and a small ellipse of cartilage is also removed where the new antihelix is to be formed. The cut edges of the cartilage are infolded with sutures, and following closure of the skin incision a pressure dressing is applied. Excellent cosmetic results may be obtained as well as relief from the inferiority complex.

Congenital or acquired deformities of the nose may interfere with function, as well as create an unesthetic appearance. Obstruction to respiration should be corrected before or, if feasible, at the time of plastic reconstruction of the nose. Local anesthesia is the anesthetic of choice for rhinoplasty. Following the usual skin preparation and draping, incisions are made between the alar and upper lateral cartilage on each side and extended medially to



Fig 89

Fig 89—Rhinophyma increasing in size steadily, causing patient to stay at home during the day to avoid embarrassment



Fig 90

Fig 90—Postoperative result after raising skin flaps, excising hypertrophied sebaceous glands and resuturing flaps after trimming away excess skin

Carved autogenous cartilage transplants and cancellous bone from the ilium are most satisfactory materials for reconstruction of a *saddle nose*. The graft is carved to the proper size and is inserted through an incision in the tip of the nose. Grafts of bone should be brought in contact with bone by sliding the upper end under the periosteum, otherwise they tend to be absorbed. If tip support is required, a separate strut of bone or cartilage is inserted in the columella and attached to the piece of graft restoring the bridge. After closing the incision, a nasal splint is then applied to control swelling. One disadvantage of the use of bone grafts in the nose is the subsequent lack of normal resiliency of the nasal tip and the possibility of fracture of the graft

separate the columella from the tip of the septum. The skin is now separated over the rest of the nose. The hump is removed with a saw at a predetermined level, depending on the individual case, and the upper lateral cartilages are trimmed to the same level. The nasal bones are separated with a saw from the septum. At this stage the septum should be shortened and the alar cartilages reduced in size if necessary. The nasal bones are now cut on each side, at their junction with the maxillary bones, and fractured. The columella is sutured to the tip of the septum with dermal sutures, and a nasal splint is applied. This is changed when necessary and discarded in a week or two.

Rhinophyma begins with acne rosacea and terminates with tremendous hypertrophy of the

sebaceous glands, often completely obliterating the normal contour of the nose. Several methods of treatment have been suggested.

1. Flaps of skin on the nose may be raised, the hypertrophied glands excised completely, and the flaps resutured after trimming away any excess skin.

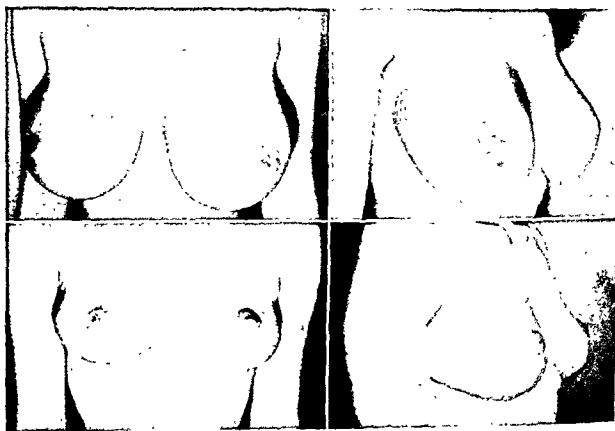
2. Complete excision of affected tissue and application of a free skin graft.

3. Finally the growth may be pared down to the normal contour of the nose without removing all regenerative elements.

hypertrophic, sometimes request a plastic operation. The breasts should be free from evidence of malignancy, no serious systemic disease should be present, and there should be no history or evidence of psychosis. Many operations have been devised, and some are suited to particular types of cases. Massive hypertrophy of the breast is easily treated by reflecting skin flaps after making an inverted "T" incision, amputating all excess breast tissue, and fashioning the remaining substance into a cone shape by interrupted sutures. The skin flaps are ap-

A

B



C

D

Fig 91—A and B, Marked fatty hypertrophy of breast, causing backache because of weight and difficulty in obtaining attractively fitting clothes

C and D, Result one year after operation, with relief of symptoms. Note cone shaped breast contour

Re-epithelization from remaining sebaceous glands occurs in about 10 days. Each method possesses certain advantages, and all factors should be considered in selection of treatment for each case.

Mammoplasty.—Individuals with pendulous breasts, either flabby and atrophic or markedly

proximated to form a tight skin brassière which supports the breast. Excess skin is excised and the wounds are closed with fine sutures. The nipples which have been excised are replaced at esthetically suitable levels, which were marked before operation. A circular patch of epithelium is excised with a sharp scalpel, and

each nipple is sutured carefully to the dermis. A firm supportive dressing is applied. A second type of operation commonly used in smaller breasts is removal of the required amount of the lateral part of the breast by an S-shaped incision. The lower free end is rotated upward and medially and sutured to the upper end of the incision in the gland, thus re-establishing the cone shape of the breast. Each nipple is transposed to a predetermined new level and brought through a circular hole in the skin which is the same size as the nipple. After replacing the nipple, draping, and suturing a tight brassière formed from the skin flaps, a pressure dressing is applied. In each case it is

Baggy skin in the eyelids may be removed by incisions placed in wrinkle lines in the upper lid and 1 mm below the eyelashes in the lower lid. The incision for the face and neck starts in the hairline above the ear, curves in behind the tragus, emerges and crosses the lobule, and extends up behind the ear to the hairline. The skin should be undermined close to the eye, nostril, and mouth and down into the neck. The skin envelope is drawn up tightly and all excess is excised. Subdermal sutures, as well as skin sutures, are used to close the wound. A pressure dressing is applied for a week, and the face should be supported at night for a month.

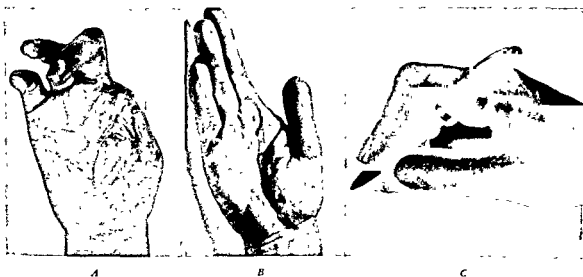


Fig 92—A, Stump of proximal phalanx of thumb following severe electric burn

B, Reconstruction with bone graft and tubed pedicle flap 10 years later.

C, Patient has powerful grasp and wide range of movement. Regeneration of sensory nerves (pain, heat, and cold) has extended to tip of thumb, which enables the patient to avoid thermal and other forms of trauma.

advisable to insert a small drain in the wound, removing the drain in 48 hours.

Meloplasty.—With the loss of weight or with increasing age, the elasticity of the skin is decreased and the skin falls into wrinkles. This sign of aging is a matter of considerable importance for many, for business as well as social considerations.

These wrinkles may be removed from the forehead by incising in the hairline and undermining the skin down to the eyebrows. The excess skin is removed and the wound sutured.

Reconstruction of Extremities.—Loss of the thumb reduces the efficiency of the hand by one half. The thumb may be reconstructed by phalangization, pollicization of the index finger, and use of local skin flaps or tubed pedicles, with insertion of a bone peg for support. When skin and subcutaneous tissues have been avulsed from the hand, exposing tendon and bone, it is necessary to cover the defect by shifting local flaps or applying a pedicle flap from another area. One of the most suitable sites is a flap from the lower abdomen,

lata for the suspension of the paralyzed face. It is thought that less tendency to form adhesions which limit motion occurs when this type of graft is used.

Skin Excision.—Following a facial paralysis of some years' duration, the skin sags in redundant folds. In conjunction with fascial or muscle suspension operations a modified meloplasty may be performed and all excess skin excised

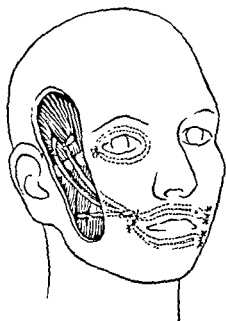


Fig 91—Method of using fascial strips to support paralyzed facial muscles

CLEFT LIP AND PALATE

Introduction

Egyptian mummies have been found showing this congenital deformity, and Celsus is credited with being the first to repair cleft lips by paring the margins and suturing the wound. According to the latest surveys it has been found that cleft lip or palate occurs once in 750-850 living births in a Caucasian population, although the incidence is only half as frequent among Negroes. A cleft lip is approximately twice as often situated on the left side and is more frequent in males.

Embryology

The classical concept of development of the component structures of the lip, alveolar process, and palate by growth and subsequent coalescence of five various processes about the primitive oral cavity has been questioned by a number of investigators during the past 30 years. According to Veau there are no processes and clefts. The nasal orifices are formed by invagination of the surface ectoderm. The ectoderm is invaded by the mesenchyme growing into the lip and subnasal region, from the lateral part of the face. Thus, the primitive primary palate and lip are formed. Only in the posterior palate is there fusion of processes from either side. Failure of mesenchymal pene-

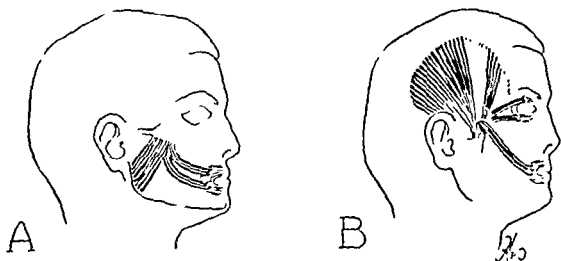


Fig 93—A, Correction of facial paralysis with two muscle pedicles obtained from the anterior part of the masseter muscle. Care must be taken to avoid injury to the parotid duct and to the nerve supply of the muscle strips.

B, Facial paralysis may also be corrected by muscle pedicles carried down to the corner of the mouth and into the upper and lower eyelids.

tration at the 11 mm. stage may thus result in various types of cleft lip or palate, due to rupture of the thin epithelial partition by the strain of constantly growing facial structures

Recently, Stark has studied serial coronal sections from the lip to the oropharynx through the heads of five human embryos with clefts of the lip or palate. The volume of mesoderm present on all sides of the clefts was calculated after making planimetric measurements. When mesoderm was present on one side and not the other, a cleft lip occurred on the side that lacked mesoderm. When mesoderm was absent on both sides, a bilateral complete cleft lip occurred. The hard and soft palates are formed by fusion of two lateral processes which grow toward each other and fuse in the midline. Since mesoderm was present in the palatal areas of all the embryos with a cleft palate, he postulated a relative lack of mesoderm rather than a complete absence to explain the presence of a cleft palate.

Fraser et al. have reported the results of experiments on the process of closure of the palate of the mouse *in vivo*. When the palate is about to close, the palatine shelves project vertically downward on either side of the tongue. A force presumably develops within the shelves, which causes a transition from the vertical to the horizontal plane. There is a bulging inward of the medial side of the shelf, with a concurrent retraction of the ventral border. This process begins posteriorly and proceeds anteriorly in a wavelike motion, until the two shelves rise above the tongue to meet in the midline, if the width of the head and the shelves is appropriate. The mechanism of palatal closure involves the delicate integration of a number of processes, each under the influence of genetic and environmental factors.

Recent work of the school of experimental embryologists shows that specific organs and tissues are induced by "organizers" and also by the action of various chemical substances upon embryonic cells. These organizers act on embryonic cells and alter their pattern of growth. The development of the optic cup from the neural plate, for example, is induced by a portion of the archenteric roof immediately beneath it. It has been shown that any

lack of direct contact of the cells with the organizer will prevent or modify the development of specific tissues or organs.

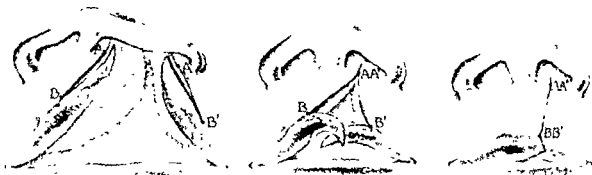
Etiology

The precise cause of this condition has not yet been established with certainty. While the commonly suspected conditions of alcoholism, syphilis, falls during pregnancy, and malposition of the fetal hands or tongue have been blamed, there is no doubt that these can be excluded in favor of other possibilities, such as vitamin or other dietary deficiencies as well as rubella and acute infections occurring during the first trimester of pregnancy, which may exert an adverse effect on the product of conception. It has been claimed that pregnancies occurring late in the reproductive life of the mother tend to produce a greater number of children born with congenital defects. A recent study does not confirm this conclusion. It must not be forgotten, moreover, that there is a definite positive family history in 20-30% of cases. Warkany believes that cleft lip and palate are inherited as a double recessive trait, one gene being autosomal and the other sex-linked. According to Fogh-Andersen, cleft lip with or without cleft palate should be considered hereditary, whereas other factors are responsible in isolated cleft palate. It has been found that offspring of pregnant mice treated with cortisone show a high incidence of cleft palate as well as other defects. X-irradiation of pregnant laboratory animals results in a high incidence of congenital anomalies in the offspring. Different types of deformities may be created by administering the x-rays at various periods of gestation, when certain organs or structures are in a critical stage of development.

Types

The cleft of the lip may be unilateral or bilateral, partial or complete. A cleft palate may involve merely the uvula or extend into the soft palate, hard palate, and finally through the alveolar ridge, when it is associated with a complete cleft lip and palate. In the simplest variety the cleft of the lip may result in notching of the vermilion border, and there is usually a muscular defect and asymmetry of the nostril on the affected side. When the

A.



B.



C.



Fig. 96—A, Thompson operation (modified) A and A' are marked with methylene blue at the base of the columella and the nostril but retaining sufficient tissue on each side to form a floor for the nostril equivalent in width to the opposite nostril. The vertical length of the lip on the normal side is measured with a compass from the base of the nostril to the vermillion border of the lip. Fixing the regulating screw at this length, measurements are taken in the lip at each side of the cleft, commencing at A and A' and extending to B and B'. The latter points are placed at the junction of the skin and vermillion of the lip. The margins of the cleft are now denuded with a single stroke of a narrow bladed scalpel. The nostril and skin of the cheek on the cleft side are undermined freely as is the internal lining of the skin of the ala and tip of the nose. Correct relationship of the external and internal skin surfaces of the ala is maintained by through and through mattress sutures tied over a small piece of Xeroform gauze. The muscle layer of the lip is approximated with several No. 5-0 catgut sutures. The skin edges are sutured with interrupted No. 4 stainless steel sutures, and after excising the excess vermillion in the form of double V-shaped flaps, these are imbricated and sutured with No. 5-0 Dermalon sutures. A Logan lip bow is then applied to immobilize the lip.

B, Appearance of preoperative partial cleft lip without palatal defect.

C, This procedure simulates the normal philtrum and in suitable cases provides a full, well shaped lip.

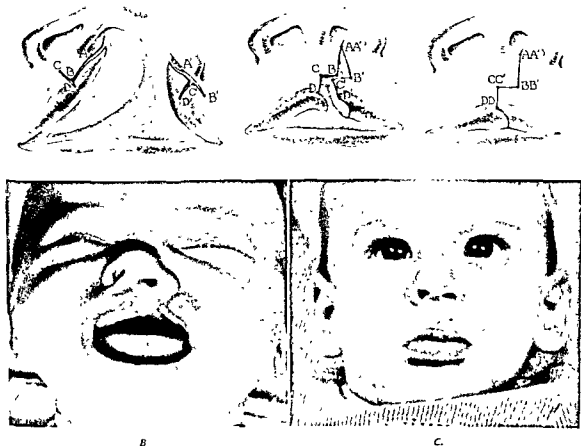
condition is more marked, the notching of the lip extends for a varying distance toward the nostril and there is frequently notching of the alveolar process. In yet another type the cleft involves the floor of the nostrils; the nostril is markedly widened and the alveolar process is cleft.

Operation

Cleft Lip.—The opportune time for operation is from 6 to 8 weeks after birth. There are

those who advocate operation within the first week of life, but since there is never an emergency due to feeding problems, there is no reason for not taking advantage of the better condition of the patient and larger and more easily handled tissues of the lip when the child is somewhat older. Consistently better results can be obtained when the child is in good condition and has reached a weight of 9-10 pounds. The ideal operation should result in

A



B

C

Fig 97—A. Hagedorn-LeMesurier operation. The points *A* and *A'* are located as in the previous method. The point *D'* is selected on the mucocutaneous border of the cleft side as high up as possible where the mucous membrane is adequate in thickness. From this point a line *D'C'* is extended perpendicularly from the vermillion border and varies from 3.5-4.5 mm. in length, depending on the age of the child. The lines *A'C'*, *C'B'* are of equal length. The latter curves slightly toward the vermillion border. This produces a moderate pouting of the lip which is a normal characteristic. On the medial side a line *AB* the same length as *A'B'* is drawn down to the vermillion border, and a line *CD* is extended into the lip at right angles to the mucocutaneous border. This is the same length as *CD'*. Incisions are then carried out as shown in *A*, and after free undermining of the cheek and lining of the ala, the flaps are approximated as illustrated in *A*. The muscle layer and skin edges are sutured with fine catgut and wire as previously described.

B and *C*. Preoperative and postoperative photographs showing the full pouting lip which is obtained.

a nostril of the size, shape, and position similar to that on the opposite side—a full, loose lip and a symmetrical vermillion border, simulating a cupid's bow if possible, and a faint scar. Prior to operation the child should be free of obvious respiratory infection, and the skin about the lip should be clean. The most satisfactory anesthetic is endotracheal gas and

oxygen supplemented, when necessary, by ether. This provides the operator with sufficient time for a leisurely and planned operation. If the hemoglobin is lower than average, a blood transfusion should be given of 100-150 ml., and this may be administered through the femoral vein or into the long saphenous vein at the level of the internal malleolus. Since

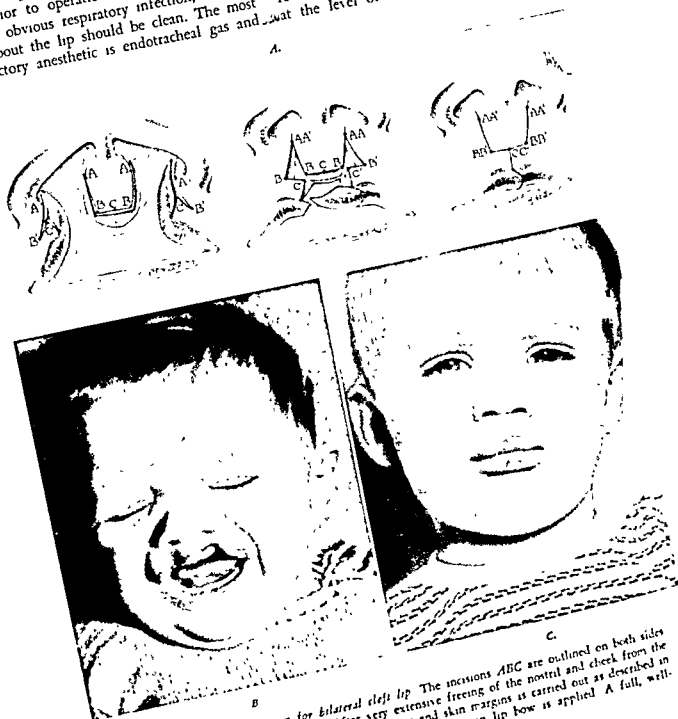


Fig. 98.—A, Operation for bilateral cleft lip. The incisions ABC are outlined on both sides of the cleft and on the probulum. After very extensive freeing of the nostril and cheek from the maxilla on both sides, suture of the muscle layers and skin margins is carried out as described in single cleft. The vermillion border is imbricated and a Logan lip bow is applied. A full, well-balanced lip is obtained by repairing both sides at the same time.
B and C, Preoperative and postoperative photographs.

the type of deformity varies considerably, it is helpful to employ the Thompson, Mirault-Blair, or Hagedorn-LeMesurier procedures when indicated, depending upon the type of cleft

Bilateral Cleft Lip—This type of deformity requires quite a different procedure from those employed in the single cleft lips, in order to

avoid unsightly tight lips, too frequently seen. The premaxilla, if protruding, should never be replaced posteriorly by excising a wedge of septum or even by splitting it obliquely and sliding the premaxilla back. In the past, this practice has been one of the most common causes of severely retracted upper lips. The object to be achieved is the displacement of

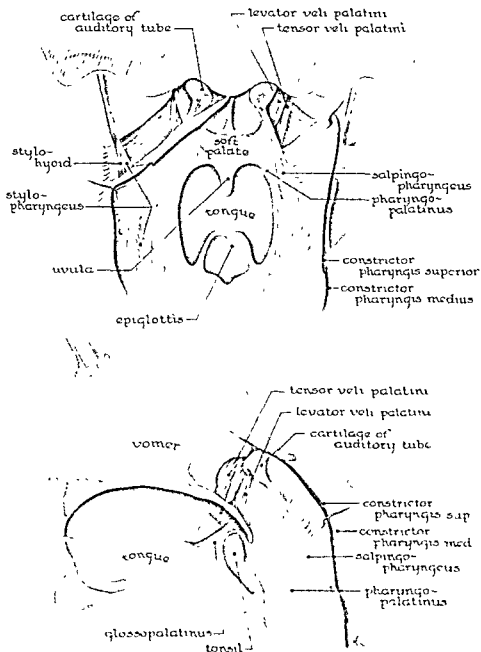


Fig. 99.—Posterior and lateral views of the palatal muscles are illustrated

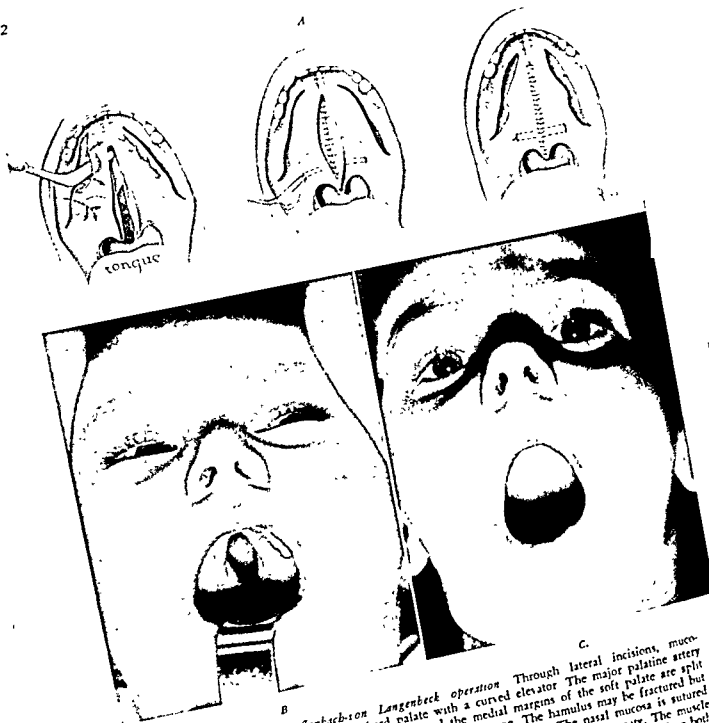


Fig 100—A, Dieffenbach-von Langenbeck operation. Through lateral incisions, mucoperiosteal flaps are raised from the hard palate with a curved elevator. The major palatine artery is spared. The palatal aponeurosis is severed and the medial margins of the soft palate are split or pared so that an even raw surface is prepared for suturing. The hamulus may be fractured but only if necessary to secure adequate mobilization of the soft palate. The nasal mucosa is sutured in a separate layer with fine catgut sutures so the knots project into the nasal cavity. The muscle layer and oral mucosa are then sutured with horizontal mattress sutures of Dermal, including both surfaces of the uvula. A single large stay suture may be inserted in the soft palate if desired.

B, Cleft of palate to alveolar ridge is shown.

C, Healing of palate with minimal scarring following von Langenbeck operation. No speech therapy was required.

the premaxilla posteriorly by traction of the repaired lip muscles, causing it to assume a relatively normal relationship to the maxilla. Marked interference with its blood supply will inhibit normal growth. In this type the cleft may be complete or incomplete, as in the single lip. Following operation for cleft lip, the suture line is kept clean by swabbing with cotton-tipped applicators dipped in penicillin solution 1:1,000, until bleeding and crust formation cease.

The baby may be fed by a small glass syringe or may be allowed to drink from a small cup or glass. The sutures are removed as indicated, starting on the fourth day. Application of arm splints for the entire postoperative period will prevent the child from injuring the healing lip.

Cleft Palate—The goal of cleft palate surgery has long since advanced from the concept of merely providing an intact partition between the oral and nasal cavities. With improvement in technique it has become obvious that the function of normal speech is the aim to be achieved in every case. This must include the services of the speech therapist, orthodontist, and, if necessary, the prosthodontist. The ability of the soft palate to move freely upward and backward and close the nasopharyngeal sphincter is the test of a successful operation, and those procedures which may interfere with this function of the soft palate should not be employed. In 1764, Le Monnier, a French dentist, reported the first successful repair of a cleft velum.

The optimum time for closure of the cleft palate is between 18-24 months. The reason for this is that there is a marked increase in length of the soft palate from birth until about 1½-2 years of age, followed by little growth until about 5 years, when a gradual increase in length occurs. A cleft palate does not materially affect the nutrition of the child, although a higher incidence of middle-ear or sinus infections occurs in these individuals. This probably results from the admission of oral contents into contact with the nasal passages. The types of operations most commonly carried out are as follows:

The von Langenbeck Procedure—Longitudinal relaxation incisions are made near the teeth. The mucoperiosteal flaps are raised from

the hard palate and elevated toward the midline after severing the palatal aponeurosis and fracturing the hamulus. These may be felt as small bony prominences in the anterior and lateral part of the soft palate on each side. The margins of the cleft are split or carefully pared, removing only the mucous membrane. The nasal side of the mucous membrane is then approximated with a number of interrupted catgut or dermal sutures, with the knots projecting into the nasal cavity. A large horizontal mattress suture is carried from the lateral border of the soft palate, on one side, to the opposite region, where it serves as a restraining hammock to splint the muscles of the soft palate during postoperative healing. The margins of the cleft in the oral cavity are then approximated with horizontal mattress, dermal sutures from the anterior termination of the cleft and extending into the soft palate and over the posterior aspect of the uvula.

The patient is fed a liquid diet, which is gradually changed to a soft diet at the end of a week. The sutures are removed in approximately two weeks, and subsequently normal diet may be resumed.

Other Procedures—In clefts involving only the soft or part of the hard palate, a U-shaped incision may be made medial to the teeth from one maxillary tuberosity to the other, and the whole mucoperiosteal flap which has been raised may be set back after stretching the major palatine arteries from the posterior palatine foramen. The cleft which is present is closed at the same time in the manner previously described.

Occasionally, a "set back" may be performed secondarily on an intact repaired palate which is too short to permit good speech function. The raw surface thus exposed on the nasal surface of the palatal flap may be skin grafted in an effort to minimize scar contracture of the raw surface.

Another method of obviating the raw area on the nasal side of the palatal flap is to dissect free the mucosa covering the nasal surface of the palatal bone but to leave it attached posteriorly to the soft palate. When the entire palate is subsequently displaced posteriorly, the strip of mucosa on either side may be sutured to cover any raw area on the nasal surface of the mucoperiosteal flap.

In complete clefts of the palate, another procedure which is of great value is the vomer flap, by means of which the cleft in the hard palate is closed by inserting a flap of mucosa from the vomer underneath the edge of a mucoperiosteal flap which has been raised on the opposite side of the palate.

Persistent small perforations which occasionally occur following a palatal operation, due to separation of the suture line, may be closed by freshening the margins of the perforation and resuturing the marginal defect, after relaxation incisions have been made.

When primary surgical repair followed by a reasonable period of speech therapy has failed to result in normal speech, other surgical procedures should be considered. One method is to elevate a pharyngeal flap containing mucosa and muscle, about 2 cm wide. The flap is based superiorly. A raw bed for the pharyngeal flap is prepared by dissecting the mucosa from the posterior border of the soft palate for a distance of about 1 cm. The free end of the flap is then sutured with mattress sutures to the nasal surface of the soft palate. The flap tubes itself as it heals, and aids in adequate closure of the palatopharyngeal sphincter. Very definite improvement in speech may be anticipated.

Following operation on children with cleft palate, many require special care to achieve the utmost measure of rehabilitation. Frequent dental examination and filling of cavities in both deciduous and permanent teeth are necessary to avoid extensive caries and formation of dental abscesses.

Orthodontic treatment begun early may be necessary to align irregularly situated teeth properly or to expand narrow dental arches. When palatal tissues have been extensively scarred, prostheses with an extension to partially occlude and aid normal closure of the nasopharynx may be necessary. Secondary adjustment of scars or contour defects of the lip or nose should be performed when the child is old enough so that the maximum permanent improvement in appearance will be obtained. Speech therapy is frequently necessary to obtain the optimum improvement in speech and should be started as soon as the child is 4 or 5 years old.

TRAUMATIC INJURIES OF THE HEAD AND FACE

Intracranial and Facial Injuries

In severe motor accidents and other types of major trauma to the head, it is always important to consider the possibility of intracranial complications, such as cerebral or extradural hemorrhage, cerebral concussion with edema, or contusion and laceration of any part of the brain. Among the signs which should be noted are periods of unconsciousness, either immediate or developing after a lucid period, vomiting; changes in pulse, temperature, and blood pressure; increasing papilledema; and loss or hyperactivity of various reflexes throughout the body. The patient may be restless or become violently disturbed and require restraints. Further signs of severe cerebral injury are indicated by a leakage of cerebrospinal fluid from the nose or of blood and spinal fluid from the ears. If at all possible, the collaboration of a neurosurgeon should be obtained so that intracranial procedures may be carried out if necessary without delay.

With the exception of a terminal condition, there is no contraindication to the taking of roentgenograms, provided reasonable precautions are taken. Morphine, however, should not be given, since it would mask the symptoms essential in diagnosing the patient's condition. At this stage it is usually wise to practice conservative care and to reduce and splint only those fractures which may cause obstruction to respiration or prevent control of hemorrhage. Supportive treatment, careful nursing, and maintenance of a free airway are the trial of care on which a successful outcome of many of these cases depends. After the first 24 hours, signs of meningitis may develop as a result of tears in the dura—usually in the region of the ethmoid bone or frontal sinuses—although basal fractures not infrequently may result in infection in the middle fossa extending through the external auditory meatus. It is important to perform a careful examination of the cranial nerves since medicolegal difficulties may arise should the patient claim injury to any of the cranial nerves following surgical intervention.

Scalp Injuries

Scalp wounds may vary from small lacerations to partial or complete avulsion. In the former group it is usually only necessary to shave the scalp about the wound, after carefully cleansing the margins and deep recesses of the wound. The wound may be closed with interrupted sutures of wire or nylon. The application of a firm pressure dressing will prevent the formation of a hematoma and usually makes stab drainage wounds unnecessary. Deep burns of the scalp may cause bald areas which may be eliminated by repeated partial excision and closure, by undermining or transposition of flaps. Not infrequently, large or small areas of the scalp may be avulsed. These should be treated as soon as possible. When the area is no larger than 2-3" in diameter, it is possible by undermining or shaping of local flaps, to close the defect immediately without undue tension. Closure is facilitated by making relaxing incisions in the galea to permit a more complete stretching of the scalp. On the other hand, in the case of extensive or total avulsion, the pericranium should be cleansed and dermatome skin grafts (0.016-0.018" thick) should be applied without delay. It has been found that by local and general use of antibiotics and scraping of purulent exudate from the surface, it is possible successfully to skin graft wounds of this type up to 48 hours after injury.

Lacerations and Other Injuries

Facial lacerations are very common and their etiologies diverse. Many are due to accidents in the home. Some are the result of industrial accidents, while others occur following crashes of transport vehicles and sporting accidents. Because of their frequency, certain basic principles should be observed, if facial disfigurement or functional deformity resulting from inadequate treatment is to be avoided.

Primary measures following serious facial injuries are control of hemorrhage, maintenance of adequate airway or its provision by tracheostomy, and treatment of shock by transfusion of blood or plasma. The blood supply of the soft tissues of the face is excellent, and there appears to be increased resistance to infection which permits healing even under un-

favorable circumstances. Local anesthetic is most satisfactory in uncomplicated cases. With the advent of new antibiotics, capable of inhibiting a wide spectrum of bacteria, clean-cut lacerations may be closed by primary suture 24 hours after the accident. Tetanus antitoxin or a booster dose of toxoid, if the patient has been actively immunized, should be administered.

Preliminary examination should determine the condition of the *parotid ducts, the branches of the facial nerve, and the possible disturbances of vision*, such as diplopia or interference with ocular movement. Subsequently, the frontal, nasal, zygomatic, maxillary, and mandibular bones should be palpated gently to form an estimate of the extent of the injury.

Wounds of the face fall into various categories. One of the common results of automobile accidents is the presence of *road burns* with deeply imbedded dirt and oil ground into the tissues, which may be removed only by careful scrubbing with a small brush with soap and water or some detergent. Only when the wound appears clean to close inspection should it be dressed with Xeroform gauze and a pressure dressing. Permanent tattoo marks will result if these foreign substances are not removed completely.

Lacerations of the face should be carefully cleansed with soap and water and hydrogen peroxide or aqueous Zephiran. The recesses of the wound should be minutely explored for embedded glass or foreign bodies. In these cases only filamentous epithelial margins or small, jagged edges should be excised since scars are invariably finer when no débridement, as is commonly understood, is carried out. It is useful, in many instances, to excise a small wedge of the dermis on each side of the wound so that when continuous, subcuticular sutures of No. 40 gauge stainless steel wire or Dermalon are inserted in the deepest part of the dermis, the margins of the wound will pout slightly. The epithelial margins of the wound are then carefully approximated with interrupted sutures of wire or Dermalon and tied without tension, this will enhance eversion of the wound edges. These sutures should be removed by the third or fourth day, since the wound is supported by the layer of deep sutures.

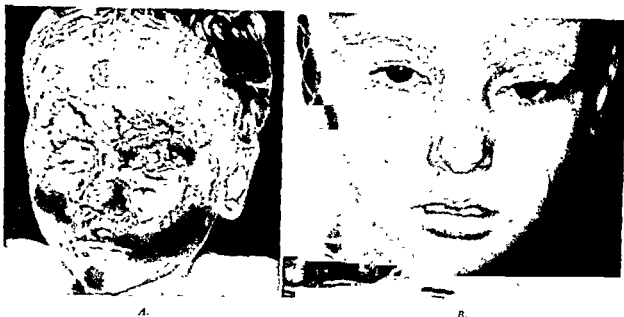


Fig 101—*A*, This child was run over by a car and dragged along the road. Photograph, taken on admission to hospital, shows avulsion of most of left eyebrow and part of skin of forehead, with multiple abrasions of the face. Dirt and oil were deeply embedded in the abrasions and lacerations.

B, Emergency treatment included antishock therapy and meticulous removal of all dirt and oil by careful scrubbing with a small sterile toothbrush and soap. A split-thickness skin graft was sutured to the skin defect. Definitive surgery at a later date included reconstruction of the left eyebrow with a full-thickness graft from the scalp and a postauricular skin graft to correct a slight ectropion of the left upper eyelid.



Fig 102—*A*, Patient was struck on head with roller from printing machine which caused concussion, fracture of temporal bone, frontal bone communicating with the frontal sinus, complete separation of malar bone from all bone and soft tissue attachments and contused wound of soft tissues.

B, The frontal bone was replaced, the malar wired to the zygomatic arch and frontal bone, and the wound closed with fine sutures after careful cleansing and minimal excision of soft tissue. Result six months postoperatively. (This patient was treated in conjunction with Dr. Arthur Fixidge.)

In a kinetic region, it has been shown that a stretching and widening of the scar which often develops gradually over a period of a few months is minimized, since the everted wound edges are first drawn level before the margins can separate to form an unsightly scar.

Lacerations caused by glass are prone to create large *trap-door flaps* with long bevelled thin margins which form a lumpy, elevated flap on healing, due to contracture. This effect may be minimized if as much of the bevelled edges as possible is removed in an effort to convert the injury to that of a simple incised wound. Furthermore, the lengthy use of a

replaced into its normal position and retained with fine interrupted No. 40 gauge sutures. Severed muscle of the lip or cheek should be retained in a similar manner to avoid the creation of a muscular hiatus which will cause a most unpleasant cosmetic defect.

A small defect of the face caused by avulsion of tissue may be repaired by undermining the adjacent tissue with direct suture or by sliding or rotation flaps. A large defect with a vascular bed may be dressed either temporarily or permanently with a free skin graft. If a large portion of the cheek, lip, or nose has been avulsed, the skin and mucosal sur-



Fig. 103—A, Large trap-door flap of cheek with base situated inferiorly. The large metal missile which created the wound also caused a depressed fracture of the malar bone.

B, The depressed zygomatic fracture was elevated directly through the wound, following which careful cleansing and suturing of the wound were performed. Result one year postoperatively.

firm pressure dressing over the flap for 3-4 weeks during the period of fibrotic contraction tends to minimize its tendency to contracture.

Through-and-through wounds involving the ear, lip, cheek, nostril, or eyelid should be approximated, in layers, with the utmost accuracy, and should remain immobilized in a pressure dressing for some time to permit complete healing. The intermediate structures should be approximated with sutures, as well as the mucosa and skin surfaces. For example, cartilage of the nose or ear should be

faces should be sutured to prevent distortion and contracture while definitive treatment is planned.

Occasionally, a portion of the ear, nose, or other feature may be severed completely. If the part can be retrieved and resutured in position within a period of a few hours after injury, there is a fair possibility of the fragment taking as a free graft. Use of a pressure dressing and systemic antibiotic therapy are important adjuncts. If seen early, wounds of the floor of the mouth and tongue may be

sutured primarily. Systemic therapy with antibiotics should be used. If Stensen's duct has been severed and diagnosis is made at the time of the injury, a filiform catheter may be inserted through the duct from the mouth into the severed posterior portion and the cut ends approximated over the catheter with fine sutures. The filiform may be removed in a few days. Chronic fistulas resulting from injury to the parotid gland or duct are most simply and effectively treated by application of a pressure dressing for several weeks and avoidance of a diet and substances during the period of healing which cause marked salivary secretion.

Deep lacerations may cause facial paralysis from severance of the main trunk or branches of the facial nerve. At time of injury an attempt should be made to locate and suture the nerve ends if possible because secondary procedures are more difficult.

Fractures of Facial Bones

These fractures should be immobilized as soon as the condition of the patient permits, since more accurate reduction of the fragments is possible, the patient is more comfortable, and healing without complication is more likely to occur. Laceration of the soft tissues if combined with fractures of the facial bones may greatly simplify reduction of the fracture, following which the soft tissues may be sutured. Comparatively simple operative procedures or methods of splinting suffice in a large proportion of facial fractures.

Nasal Bones.—Fracture of nasal bones usually causes bleeding from the nose, nasal obstruction, ecchymosis of eyelids, and frequently visible deformity. One nasal bone may be depressed, there may be a fracture dislocation of both bones and septum, or the nasal bones and frontal processes of the maxilla may be comminuted and flattened. Simple fractures of the nasal bone may merely require elevation of the bone by means of a rubber-sheathed hemostat within the nose, assisted by external molding with the fingers. When the bone is extensively comminuted and depressed backward between the frontal processes of the maxilla, in addition to elevating the bones and straightening the crumpled septum, it will

usually be necessary to support the bridge by means of two No. 28 gauge wire sutures passed through the nose beneath the fragments from one side to the other. The wires are tied over the lead plates so that the whole acts as a hammock as well as a splint, thus elevating the bridge of the nose and compressing the multiple fragments together.

Zygoma.—An obvious symptom is a flattening of the malar region on the affected side, although subsequent edema may conceal this feature. There may be anesthesia of the upper lip, limitation of movement of the mandible, and diplopia. Fractures of the zygoma may result in a depressed fracture of the zygomatic arch or in the body of the zygoma, or the bone may be extensively comminuted. In the first two varieties the bone may be elevated through a small incision made in the temporal region. A heavy, curved elevator is passed downward, deep to an incision in the temporal fascia, and by an upward prying motion the bone may be raised to correct position. When the bone is comminuted, it is necessary to expose the fragments through an incision in the mouth. An opening is made into the maxillary sinus, and the depressed bone fragments, especially the floor of the orbit, are elevated into normal anatomic position. Then a Kirschner wire is drilled through an intact portion of the zygomatic arch and driven under direct vision beneath the floor of the orbit into the medial wall of the maxillary sinus. This procedure may be supplemented with gauze packing in the maxillary sinuses for a week.

Maxilla.—The most characteristic feature of maxillary fractures is mobility of a segment of the upper teeth or the entire middle third of the face, which is usually elongated. The teeth do not occlude and the patient cannot masticate food. There may be associated fractures of the zygoma and nasal bones, with leakage of cerebrospinal fluid through the nose. The commonly observed types of fracture of the maxilla are fracture of one half of the maxilla, bilateral fracture of the maxillae through the floor of the maxillary sinuses, and pyramidal fracture of the maxillae which usually involves one or both of the zygomatic bones and traverses the nose through the

region of the glabella. The first and second varieties of fractures merely involve placing the upper and lower teeth in correct occlusion after the reduction of the fracture and wiring the jaws together. In these cases much comfort and necessary support will be afforded by a firm bandage which encircles the head and jaw. The pyramidal type of fracture usually involves applying a rather complicated apparatus in which the maxilla is maintained in correct position by means of a Kingsley splint fastened to a plaster headcap by adjustable metal rods or by transverse fixation with Kirschner wires.

the facial bones being blown away together with some of the soft tissues.

The appearance of a patient who has suffered a recent fracture with displacement of the mandible is alarming to those who may not have had much experience with this type of injury, but if a fair number of the patient's maxillary and mandibular teeth are present, there is probably no other bone that may be so simply and accurately replaced following trauma. The maxilla, if intact, forms an exquisitely accurate counter die in which the separated fragments of the mandible and its contained teeth may be interlocked and retained



Fig. 104.—A, The bridge of the nose is crushed and displaced down and backward, forcing the medial canthi apart. Deep lacerations expose the bony fragments and nasal passages.

B, After elevation of the bony fragments and replacement of the fractured septum they are retained by a double wire sling passing through the nose and tied over aluminum plates on each side.

C, Postoperative appearance.

The period of fixation of fractures of the maxilla varies from 3-8 weeks, depending upon the type of fracture and possible complications which may have developed.

Mandible.—Fractures of the mandible are usually classified according to anatomic location and may occur at the symphysis, body, ramus, neck of condyle, or coronoid process. Quite commonly fractures are bilateral, and occasionally three or four separate fractures are present. Gunshot wounds may result in large segments of the mandible or of any of

with eyelet loops or arch bars and intermaxillary wires. It should be noted, moreover, that about 90% of fractures of the mandible fall into this category.

When the lower jaw is edentulous, wires may be carried circumferentially about the mandible with the ends projecting into the mouth so that they can be twisted over the patient's lower denture or a substitute. The simplest way to circumscribe the mandible with wire is to carry one end of the wire down the outside of the mandible close to the periosteum,

and through a (2 mm) incision in the skin with a large, straight abdominal needle; it is then reinserted through the same skin incision and carried up on the inside of the mandible close to the periosteum until it perforates the mucosa on the inner aspect of the alveolar ridge. Pin fixation appliances such as the Roger

Anderson splint or individually molded acrylic splints may be employed when it is necessary to treat the fracture without immobilizing the mandible.

Diet.—When the jaws are wired together, it is necessary to feed the patient a diet of semifluid consistency. It has been found that



A



B



C

Fig. 105—A, Markedly depressed fracture of the right zygoma nine days after accident

B, After freeing and elevation through the mouth, the bone did not remain in position. A Kirschner wire was drilled through the intact part of the zygomatic arch and driven through the zygoma beneath the orbital floor into the medial wall of the maxillary sinus

C, X-ray showing position of Kirschner wire following operation

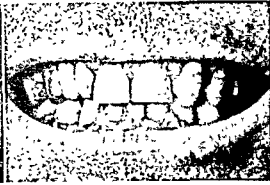
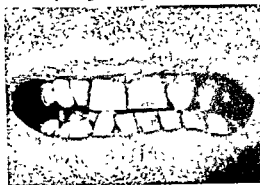
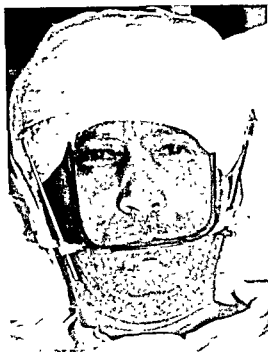
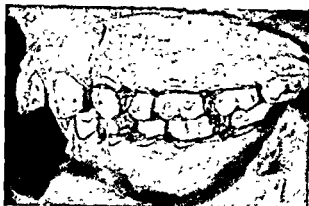


Fig 106—A, Pyramidal fracture of maxilla and nasal, ethmoid, and frontal bones, with displacement posteriorly. Displacement of maxilla upward and backward is shown by the position of the teeth.

B, Result following reduction of maxilla and retention with intermaxillary wiring and reversed Kingsley splint.

C, X-ray showing extensive comminuted fractures of nasal, frontal, and ethmoid bones. Note aerocele from air which has entered through the fractured cribriform plates and torn dura.

A.



B

Fig 107—A, Model showing the method of applying an eyelet loop of No 25 gauge stainless steel wire about teeth

B, Arch bars wired to upper and lower teeth and held together by intermaxillary wires Displaced fragments reduced

even with a complete complement of teeth the patient can manage with little weight loss if a diet of approximately 3,000 ml. of fluid and semifluid substances containing 3,000 calories are taken each day. It is advisable to supplement this diet with Casec or skimmed milk powder and add to the mixture a high multivitamin concentration which may be dissolved in the liquid feedings. Careful oral hygiene measures should be maintained throughout

Anesthetics.—The anesthetic of choice is local, supplemented when necessary in the more complicated fractures by a short period of Pentothal anesthesia. Thus the troublesome complication of vomiting is rarely encountered

Complications.—A tooth in the line of fracture should be preserved even if loose, since

it may prevent displacement of a posterior fragment until sufficient time has elapsed to permit adequate callus to form. At the first sign of development of invasive infection the tooth should be removed. Osteomyelitis is rarely seen if antibiotic therapy has been employed. Adequate drainage should be provided and sequestrum formation awaited before removal is attempted. Nonunion may occur because of malalignment or loss of bone. The bone ends are freshened and wired in proper alignment, or a bone graft may be inserted to replace missing tissue. Trismus may occur as a result of prolonged immobilization of the mandible or from scar tissue formation or bony obstruction. It usually disappears spontaneously, but mechanical dilating appliances may be used if required. Surgical removal of scar tissue or displaced bone fragments may be necessary in some cases. Ankylosis due to intra-articular bony or dense fibrous union may follow a blow on the jaw or a fracture. The condyle may be removed or a segment of bone 1 cm. or more in width is removed across the width of the ramus below the condyle if it is solidly united.

Dislocation.—The usual dislocation is forward and may be unilateral or bilateral. Upward, backward, and forward dislocations may occur but are associated with fracture of the condyle. Forward dislocation may occur by yawning, vomiting, undue use of a mouth gag, or a blow on the chin with the mouth open. Chronic recurring dislocation may occur if the temporomandibular ligaments become relaxed. Diagnosis is made by noting that the mouth is held open and fixed, chewing is impossible, and the condyle may be felt in front of the mandibular fossa, which is empty. In unilateral dislocation the chin is deviated to the opposite side. Treatment consists of lifting the chin with the fingers, while the thumb is protected in gauze are placed on the lower molar teeth, and pressure is exerted downward. A general anesthetic may be necessary, if muscle spasm is present. To prevent recurrence, opening of the jaw may be limited by applying a chin bandage for three weeks or intermaxillary wires which will only permit excursion of a centimeter.

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Obtainable From</i>
Rhinoplasty Using Profilometer (1937)	16 min	Silent	Claire L. Straith, M.D. 2605 W. Grand Blvd Detroit 8, Mich
Treatment of Crushing Facial Injuries (Due to automobile accidents) (1939)	30 min	Silent Color	Claire L. Straith, M.D. 2605 W. Grand Blvd Detroit 8, Mich
Hand Reconstruction (1946)	17 min	Silent Color	William H. Frackelton, M.D. 324 E Wisconsin Ave Milwaukee 2, Wis
Plastic Reparative Surgery—Surgical Repair of Facial Paralysis (1953)	30 min	Magnetic Sound Color	Arthur J. Barsky, M.D. 172 E. 72nd St New York 21, N. Y.

Film References—Cont'd

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Scalp Avulsion (Report of a case and suggested treatment) (1946) (By Claire L. Straith, M.D., Detroit)	34 min	Silent Color	Parke, Davis & Co Joseph Campau at the River Detroit 32, Mich.
Skin Bank Storage of Post-mortem Homografts, Methods of Preparation, Preservation and Use (1955) (By James Barrett Brown, M.D., Minot P. Fryer, M.D., and Thomas J. Zaydon, M.D., St. Louis)	22 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn
Hemimandibulectomy and Immediate Restoration With Acrylic Implant (Demonstrates a new technique for immediate reconstruction of surgically resected mandible) (1953) (By Martin J. Healy, Jr., M.D., Yonkers)	25 min.	Sound Color	Central Office Film Library Veterans Administration Vermont Ave. and H St., N.W. Washington 25, D. C.
A New Surgical Technic for the Surgical Reconstruction of Inferior Maxillary Prognathism (1941)	30 min	Silent Color	Arthur E. Smith, M.D. 1930 Wilshire Blvd Los Angeles 5, Calif
Single Harelip by Hagedorn-LeMesurier Technique (1948)	27 min	Silent Color	Claire L. Straith, M.D. 2605 W. Grand Blvd Detroit 8, Mich
The Treatment of Chronic Stasis Ulcer by immediate split-thickness skin grafting after excision of the ulcer and the abnormal surrounding skin delimited by the lymphatic injection technique of Hudack and McMasters (1955) (By Carl A. Moyer, M.D., and Harvey R. Butcher, Jr., M.D., St. Louis)	26 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn

Neurosurgery

Arthur R. Elvidge, MD

THE SCALP

Much can be learned from an examination of the scalp. In traumatic cases areas of ecchymosis and swelling give information which may be valuable from a diagnostic and from a medicolegal point of view. In a practical sense a severe contusion may indicate underlying fracture. Marked edema may indicate fracture and even rupture of the dura mater. Hemorrhage and edema of the soft extracranial tissues may form a ridge round the edge of an area of contusion which may simulate a depressed skull fracture. This line of swelling can be smoothed out with the examining finger. The crepitation of loose bone fragments can sometimes be palpated.

A contusion in the temporal region should suggest the possibility of an extradural hemorrhage from the middle meningeal artery. Contusion over the general region of the venous sinuses suggests the possibility of subdural hematoma. Discoloration and swelling of the eyelids and periorbital tissues associated with the development of subconjunctival hemorrhage suggests fracturing of the base of the skull. If fracturing is slight, or if it lies at some distance, the discoloration may take a day or more to develop. The subconjunctival hemorrhage generally appears first in the temporal segment. In severe cases it may involve the whole area about the iris. In direct orbital or neighboring injury, periorbital swelling and subconjunctival hemorrhage appear immediately.

Hematomas, excoriations, and lacerations of the scalp hidden by hair may easily be missed, and serious infection results, if neglected. Ecchymosis of the mastoid region usually accompanies a fracturing of the floor of the middle fossa with linear extension into the petrous bone. In this case, bluish discoloration of the eardrum generally develops at once in severe cases, or it may take one or two days to appear if the fracturing is slight or lies at some distance.

Acute infections of the scalp are now uncommon, due to early diagnosis and improved methods of treatment. Swelling about the eyelids may be associated with some neighboring inflammatory or vascular lesion either extracranial or intracranial, such as frontoethmoidal sinusitis, osteomyelitis of the frontal bone, cellulitis of the orbit, epidural or subdural abscess. Periorbital swelling also results from venous congestion in thrombophlebitis and arteriovenous aneurysm.

In nontraumatic cases the general appearance and contour of the scalp is also informative. Sebaceous cysts frequently occur. They may be removed if the patient desires and must be treated when infected. Epidermoid and dermoid tumors call for removal. They may arise in the cranial diploe and there may be intracranial involvement. Cranium bifidum with meningocele or encephalocele will be discussed later under the heading of congenital anomalies. Angiomatous lesions and congenital malformations of the dermis can be treated according to standard methods.

THE CRANIUM

General examination reveals the size, contour, and general shape of the head, the presence of hydrocephalus, craniostenosis, microcephaly, megalcephaly, gross skull defects, irregularities, the relative size of the posterior fossa, and in infants the size of the fontanels and the width of the suture lines. The intracranial pressure can be judged to some extent



Fig 108—Discoloration and swelling of eyelids, subconjunctival hemorrhage, excoriations, and contusion of the side of the head in a case of fracturing of the skull

on palpation over the anterior fontanel. An unusual prominence may indicate the presence of an osteoma or meningioma. Special curiosities of cranial configuration may indicate such conditions as acromegaly or Paget's disease. Auscultation sometimes reveals a bruit which is due to an arteriovenous fistula and occasionally to a large neoplasm.

Roentgenologic examination of the skull reveals the general texture of the bone; congenital lesions, such as cranium bifidum, biparietal foramina, platybasia, and basilar impression, certain changes which result from



Fig 109—Epidermoid cyst of the temporal bone. Lower white area, normal bone; upper white area, epidermoid

increase of intracranial pressure—the beaten silver appearance, enlarged foramina for passing vessels and decalcification of the dorsum sellae, the size and the shape of the sella turcica; disturbances of the endocrine system as in hyperpituitarism; osteomyelitis, tuberculosis,

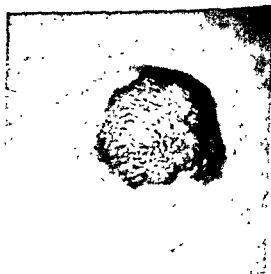


Fig 110—Angioma of the scalp



Fig 111—*A*, Microcephaly *B*, Hydrocephalus *C* and *D*, Craniosynostosis (note congenital biparietal foramina)

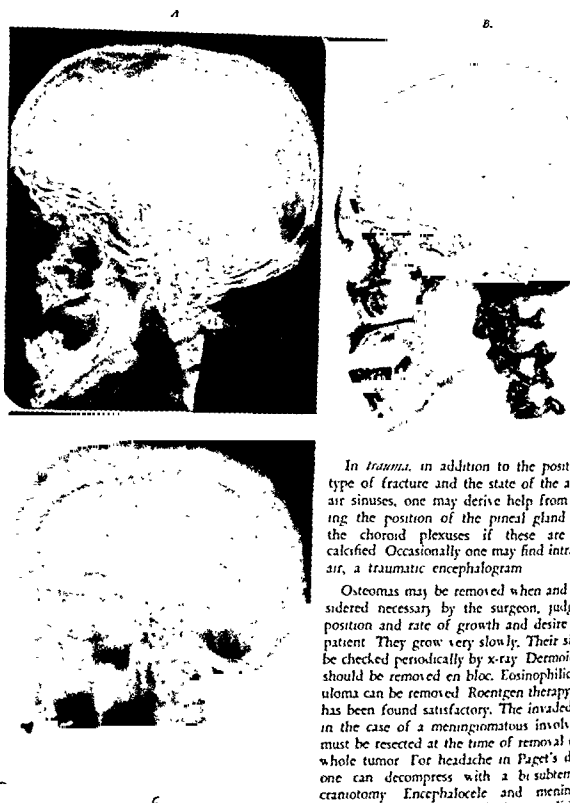


Fig 112 A. Hydrocephalus in adult B. Acromegaly C. Paget's disease

and syphilis; Paget's disease, eosinophilic granuloma and Schüller-Christian disease, the presence of benign and malignant tumors involving the skull.

In *trauma*, in addition to the position and type of fracture and the state of the accessory air sinuses, one may derive help from observing the position of the pineal gland and of the choroid plexuses if these are visibly calcified. Occasionally one may find intracranial air, a traumatic encephalogram.

Osteomas may be removed when and if considered necessary by the surgeon, judged on position and rate of growth and desire of the patient. They grow very slowly. Their size can be checked periodically by x-ray. Dermoid cysts should be removed en bloc. Eosinophilic granuloma can be removed. Roentgen therapy alone has been found satisfactory. The invaded bone in the case of a meningiomatous involvement must be resected at the time of removal of the whole tumor. For headache in Paget's disease one can decompress with a bi-subtemporal craniotomy. Encephalocele and meningo-encephalocele can be repaired. Osteomyelitis can be cured by block removal including, if necessary, extirpation of the frontoethmoidal accessory air sinuses or mastoidectomy if either constitutes the primary focus. Craniostenosis can be corrected by certain surgical procedures which create artificial suture lines.

THE MENINGES

The meninges are involved in the formation of congenital anomalies as in cranium bifidum and spina bifida with meningocele. The meninges may be involved in infection—acute, subacute, pyogenic, tuberculous, granulomatous, fungiform, and parasitic. Streptococcic, staphylococcic, and pneumococcic meningitis used to be frequently fatal. A few patients recovered from streptococcic meningitis by the use of forced fluids and adequate drainage by lumbar puncture or other means. Pneumococcic meningitis was 100% fatal. Patients with staphylococcic meningitis rarely recovered. Since the employment of chemotherapy and antibiotics the mortality rate, even of pneumococcic meningitis, has been lowered to 30%. In fact, in an otherwise uncomplicated and well-treated case, with eradication of the primary focus, recovery should occur. Even often-repeated daily lumbar punctures seem to be unnecessary except for exact evaluation of progress. At present streptomycin is being used in the treatment of tuberculous meningitis with success. Occasionally one used to encounter pachymeningitis associated with a rapid spread of infection throughout the subdural space. This was invariably fatal.

Sometimes the subdural space may become primarily involved with the formation of a subdural abscess, and occasionally an extradural abscess may be encountered.

The case for subdural abscess has also improved. At one time from 60-100% fatal, the results of treatment have improved from better appreciation of the condition, better surgical treatment, and the antibiotics. Infection in the subdural space, untreated, may spread over the whole central nervous system. It is always localized in the beginning and may remain so, arising from an infected accessory sinus or patch of osteomyelitic bone. Early diagnosis is difficult and important. Focal signs may appear, such as epilepsy, headache, and signs of infection. The onset may be insidious. Treatment is by diagnostic trepanation. If pus is obtained, one anterior and one posterior skull trepanation can be made. The pus is evacuated by suction, and antibiotics are left inside the cavity. Drainage tubing or Penrose drains are suitably placed. These are left in for a variable period of days according to the type of case.

With this combined local and general treatment the mortality rate is greatly reduced.

Extradural abscess must be treated by enlarged skull trepanation, evacuation, drainage, and use of antibiotics. A search for the primary focus of infection must be made and the source eradicated. Osteomyelitis or sinusitis must be dealt with appropriately.

The spaces adjacent to the meninges are of great importance in the pathology of trauma. The problems of subdural effusion and hematoma and of epidural hematoma will be discussed under the appropriate heading. Tumors of the meninges will be discussed under the section on brain tumor.

CEREBROSPINAL FLUID CIRCULATION

The skull is a relatively rigid container, and the brain within is lined and protected by three membranes. The *pia mater*, which carries the blood supply for the cortex, clings to its surface, dipping into all the sulci. The avascular *arachnoid* bridges the small sulci but dips into the major clefts such as the median and horizontal sulci. Primitive mesenchyme separates with the coming of the cerebrospinal fluid into the *pia mater* and the *arachnoid*. The resulting space is known as the *subarachnoid space* and is continuous over the central nervous system. The *dura mater*, the outer tough sheath, is really composed of an inner more elastic and an outer fibrous layer. The *dura mater* clings more or less to the inner table of the skull. It splits along the midline of the skull, and at the level of the tentorium and in the midline of the posterior fossa, to form the medial longitudinal sinus, the lateral sinuses, and the occipital sinuses, respectively. It dips into the median cleft between the cerebral hemispheres as the *falx cerebri* and between the cerebellar hemispheres as the *falx cerebelli* and in the horizontal cleft between the cerebral and the cerebellar hemispheres to form the tentorium cerebelli.

Between the *dura* and *arachnoid* is the subdural space, which normally contains a little yellow fluid. In pathologic states this potential space becomes of great significance as in the case of subdural hematoma and of subdural abscess.

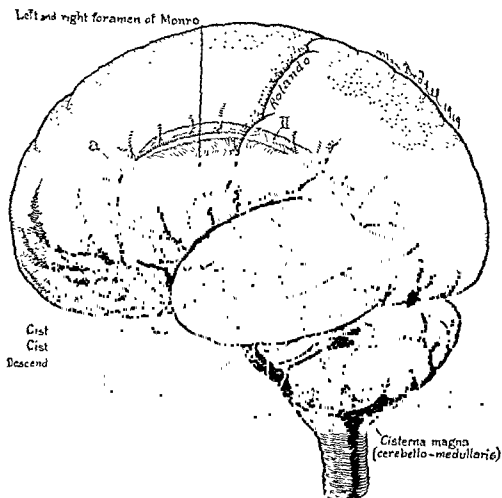


Fig. 113—The ventricular system (Dandy), and subarachnoid spaces through which the cerebrospinal fluid circulates (From Dandy, W. E. *Surgery of the Brain*, Lewis' Practice of Surgery, Hagerstown, Md, 1915, W. F. Prior Co., Inc.)

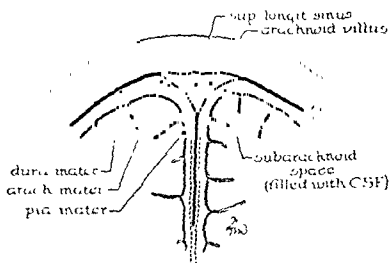


Fig. 114—Coronal section of the superior sagittal sinus showing relationship with adjacent meninges

Between the dura and the inner table of the skull there are generally some areas with little adherence. In certain injuries it is common for bleeding to occur from the middle meningeal artery and vein, which generally lie in and on the outer layer of dura. Extradural bleeding may strip the dura from the skull and give rise to an expanding extradural clot. Pus from an osteomyelitis may collect in the extradural space. Thus this area under pathologic conditions assumes importance.

The cerebrospinal fluid is formed, few doubt, from the choroid plexuses and is absorbed from the subarachnoid spaces through the vascular bed. Choroid plexus is present in the lateral ventricles. There is a smaller amount in the 3rd and in the 4th ventricle. Both clinical and experimental evidence shows that the fluid formed passes from the ventricular cavities by way of the foramen of Magendie and the foramina of Luschka into the subarachnoid cisternae, and it is thought that much absorption takes place over the surface of the hemispheres.

Under healthy conditions arterial blood enters the cranial cavity at a certain normal pressure, e.g., 1,500 mm of water, and venous blood may leave with a pressure of 100 mm of water.

The intracranial veins must carry a pressure above cerebrospinal pressure, otherwise they would remain collapsed.

The normal cerebrospinal fluid pressure is generally 120-200 mm water, and true normal pressure might be considered to be 150 mm, with the patient lying on his side.

The normal contour of the ventricular and subarachnoid system may be visualized by the injection of air or oxygen into the spinal subarachnoid space or directly into the lateral ventricle. The indications for these procedures are beyond the scope of this summary, suffice it to say that, generally speaking, in cases of high intracranial pressure ventriculography is performed and in other cases encephalography.

If a block occurs in the ventricular system from any cause, be it inflammatory adhesion, congenital atresia of the aqueduct of Sylvius, or tuft of choroid plexus, tumor, parasitic cyst, or abscess, there will arise dilatation of the ventricles proximal to the point of block. The

result is internal hydrocephalus of a noncommunicating variety.

If blockage occurs in the subarachnoid space by adhesions, plastic exudate, diffuse neoplasia, or hemorrhage, dilatation will occur not only of the ventricles but also of the subarachnoid cisternae posterior or proximal to the point of block. This is known as a communicating type of hydrocephalus.

If an expanding lesion arises in the brain it will cause distortion of the ventricular system and may cause some obliteration of subarachnoid markings, which may be seen in the ventriculogram.

If a contracting lesion or atrophic lesion is present, a passive increase in size of the ventricles and spaces may occur, and sometimes areas of focal dilatation of a ventricle give rise to an appearance of traction of a portion of the ventricle, called by Foerster and Penfield "ventricular wandering."

In cases of high intracranial pressure lumbar puncture can be a source of great danger. The pressure is released below but the intracranial pressure forces the brain into the foramen magnum, causing herniation of the cerebellar tonsils which may result in strangulation of the blood supply to the medullary centers and death. In the case of a lesion above the tentorium a second mechanism comes into operation. The uncinate gyrus of the temporal lobe may be crowded into the incisura tentorii adjacent to the cerebral pedicle, compressing the ipsilateral 3rd cranial nerve and even the posterior cerebral artery. The opposite or both cerebral peduncles may be notched against the sharp edge of the tentorium. Serious signs will arise from this type of herniation which include deepening coma, 3rd nerve paralysis, decerebrate rigidity, and hemiplegia, which in 10% of cases may be ipsilateral and may be to some extent bilateral.

Compression of the 3rd cranial nerve causes dilatation and fixation of the ipsilateral pupil. This is sometimes bilateral in terminal stages. Infarction of the occipital lobe from compression of the posterior cerebral artery and infarction of the brain stem may occur as late phenomena preceding death. These mechanisms operate in cases of high intracranial pressure. They are, on occasion, brought to acute fulfillment by lumbar puncture.

SYMPTOMATOLOGY

Symptoms and signs may be briefly discussed under two headings

1. General symptoms and signs of increased intracranial pressure
2. Focal signs

General Symptoms and Signs of Increased Intracranial Pressure

Headache may be generalized, bioccipital, bifrontal, severe, and tending to become continuous. It varies to some extent with posture. Some believe that the headache is more severe in the morning. In final stages it is violent. Even raising the head from the pillow may accentuate it. In the rare case of a ball valve block at the neck of a ventricle, change of head posture may relieve or accentuate the headache. The history must be carefully taken to differentiate headache from other causes, e.g., migraine, arterial hypertension, trauma, psychosis, low intracranial pressure, hypoglycemia, sinus disease, or eyestrain.

Vomiting is due to a disturbance of centers in the medulla, probably on a vascular basis due to pressure anemia.

Papilledema is present in the majority of cases of expanding lesions which have raised the intracranial pressure, although in many cases it is absent. Blurring of the edges of the disc first occurs on the nasal side and may rapidly invade the whole edge. Swelling generally develops in the same order and eventually the normal cup becomes a swollen convex surface. Hemorrhages and exudate often appear relatively late or not at all in these cases of increased intracranial pressure. In arterial hypertension one generally sees more widespread patches of exudate and hemorrhage for the degree of papilledema present.

Clinically during this time the blind spot enlarges and the visual fields become constricted and visual acuity eventually diminished. If the pressure is removed, recovery, good or relative, will occur. If allowed to continue, blindness will ensue. Sometimes when intermittent fluctuations of intracranial pressure occur, transient blackouts may be premonitory signals of increasing intracranial pressure.

In cerebral lesions the papilledema may be greater on the side of the lesion, and papille-

dema does not always occur. In lesions of a frontal lobe near the optic nerve, direct pressure may be exerted upon it, giving a *primary* type of atrophy with pallor, while papilledema may be present in the opposite nerve head from the general rise of intracranial pressure, the Foster Kennedy syndrome. The mechanism is considered to be due to blockage of the sub-arachnoid space around the nerve or pressure on the central retinal vein as it crosses it. The result is swelling of neurofibrils and their eventual destruction unless intracranial pressure is relieved in time.

Papilledema generally develops in a matter of days, weeks, or months but can occur in a matter of hours. Expanding lesions in the posterior fossa do not always give papilledema, on the contrary, pallor may occur.

Signs of acute increase of intracranial pressure may or may not include all or any of the above. In addition, there are some signs that are very characteristic of acute compression and such occur in varying degrees. They are of very great importance in head injury and are described again under that heading.

Third nerve paralysis with progressive and rather rapid dilatation of the pupil and loss of the light reflex on the side of the lesion, often associated with some degree of extrinsic ocular paralysis, is believed to result from herniation of the hippocampal gyrus over the free edge of the tentorium which compresses the oculomotor nerve. In late cases the posterior cerebral artery may be occluded, giving infarction. In addition, a similar sequence of events may follow on the opposite side.

The pulse is generally slowed by acute rises of intracranial pressure and the pulse pressure widened with increase of systolic and some lowering of diastolic pressure. This is no doubt an effect on the medullary centers, as are also vomiting and hiccoughs.

Drowsiness and unconsciousness are signs of compression. Yawning is said to be a sign of a lesion within the 3rd ventricle. Rubbing of the nose has been given a similar significance. The respiratory rhythm may be affected in increased intracranial pressure. In severe cases this may lead to Cheyne-Stokes rhythm and finally cessation of respiration. Pressure on the medulla readily causes cessation of respiration, and the slightest manipulation of the lower

brain stem will cause a variation in rhythm. A rapid, stimulated, but regular respiratory rate occurs in certain types of upper brain stem or diencephalic involvement.

Herniation at the level of the incisura tentorii may cause some compression of the cerebral peduncles against the edge of the tentorium and thus give rise to various degrees of corticospinal tract involvement, including hemiparesis, spasticity, and reflex changes. In some cases the paralysis is on the ipsilateral side which is due to notching of the opposite cerebral pedicle. Frequently there is some involvement of both sides and a tendency toward decerebrate rigidity. Herniation of the cerebellar tonsils through the foramen magnum

upon the physiology of the particular areas involved.

The frontal lobe is that portion of the cerebral hemisphere which lies in front of the fissure of Rolando. Lesions of the frontal lobe are likely to be associated with various mental and behavioral disturbances which may be obvious or may be so slight as to pass recognition. Lack of initiative, concentration, and impaired judgment, laissez-faire attitude, slovenly habits, loss of ethical standards, and moral impairment have been described. These disturbances also may accompany a temporal lobe lesion. It is thought that such symptoms are more severe when the lesion involves the dominant hemisphere.

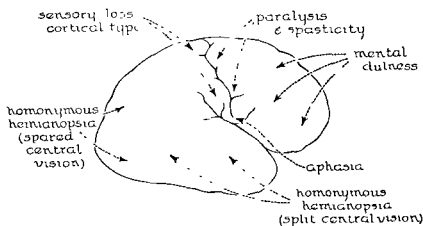


Fig. 115—Localization of certain lesions in the cerebral hemispheres which produce obvious disturbance of function (After Penfield and McEachern, 1938)

also occurs as a result of increased intracranial pressure. In the extreme this results in death as a result of involvement of the respiratory center.

Focal Signs

Focal signs must be assessed on the basis of the anatomy and the physiology of the brain. A full description of typical syndromes would require considerable space and is beyond the scope of this chapter. However, one may work out many possible combinations of symptoms and signs by taking cognizance of simple anatomic and physiologic data.

Localization can generally be made upon accurate history and clinical examination. Focal signs are produced by the local compression or disturbance of the lesion. They will depend

If the lesion is large enough or if the lesion is so placed as to disturb the motor cortex or the radiations from it, various grades of paralysis will result according to the exact position relative to the motor cortex. Thus a lesion anterior to the motor cortex in the parasagittal region will give maximal disturbance in the foot, whereas one in the fissure of Sylvius will give most change in the face and speech mechanism.

In the dominant hemisphere motor speech seems to be subserved by an area immediately above the fissure of Sylvius and anterior to the motor cortex in the posterior portions of the inferior frontal gyrus, where it is subdivided by the two anterior rami of the lateral sulcus, forming the pars triangularis. A lesion in this general area will give rise to a disturbance in

speech in which the patient understands and may even know what he wants to say but cannot utter the words. When this is on an intellectual level it is called motor aphasia or expressive aphasia. This is to be differentiated from dysarthria, which consists of an inability to execute the movements necessary for normal articulation.

Generally, disturbances of speech are of a mixed type and difficult of anything but the simplest analysis. It must also be realized that much of the speech mechanism must lie beneath the cortex.

cur from lesions of certain areas on the under-surface of the frontal lobe. Disturbances of autonomic function are thought to arise from lesions of the frontal lobe.

The parietal lobe is bounded in front by the fissure of Rolando, posteriorly by the parieto-occipital fissure, and below by the fissure of Sylvius. It contains the postcentral convolution which includes cell stations for discriminatory or deep cortical sensation. Lesions in the parietal lobe which encroach on or disturb this convolution cause loss of discriminatory sense from muscles and tendons

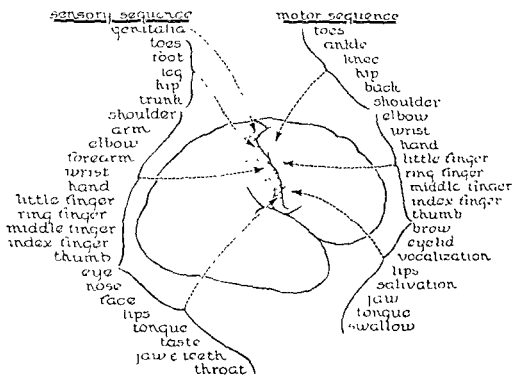


Fig 116—Motor and sensory sequences along the fissure of Rolando (After Penfield and Boldrey, 1937)

Stimulation of the brain at certain points in front of the motor cortex in the second frontal convolution provokes conjugate eye turning to the opposite side. Sometimes one encounters an irritative lesion, which causes the eyes to be rotated to the opposite side, and rarely a paralytic lesion, which allows them to swing to the side of the lesion, presumably from relative overstimulation from the opposite side. In an epileptic seizure this is more commonly seen in the phase of exhaustion. It is known that other areas of the cortex, when stimulated, will cause deviation of the eyes to the opposite side. Irritability and hyperactivity are thought to oc-

cur from sensory receptors in the skin, all of which can be tested practically by testing for sense of position and two-point discrimination. Such other tests, as for the recognition of texture and weight, can be added.

The parietal lobe contains other functions which have to do with the elaboration of receptive mechanisms, but these are not well understood. Astereognosis falls into this category.

Along the upper margin of the fissure of Sylvius there are probably centers which have to do with speech, and the author feels that when this area is involved the principal dis-

turbance is one of impaired memory for words, giving rise to degrees of anomia. In addition, lack of understanding is also evident

Disease in the region of the angular gyrus produces a disturbance of reading, alexia, and of writing, agraphia, which is part of the speech mechanism.

Lesions deep in this region may cause degrees of pressure upon a segment of the optic radiation. This results in a lower quadrantic field defect on the opposite side

The occipital lobe lies posterior to the parieto-occipital fissure and contains the calcarine cortex. Lesions here produce homonymous hemianopia to the opposite side, lesions above the calcarine fissure cause a homonymous

in the temporal lobe beginning 5 cm. posterior to the tip. No speech representation is found in the first, second or third convolution anterior to this and speech representation seems to be more in the second temporal convolution. However, it is impossible to distinguish between first and second in this area." Regarding the function of olfaction, hearing, and memory, the reader must be referred to standard works on physiology and current literature

Many patients with cerebral disease suffer convulsive seizures. The seizure itself may be of localizing value. Thus a seizure arising in one frontal lobe may start with the head and eyes turning to the opposite side, followed by jerking of the contralateral arm and leg and

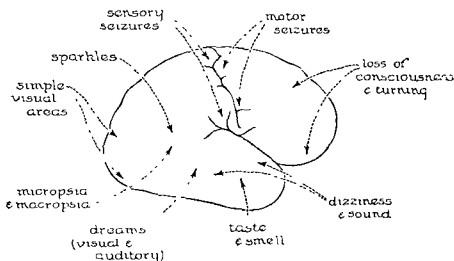


Fig. 117—Common initial phenomena in epileptic seizures arising in different areas of the brain (After Penfield and McEachern, 1938)

quadrantic hemianopia below and to the opposite side; and lesions below the fissure quadrantic homonymous field cause defects above the horizontal. Lesions posterior to the lateral geniculate body may show sparing of macular or central vision.

The nondominant temporal lobe seems to be relatively silent, but on the dominant side the first temporal convolution contains certain connections for speech which is generally severely disturbed in lesions of this region. The patient can utter words and phrases, but they are meaningless and confused. This is sometimes referred to as a jargon aphasia. It has been stated by Penfield that "speech is located

loss of consciousness. Discharges further anterior can give rise to turning of the head followed by rotation of the trunk

A lesion adjacent to the motor cortex may cause clonic movements in the appropriate limb. The discharge generally spreads to neighboring areas, giving rise to a Jacksonian march. Speech becomes involved if the dominant hemisphere is affected.

The accurate description of the march of events in a seizure determines its value in localization. In this the recognition of an aura is all-important. Auras may consist of a sensation rising from the epigastrium, numbness of a limb, an olfactory, gustatory, visual, or audi-

tory hallucination, according to location. For example, an attack arising in the parietal cortex may commence with numbness of the arm, with clonic movements following as the wave passes forward. An aura of twinkling lights may be described from a lesion in the posterior parietal region. Attacks from the occipital lobe may precipitate transient hemianopia. Dizziness is a frequent aura in lesions of the temporal lobe. Micropsia and macropsia are described and occasionally auditory hallucinations. Sometimes a past event will reappear in the patient's mind, "déjà vu" phenomenon. Autonomic phenomena may be observed in lesions of the diencephalon.

Lesions in the neighborhood of the 3rd ventricle which involve the thalami cause hypesthesia or paresthesia of the contralateral side of the body. As the internal capsule is affected there are various degrees of hemiparesis.

If the hypothalamus is involved diabetes insipidus becomes a feature. Autonomic disturbances with changes in pulse rate and blood pressure are sometimes found. Occasionally in an expanding lesion one encounters changes in the visual fields from involvement of the neighboring optic tracts. If convulsions occur they cause flushing of the face and neck, sweating, rise in blood pressure, and alteration of pulse rate.

Expanding lesions within the 3rd ventricle cause blockage of the cerebrospinal fluid, with resulting dilatation of the ventricular system proximal to the lesion. There may be few objective signs except papilledema, and the patient experiences severe headache which may be intermittent if the lesion is of a ball-valve type.

Lesions in the midbrain give rise to various degrees of ocular paresis with pupillary changes, large pupils, sometimes small and unequal pupils, impaired reflex reactions and failure of conjugate upward eye movement may be observed. Blockage at the aqueduct of Sylvius causes internal hydrocephalus. In neoplasms of the pineal gland pubertas praecox is described.

Lesions of the cerebellar hemispheres generally cause incoordination of the movements of the ipsilateral extremities. Hypotonia with

reduced reflexes is typical. Nystagmus is frequent in cerebellar lesions and is usually greater on gaze to the side of the lesion. Lesions of the vermis cause severe trunkal ataxia and rombergism.

Associated compression of the brain stem produces hypertonicity with increase of deep reflexes from involvement of motor tracts. Lesions of the pons and the medulla oblongata involve cranial nerve nuclei and various combinations of involvement of the long tracts. Hiccoughs, vomiting, and severe disturbances of respiration are likely to occur.

If blockage of the 4th ventricle occurs, internal hydrocephalus results with symptoms of increased intracranial pressure. Headache is severe and there may be few signs.

Tumors involving the coverings of the brain cause neurologic focal signs by local compression of the brain. For example, a parasagittal meningioma may compress the midline sensorimotor cortex, causing characteristic signs. Among other symptoms and signs a meningioma of the lesser wing of the sphenoid bone may cause compression atrophy of the adjacent optic nerve and, in addition, pain referred to the area supplied by the first division of the 5th cranial nerve.

An expanding lesion in the cerebellopontine angle, e.g., a perineurial fibroblastoma of the acoustic nerve, causes paralysis of the 8th and the 7th, often the 5th, and sometimes the 9th, 10th, and 11th cranial nerves, as well as signs of cerebellar and brain stem involvement.

It may be added that there do seem to be sites of predilection for tumors with characteristic signs, but these are too many to list in detail.

HEAD INJURY

The subject of head injuries can be discussed under the following headings:

I Injuries of the Brain

- 1 Cerebral concussion
- 2 Cerebral contusion
- 3 Cerebral laceration
- 4 Cerebral compression
- 5 Cerebral edema

II Traumatic Intracranial Hemorrhage

- 1 Subarachnoid hemorrhage
- 2 Subdural hemorrhage

3. Epidural hemorrhage
4. Intracerebral hemorrhage
5. Subpial hemorrhage

III Fractures of the Skull

1. Linear fracture of the vault
2. Linear fracture of the base
3. Depressed fracture, simple
4. Depressed fracture, compound

IV. Complications, Early and Late, From Intracranial Injury

1. Meningitis
2. Osteomyelitis
3. Brain abscess
4. Post-traumatic headache
5. Post-traumatic dizziness
6. Facial paralysis
7. Post traumatic epilepsy

Injuries of the Brain

Cerebral Concussion.—From a practical point of view cerebral concussion may be defined as a transient lapse of consciousness or as memory loss following immediately upon an adequate blow to the head, without obvious pathologic findings. Because the lapse of consciousness in pure cerebral concussion is brief, it has been difficult in a clinical way to demonstrate any particular characteristic phenomena associated with it; however, recent researches have brought forth some interesting facts which give rise to further speculation.

Denny-Brown and Russell in 1941 published an account on experimental cerebral concussion in the cat and in the macaque in which they studied the effect of concussive blows with the aid of a pendulum. They found that concussion was more easily produced when the head was free than when supported on a hard surface. From this experience they coined the terms acceleration concussion and compression concussion. They found that acceleration concussion in the monkey and cat could be produced by a heavy mass with a velocity of 28.3 feet per second and the energy transmitted, 17.83 foot pounds. Following the concussive blow there was an interference with brain stem reflexes for about 90 seconds which might terminate in death or recovery within five minutes. They concluded that experimental concussion results in the following typical phenomena:

1. Loss of corneal reflex, the most important sign

2. Loss of the pinna reflex, which consists of any flicking movement of the ear, shaking of the head, or scratching with the hind limb, from a stimulus applied to the inner pinna.

3. A sharp rise of blood pressure, not constant

4. Lasting inspiratory spasms only with light anesthesia.

5. An initial jerk in the musculature followed by delayed spasms, and then loss of postural tone. They are abolished by deep anesthesia.

They concluded that death from concussion results from a variety of traumatic primary shock. The primary shock is associated with and proportional to effects which suggested a stimulation of the vagoglossopharyngeal system in the medulla.

In 1944 Walker, et al. made a study of the physiologic basis of concussion by the method of falling weights. They noted that concussion in the cat under Vinethene-Novocain anesthesia is frequently associated with an attack, tonic and clonic. In the monkey, seizures consisted of a tonic phase and were less common. The authors interpret the attacks as an intense stimulation of the nervous system. Changes in respiration and circulation may occur. Usually there is a respiratory gasp followed by irregular respiration, or apnea. Usually a rise of blood pressure occurs at once or a few seconds after the blow, due to an effect upon the brain stem, thus giving evidence of intense stimulation of vasomotor centers leading to peripheral vasoconstriction. They noted sometimes slowing of the pulse due to vagus excitation. Abolition of reflexes occurred only if the spinal cord was intact. These authors believed that intense stimulation of the cerebral cortex and brain stem produces excitation at the moment of the concussive blow, and that this is not due to any alteration in circulation, oxygenation, metabolism, or acid-base regulation of the cerebral cortex because it occurs within a fraction of a second and before such alteration can take place.

In 1916 Windle, et al. found a slight increase of water content 8-17 hours after experimental concussion in animals.

Electrical changes were studied in experimental concussion by Williams and Denny-Brown in 1911. They found that concussion

was associated with "diminution or cessation of the electrical activity of the whole cerebral hemispheres." They observed a delayed appearance of abnormally slow waves which they felt might represent a stage in recovery from concussion. They concluded that concussion was the direct result of mechanical violence to cerebral cells and was not dependent upon secondary changes such as "edema, anoxia, hypoglycemia for its initiation."

Dow, et al (1945) examined electroencephalograms in patients with head trauma who were employed in shipyards. They examined 213 patients, and in 71, electroencephalograms were taken within 30-60 minutes after the accident. They estimated the velocity attained at the moment of impact in split seconds and determined the force involved. The percentage of abnormal records was slightly greater in patients examined within 30 minutes. Rapid disappearance of abnormal electroencephalographic findings, they felt, pointed to a mechanism in concussion other than petechial hemorrhage or contusion. The forces at impact were of the same order as those found by others to produce experimental concussion. Patients who had to stay off work more than 24 hours showed a greater percentage of abnormal records and a more severe injury as judged clinically, but the electroencephalographic records they found less reliable. They felt that there was a difference in susceptibility to electroencephalographic changes in different individuals on the basis of a few observations; also, they did not feel that there was any particular activity that seemed characteristic of the electroencephalogram following mild trauma.

Jasper, et al (1940) found that the electroencephalogram was the most sensitive indicator of cerebral injury and provided a sensitive objective measure of recovery. Electroencephalographic changes found were listed as follows:

1. Random or regular delta waves varying in frequency from less than one to six per second
2. Poor regulation or disorganization of the alpha rhythm
3. Epileptiform discharges

In some cases of severe injury, delta and epileptiform waves and disorganized activity were observed in the electroencephalogram several years after the injury. These were associated clinically with changes in personality, seizures, irritability, disorders of thinking, and, in a few instances, with no remarkable clinical abnormality. Post-traumatic syndromes due to malingering or hysteria were clearly evident since in these none of the characteristic abnormalities associated with genuine head trauma were obtained. The electroencephalogram provides an extra tool in the differential diagnosis of subdural hematoma and effusion, epidural hematoma and intracerebral hemorrhage, and focal and general trauma. According to Jasper, et al. (1945), the more severe the brain injury, the more complete is the disappearance of alpha rhythm and slowing of the delta frequency, and the electroencephalogram would appear to be a more sensitive measure of cerebral damage than routine clinical estimate. It was found that amnesia and confusion, irritability, and drowsiness without loss of consciousness were just as important as the history of unconsciousness in indicating the severity of cerebral damage shown by the persistent electroencephalographic abnormality during the first 10 days. It was found that in skull fracture severe electrical changes are more likely to be persistent and, also in cases with blood in the cerebrospinal fluid, are more likely to show persistent and severe changes, and, furthermore, in the presence of abnormal neurologic signs immediately after the injury, are associated with more severe and persistent electrical changes during the first 10 days. In cases with increased cerebrospinal fluid pressure there was usually more severe abnormality. Young patients seemed to be more vulnerable from the electroencephalographic point of view. The electroencephalogram might return to normal within 24 hours after concussion with loss of consciousness. It was concluded, however, that records taken after the first day and up to 10 days following the injury provided a more reliable picture of residual damage to cerebral tissue and were of greater diagnostic and prognostic value. A normal or mild change in the electroen-

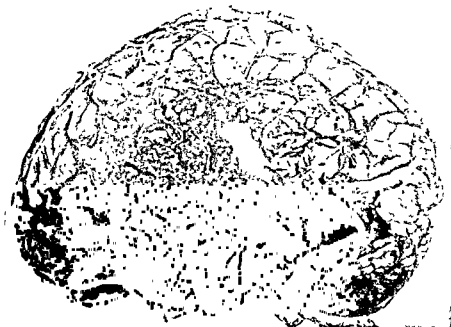


Fig 118—Severe contusion of the brain



Fig 119 Contusion or infarction of the brain stem.

cephalogram provides a useful additional guide to the management of convalescence. Local electroencephalographic abnormality may persist for years and may develop into focal epileptiform discharges associated with areas of degenerating nerve tissue. If the nervous tissue is completely destroyed the electroencephalogram should be normal. Such an area may be seen, however, by pneumography. Minor epileptiform discharges may be seen without clinical seizures but may be accompanied by other symptoms. The electroencephalogram may aid in the diagnosis of intracerebral and extracerebral hemorrhage. It is not

a substitute for careful clinical study of the patient but in conjunction with it makes a valuable contribution.

Cerebral Contusion.—The term cerebral contusion as used in the present instance signifies merely a bruising of the brain. This may be from the effect of the direct blow and may include so-called contrecoup lesions. Contrecoup lesions apparently occur as a result of the skull moving when struck. According to the hypothesis of Holbourn (1944) cerebral contusion and contrecoup can be explained upon the basis of rotary acceleration. The ex-

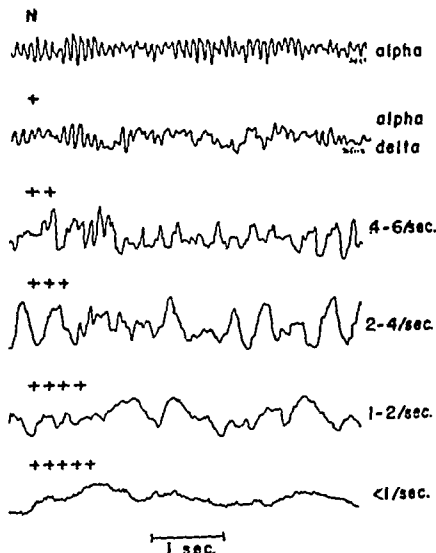


Fig. 120.—Brain waves Electroencephalogram

Typical electroencephalograms showing the changes which occur after head injury, arranged in order of severity. N is a normal record, +++++ is the most severe type of disturbance with marked depression of cortical activity. The intermediary stages are gradual and often overlap. The characteristic frequencies for each type are shown above. (From Jasper, Kerthman and Fludge A. Research Nerv. & Ment. Dis., Proc., 1943, 24: 389-420, 1945.)

periments of Pudenz and Shelden (1946) afford some evidence for this. The mechanism for contrecoup still seems to be very controversial and is obviously partly a function of the inertia of the brain. Very extensive contusion of the brain, such as that seen in the temporal lobe, which has recently passed under the caption "explosion of the temporal lobe" (Botterell) may give rise to considerable cerebral compression due to concomitant cerebral edema.

Cerebral Laceration.—The term laceration simply means a tear in the continuity of the brain substance. This may occur as a result of a depressed fracture or as a gunshot injury and can be diagnosed only by exposure.

Cerebral Compression.—A pathologic degree of cerebral compression may be brought about by intracerebral epidural and subdural hematomas, subdural effusion, and massive cerebral contusion with cerebral edema. The usual signs of acute cerebral compression include increasing drowsiness, slowing of the pulse, widening of the pulse pressure, changes in respiration, progressive dilatation and fixation to light of the ipsilateral pupil, and finally extensor rigidity of varying degree with plantar extension and stiff neck. Homonymous hemianopia may occur as part of the process. Hemiplegia is usually present as a focal sign of the primary lesion.

Two extremely important mechanisms operate in cases of marked increase of intracranial pressure, apart from the focal compression produced by the lesion in a particular case.

1 *Herniation of the brain at the incisura tentorii* is characterized by a herniation of the hippocampal gyrus over the free edge of the tentorium due to an ipsilateral expanding lesion. This causes pressure on the adjacent 3rd nerve, which results in various degrees of paralysis of the oculomotor nerve, commencing usually with progressive enlargement of the pupil and fixation to light. Reid and Cone (1939) found that extradural compression in monkeys would cause an ipsilateral pupillary dilatation which would return to normal on release of pressure. The fixed pupil, they believed, was an early sign of cerebral herniation at the incisura tentorii. Fischer-

Brugge (1951) has brought forth evidence to show that the syndrome is brought about by compression of the 3rd nerve against the edge of the clivus. Reid and Cone (1939) also confirmed the observation of Meyer that associated infarction of the ipsilateral occipital lobe might occur as a result of occlusion of the posterior cerebral artery. In severe cases, the posterior cerebral artery may be compressed, with resulting hemianopia. In some cases, notching of the opposite cerebral peduncle against the free edge of the tentorium occurs to the extent that an ipsilateral hemiplegia is produced (Kernohan and Waltman).

2 *Herniation of the brain at the foramen magnum* consists of a forcing down of the cerebellar tonsils into the foramen magnum. This wedging causes compression of the medulla oblongata with disturbance of vital centers, causing alterations in pulse and blood pressure and early cessation of respiration. Abundance of mucus from the respiratory tubes is common.

Decerebrate rigidity with tonic seizures is due to a lesion or disturbance of the brain stem lying between the red nucleus and the acoustic striations. It may result from ischemia, probably from compression of the basilar artery and its branches.

Cerebral Edema.—This is the most feared complication in head injury and seems to be generally associated with massive cerebral contusion. If such an area of contusion consists of completely destroyed brain, and if such an area of contusion is sufficiently well localized and confined to relatively unimportant parts, the best result may be obtained with a débridement of the destroyed brain. Hypertonic solutions and subtemporal decompression alone are generally completely ineffectual.

Treatment

Cerebral Concussion.—Modern tendency has been to treat cases of simple cerebral concussion with a minimal amount of bed rest. This will be graded according to the duration of unconsciousness and duration of confusional disturbance or memory loss. Usually the patient remains in bed 2-5 days. It is generally felt that the sooner the patient can be rehabilitated and sent back to work, the better.

His case, of course, should be properly evaluated as soon as possible after the injury, and he should be given a clear understanding as to the diagnosis and the length of time that he will probably be off work.

Cerebral Contusion and Laceration.—The treatment of cerebral contusion will be very much the same as that for concussion, except that the more severe cases with neurologic signs will require longer hospitalization. Cerebral laceration is generally combined with depressed skull fracture or gunshot injury. Débridement of devitalized brain must be carried out at the time of elevation of the depressed fracture. The treatment of cerebral compression will resolve itself largely into the question of diagnosis and removal of the cause where that is possible.

Special Tests

X-rays of the skull should be obtained in all cases of head injury. It is safer for all patients with concussion and, of course, more severe injuries, to be hospitalized, especially for the first 24 hours. The electroencephalogram is of considerable interest in the diagnosis and prognosis. Lumbar puncture is of value in demonstrating blood in the cerebrospinal fluid, which gives some indication of the degree and type of injury but is of greater use in estimating increase of intracranial pressure when that is necessary. The tendency is to do few lumbar punctures and to limit their use to the special indications in diagnosis and treatment suggested by continued headache.

Traumatic Intracranial Hemorrhage

Subarachnoid Hemorrhage.—This merely signifies that sufficient injury has taken place to cause bleeding into the subarachnoid space. This, in itself, may not be particularly serious.

The principal sign is stiff neck. It can be diagnosed by lumbar puncture, which probably should be done in the more severe injuries in any case. This gives some idea of the severity of the injury but is only one point in the evaluation of the case. Frequent lumbar punctures to eliminate the blood are unnecessary, and it was shown by Sprong (1934)

that very little blood is removed by this method. However, less post-traumatic headache is encountered when the patient has had a combination of adequate bed rest and lumbar puncture, nevertheless, the general trend is to omit lumbar puncture unless specific information is desired. The general management of the patient, both physically and psychologically, is all-important.

Subdural Hemorrhage.—The diagnosis and treatment of subdural hemorrhage is often difficult, though superficially it would appear simple. It must be suspected from the history, the nature of the injury, and the clinical examination. Symptoms may develop rapidly with signs of increasing intracranial pressure, drowsiness, coma, stiffness of neck, hemiparesis, plantar extension, dilated pupil, pulse and blood pressure changes, or slowly, as an expanding lesion with headache. X-rays may show a shift of the pineal gland or of the choroid plexus if calcified. Ventriculography may be advisable. The final proof, of course, is in seeing the hematoma.

One may puncture the skull with a twist drill according to the technique of Cone. The dura can be opened simultaneously with the drill or with a needle. With a small brain needle the depth of the epidural and subdural spaces can be estimated and in many cases an epidural or a subdural hematoma recognized. If a fluid effusion is present under pressure, the measure may even be lifesaving. Strategically placed burr holes through which one can inspect the subdural space may be preferable. They must be suitably placed to discover the clot. It generally lies over the convexity of the hemisphere but may lie toward the base. Usually biparietal or bitemporal openings suffice. A Penrose drain can be left in the subdural space and brought out through a separate wound. If the subdural hemorrhage is of clotted blood or of long standing with membrane formation, and especially if an inner membrane is formed, it is better policy to turn a reasonably sized bone flap and remove the membranes completely. It is important, of course, to consider both sides in dealing with subdural hemorrhage, as frequently they are bilateral.

Epidural Hemorrhage.—This is diagnosed again on the basis of history, examination,

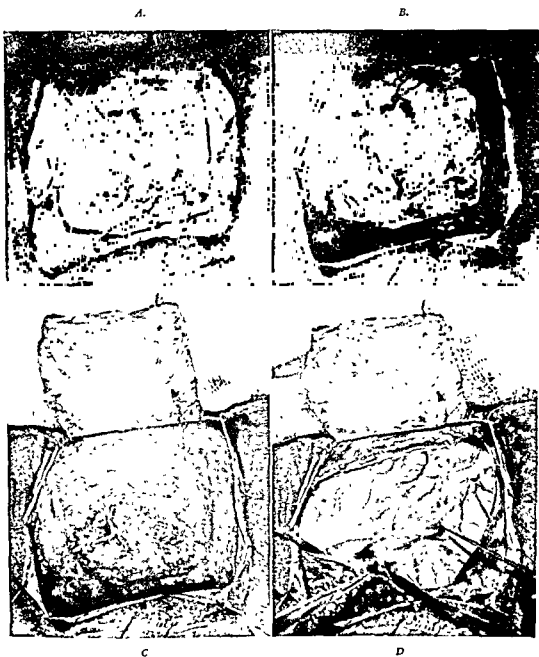


Fig. 121—Case of chronic subdural hematoma, with membrane formation

A, Initial incision of dura, leaving partial attachments at the corners or at the crossing of the middle meningeal arteries

B, Bulging of the outer membrane of the hematoma upon section of the dural attachment

C, Reflection of dura and exposure of the outer membrane of the hematoma

D, Outer membrane already partially removed and semifluid portion of the hematoma partially evacuated. Note hook lifting inner membrane away from the arachnoid. Entire membrane is removed in continuation of operation

progress, and x-ray. A head injury followed by loss of consciousness, a so-called lucid interval and a relapse into a state of coma with hemiparesis as a focal sign and ipsilateral 3rd nerve involvement is typical. An infinite number of variations occurs but the condition can usually be diagnosed on clinical grounds alone. In some cases the acuteness of the compression will be so extreme that the patient will have to be operated upon immediately in order to save his life. On the other hand, gradually increasing drowsiness may be the important factor. One can again choose the method of Cone, with

If there is unnecessary delay or postponement, the brain pressure will cause death from cerebral herniation and irreparable brain stem damage.

Intracerebral Hemorrhage.—If a sizeable intracerebral hemorrhage is demonstrated, it can be removed in part by aspiration or more completely by open operation. This type of lesion is not uncommon. It may be suspected on clinical grounds and verified by ventriculography.

Subpial Hemorrhage.—This need hardly be mentioned but does occur. It is seen during



Fig 122—Case of epidural hematoma after removal. A small, free bone flap has been turned on the fracture line to permit evacuation of the clot. Note drain in lower left trephine hole. Bone has been wired back into place.

a twist drill to verify the diagnosis. This is somewhat quicker than waiting for x-rays, etc. This procedure is used only if one is certain that the intracranial pressure is above normal, which tends to ensure against bleeding from any vessels that are encountered as a result of the tamponading effect of the brain against the dura. When calcified, the position of the pineal gland should be observed by x-ray and the course of the fracture, if one is present. When the diagnosis is sufficiently clear it is essential to do an immediate subtemporal craniotomy and evacuate the clot

intracranial operation and occurs as a result of trauma. It has no particular significance unless it acts as an early or late cortical irritant.

Fractures of the Skull

Linear Fracture of the Vault.—The position of the fracture and its extent will afford some idea of the type of complication to be expected. A fracture over one of the major blood sinuses may be associated with subdural bleeding; if over the temporal region, with epidural hematoma. If a fracture involves the accessory air sinuses, the question of infection

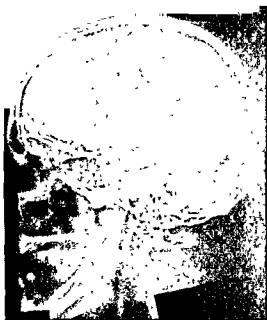


Fig 123—X-ray of basal skull fracture showing intracranial gas

has to be considered. Ingress of air into the cranial cavity may even occur. The extent of fracturing gives some idea of the force of the injury which will help one in the determination of prognosis and the length of treatment. The skull should be x-rayed in all cases of head injuries.

Linear Fracture of the Base.—The patient with a basal skull fracture will, generally speaking, need a longer period of bed rest. There may be associated contusions of the brain and involvement of cranial nerves. The line of fracture frequently crosses the petrous bone to involve the mastoid air cells, with the

risk of infection. Fractures often enter the frontal, ethmoidal, and sphenoidal accessory air sinuses. A leak of cerebrospinal fluid from the ear or from the nasopharynx is common. Prophylactic treatment with suitable antibiotics or chemotherapy is indicated, but meningitis may still occur. It is important to advise the patient not to sneeze forcibly or blow his nose in the presence of a cerebrospinal fluid leak from the nose. He should in that instance open his mouth widely and relax. A semi-Fowler's position is probably helpful. For a leak through the ear, it is probably wisest to do no more than cover the ear with clean dressing on the outside. Some prefer to instill a suitable antibiotic into the ear canal. Most leaks stop in the first 2-3 days. Occasionally there is a recurrence. The leak may call for operative repair. A very free and dangerous cerebrospinal fluid leak into the nasopharynx is frequently associated with a so-called pyramidal fracture in which the facial bones have become severed from the base of the skull. This is common as a result of crashes by airplane and automobile dashboard injuries. In this type of case one must arrange a splinting device to fix the facial bones to the skull, this can best be carried out by the oral surgeon. For certain difficult cases a method of employing a modified Roger Anderson splint applied to the skull has been described by Elvidge and Baxter (1944), which eliminates the necessity for fixation to a plaster cap.

Depressed Fracture, Simple.—In the case of a simple depressed fracture it is routine to elevate the fragments unless the depression

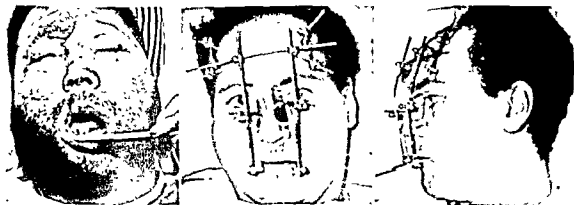


Fig 124 Roger Anderson splint used in case of multiple facial fractures

is very shallow and situated over a silent area of the brain. It is not urgent to reduce this in the first few hours, but it is probably wiser to elevate it within a day or two. It is advisable to make a small opening in the dura to inspect for the presence of subdural hematoma or effusion. Contused brain in the area of a laceration may be débrided suitably.

Depressed Fracture, Compound.—In this instance where the depressed fracture is covered by a laceration of the scalp, the earliest possible débridement of the wound and elevation of the depression is required. Softened brain tissue is débrided when this is irrevocably damaged and often herniated.



Fig. 125—Case of untreated compound depressed skull fracture, showing cerebral herniation covered by dirty, infected granulations and requiring prolonged period of dressings and plastic surgery.

Scalp Laceration.—It is of the utmost importance that scalp lacerations be débrided, cleansed, and properly explored lest foreign bodies or a depressed fracture be overlooked. The hair must be shaved generously about the wound, and if, in the male, the wound is extensive, the whole head should be shaved. The same applies even more if associated with a depressed skull fracture.

Complications, Early and Late, From Intracranial Injury

Meningitis.—Treatment is carried out with the use of suitable antibiotics and sulfa drugs. If penicillin is chosen it can be administered in six hourly doses of 300,000 units, and some combination of antibiotic therapy should be continued until one week has passed following the return of the temperature to normal. Sulfa drugs can also be administered, giving 1 Gm. of sulfadiazine every 4 hours after an initial dose of 2 Gm. If sulfa drugs are used, it is important to examine chemically the level which is maintained in the blood stream and also to make sure that the patient is voiding properly. The result should be followed by daily lumbar punctures. In many severe cases intrathecal spinal injection of suitable antibiotics can be made. Before the introduction of penicillin and other antibiotics and of sulfa drugs, it was possible to cure some patients with streptococcal meningitis by forcing fluids and making repeated lumbar punctures every day. The treatment was called forced drainage. This type of treatment is no longer necessary. However, adequate fluid intake and lumbar punctures to check progress are essential.

Osteomyelitis.—For osteomyelitis of the skull, x-ray examination is important along with the question of tenderness and edema. The treatment is block removal of the osteomyelitic area supported by suitable antibiotics or chemotherapy.

Brain Abscess.—The treatment of brain abscess is discussed in the following section. In the final analysis it is a question of evacuation of the abscess, its proper drainage or its complete removal. If it has been removed completely and cleanly, it is possible to obtain primary closure.

It is extremely important to prevent the production of a brain abscess by adequate débridement of the original scalp laceration.

Post-Traumatic Headache.—Post traumatic headache is part of a so-called post concussional syndrome and may be due to too long or too short a period of bed rest. It may be due to a slight residual collection of fluid, increase of pressure, or disturbance of proper cerebrospinal fluid circulation which could have been relieved by lumbar puncture or could be

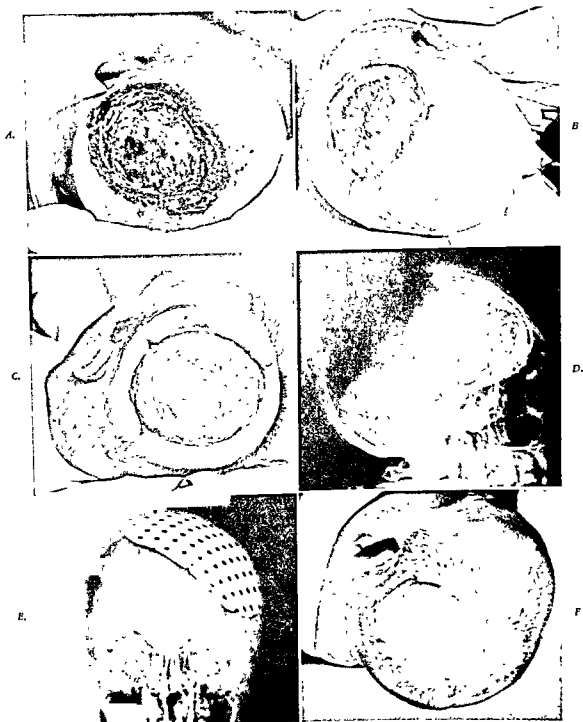


Fig 126—A, Extensive electric burn which has destroyed the scalp and caused necrosis of the skull seen in the picture. The dura beneath the bone was found to have completely disappeared.

B, Preliminary burr hole opening made through the necrotic skull, and escape of large quantity of extra-arachnoidal pus.

C, Fascial graft to replace dura now covered with healthy red granulations about 12 days after removal of necrotic skull.

D, Residual skull defect.

E, Tantalum cranioplasty.

F, A pedicle flap was transferred from the abdomen to the defect in the scalp, using wrist as a carrier (Baxter).

on a psychologic basis if the patient feels that there is something more which should have been done. It can often be relieved by lumbar puncture or encephalogram. It is possible, in rare instances, for a subdural clot or effusion to be overlooked, and, in an occasional case, even brain tumor. The headache may be focal in type or generalized. In some instances it follows lumbar puncture and is then due to a low intracranial pressure from a cerebrospinal fluid leak through the opening in the spinal dura.

Post-Traumatic Dizziness.—This is generally described as an unsteady feeling which the patient states is within his own head. He notices it especially on head movement, e.g., on getting up in the morning. It is common especially when patients have not been well treated, referring especially to the minor closed head injuries. Sometimes it is associated with depressed fractures which may be associated with adhesions. Psychologic adjustment in the form of simple explanation and discussion with the patient, exercise, and work generally suffice to cure. If more radical measures are necessary encephalography may be advisable, which has the advantage of eliminating the possibility of a subdural hematoma and also causes sufficient shift in the pressure relationships that symptoms may disappear. It should be used with psychologic reinforcement. When the dizziness due to a head injury is more vertiginous in character so that objects appear to move and to turn before the eyes of the patient, it is then probably due to a fracture involving the labyrinth. This condition clears up spontaneously in time.

Facial Paralysis.—If the 7th nerve has been paralyzed and fails to show any evidence of recovery, it indicates involvement by pressure of bony fragments within the facial canal. In some selected cases, when spontaneous recovery of function does not occur, an exploratory decompression of the nerve by the otologist is advisable and has given good results.

Post-Traumatic Epilepsy.—To avoid post-traumatic epilepsy, meticulous débridement should be performed at the original operation. Further than this there is little that helps. When epilepsy has developed it is a question then for evaluation and for trial on anti-

convulsive therapy, and the question of surgery may eventually be considered. For a discussion concerning the incidence of post-traumatic epilepsy, see Brock.

BRAIN ABSCESS

MacEwen of Glasgow obtained remarkable results by simple drainage of brain abscess. Nevertheless, since his time, results have been variable and frequently unsatisfactory for certain types of abscess. The care and skill of the surgeon has much to do with the prognosis. Much time and labor are now spared and success more likely to be assured with the antibiotics. Metastatic brain abscesses have generally had a high mortality in the past. Further, the type of organism has affected the results. Modern treatment with the use of antibiotics has considerably reduced the mortality rate.

Abscesses may be divided as follows:

- 1 Single
- 2 Multiple
- 3 Metastatic
- 4 Post-traumatic
- 5 Phlegmonous

Etiology

Single abscesses most commonly arise in civilian life from mastoiditis or from frontal and ethmoidal sinusitis. From the mastoid, the abscess is generally located in the temporal lobe or the adjacent cerebellar hemisphere. From the frontoethmoidal sinuses, it is generally found in the frontal lobe.

Multiple abscesses may occur in one lobe or in scattered areas of the brain. Two abscesses have not been uncommon, one in the frontal and one in the temporal lobe, presumably from sinus infection.

Metastatic brain abscesses, which may be single or multiple, disseminate most frequently from the lungs in cases of bronchiectasis. A so-called paradoxical abscess occurs as a result of septic emboli from a peripheral area passing via a patent foramen ovale to the brain.

Post-traumatic abscess, as the name implies is found beneath or in association with a traumatic wound of the scalp, cranium, or both.

Phlegmonous designates a cluster of small abscesses bound with much interstitial inflammatory tissue. This state occasionally occurs in any of the above groups.

In the case of abscess from sinus infection, the process may be by direct extension through involvement of the successive coverings of the brain and spread via the Virchow-Robin spaces. This process includes focal osteomyelitis and focal pachymeningitis and leptomeningitis. Spread may occur by retrograde venous thrombophlebitis by way of veins passing from the infected mastoid antrum, tympanic cavity, or involved sinus.

The infection in the metastatic type is carried by the arterial blood current as a septic embolus from a distant focus, such as a pulmonary or renal abscess. Septic emboli lodge generally in the middle cerebral distribution. Forty-five per cent of the metastatic type are solitary.

In the case of trauma, cerebral abscess formation is generally by direct extension. It may follow a simple blow but usually complicates a penetrating wound. It may come from the fracture of the base involving a sinus, or an area of softening may serve as a *locus minoris resistentiae* in which bacteria may lodge. The reaction of brain to infection is similar to that of other tissues. There occurs an intense mesoblastic reaction, with exudation of leukocytes, proliferation of endothelial granulation tissue, production of macrophages from microglia and from mesothelial elements, and formation of a connective tissue capsule. The final result is a pus-filled cavity surrounded by a variably formed connective tissue capsule. The surrounding glial elements undergo hypertrophy and hyperplasia. A brain abscess from within out shows formation of a typical structure in concentric layers, with a pus-filled core surrounded by radially disposed granulation tissue and macrophages, a connective tissue capsule, and with the whole surrounded by a variable thickness of reacting glial tissue.

Diagnosis

The general symptoms and signs are those usually seen in cases of increased intracranial pressure which may or may not be associated with headache. Focal signs may be present or absent. Convulsions may occur. A careful his-

tory will generally reveal otitis media, mastoiditis or sinusitis, a furuncle, an infected tooth, infected wound, or bronchiectasis.

The story is often confusing and clinical diagnosis difficult as the following case summaries will show.

A patient who was admitted in decerebrate rigidity had suffered with his right ear since an attack of measles at the age of 2 years, and had undergone two operations on his right ear (mastoid) before admission. He had had a head cold a few days before, with pain in the right ear, headache, drowsiness, and unconsciousness on the day of admission, with bilateral plantar extension, twitching of the left arm and leg, and attacks of decerebrate rigidity and weakness of the left foot. The optic discs were hazy.

A patient who was found to have two abscesses suffered from a draining ear, drowsiness, and aphasia.

Another patient developed sudden headache, vomiting, dizziness, chills, prostration, normal pulse rate, stupor, coma, and aphasia. Temperature 102° - 103° F, hemiparesis, hemianopia. Diagnosis, metastatic brain abscess, probably arterial spread.

Major events taken from a more typical history can be listed briefly thus: pneumonia 4 months before admission, three seizures in 3 weeks, headache $3\frac{1}{2}$ weeks, drowsiness 3 weeks, papilledema, and weakness of left arm recent.

Another history from a case of trauma follows: scalp laceration while tobogganing, repaired, infection, depressed fracture discovered, osteomyelitis, brain abscess drained with cure, result satisfactory but with very occasional convulsions.

The above will give sufficient idea of the variable course of the symptoms and signs in brain abscess.

Diagnosis is difficult. Accurate history of essential points and general physical and neurologic examinations should make one suspect the possibility of brain abscess in most cases and in many, localization can also be given.

For final diagnosis ventriculography may be used, but with an accurate clinical conclusion some would prefer to omit this. The author prefers the precision of diagnosis by ventriculography. This last is dangerous and so must be done carefully, as release of pressure places extra strain on the capsule of the abscess which may rupture. Encephalography by the spinal route might be done in very simple cases but is very dangerous on account of the possibility of cerebral herniation at the incisura tentorii or the foramen magnum.

Lumbar puncture is highly dangerous, and the leak which persists, as also the immediate drop of pressure, may cause rupture of the abscess or a cerebral herniation. The main use is to measure intracranial pressure, which, however, often gives a false reading. If checked by slight jugular compression, this adds to the risk. The principal excuse for lumbar puncture concerns the cell count in the cerebrospinal fluid, as many cases of brain abscess are associated with meningitis. The presence of both meningitis and abscess makes diagnosis very difficult. Enough has been said to show that lumbar puncture is always a hazard. It is prudent, therefore, that this be done when necessary by the man who is ready to operate.

Treatment

Treatment varies from time to time, and in the hands of different surgeons. The basic principle is evacuation and drainage. There are many methods, all of which must be carried out by a trained surgeon, and the postoperative treatment must be carefully supervised, otherwise re-collections are likely to occur.

Various methods of surgical treatment which have been described may be briefly listed as follows:

1. Drainage with suitable large plastic or rubber tube

2. Aspiration of contents and insertion of suitable antibiotic on one or more occasions may suffice and, if necessary, subsequent drainage or surgical exposure, marsupialization, or removal

3. Decompression by craniotomy with subsequent removal of the abscess as it wanders toward the brain surface and its wall thickens

4. Marsupialization of the abscess so that the abscess will eventually turn itself inside out at the surface of the brain

5. Tube and balloon drainage

6. Packing of the exposed cavity in an acute case with moist gauze

7. Simple removal without rupture in a well-encapsulated case

Simple tube drainage, though perhaps more crude than other methods, is in any event a good standard method for all types of cases, and the mortality rate is very low.

Of all the methods of treatment which include the above, the author believes that simple tube drainage is the safest method in the long run and gives consistently good results. In old cases the well-formed capsule and its contents can frequently be removed without rupture.

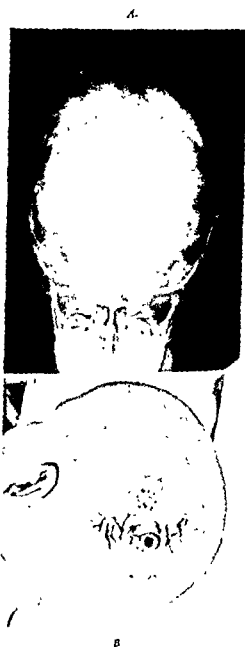


Fig. 127.—A, Extreme ventricular displacement from case of metastatic left parietal brain abscess.
B, Drainage by tube method.

case of metastatic abscess of the temporal lobe simulating tumor was removed in this way in toto as is customary, by the author for tumor. This proved on section to be a typhoid abscess.

containing motile bacilli. The patient had had typhoid fever several months before the abscess was found.

Treatment by the tube drainage method has to include rigid supervision of the postoperative dressings and control of intracranial pressure. It is important to bring the pressure low at dressings by lumbar puncture as pointed out by Cone, so as to open up the cavity. The author prefers to keep the pressure below and to normal in the first two days. After the period of swelling is over, pressure is allowed to rise from normal to slightly above, in order to collapse the cavity more quickly. Before the era of antibiotics, the drainage tube from a cerebral abscess cavity could be removed in 12 days. Today with antibiotics it is removed in 3-5 days.

The mortality with the help of chemotherapy is now low, if all precautions are taken. It was low before chemotherapy, but wounds had to be more carefully supervised. The only feared complication is late epilepsy which develops in approximately one third of the cases.

INTRACRANIAL TUMORS

Intracranial tumors may be divided into the following main groups (after Cushing):

1. Gliomas and ganglioneuromas
2. Meningiomas
3. Perineurial fibroblastomas
4. Pituitary tumors
5. Congenital tumors
6. Blood vessel tumors
7. Papillomas
8. Sarcomas
9. Metastatic tumors
10. Granulomas
11. Cysts

The history and the general symptoms and signs, both focal and general, produced by the majority of the above lesions seem to become more and more characteristic for any particular tumor as one gains experience. Much can be deduced, however, from the few observations made under the preceding sections.

The symptoms and signs of disease of the central nervous system must be elucidated in terms of anatomy and physiology.

The biologic behavior of the various types of tumor is exceedingly varied.

Gliomas

The gliomas form 44% of all brain tumors in patients coming to a neurosurgical clinic. They arise, as the name indicates, from the neuroglia, which form the normal supportive tissue of the central nervous system. Nevertheless they are ectodermal elements. The term does not include the microglia which are thought to be, and behave physiologically as, mesodermal derivatives.

To understand the classification of the gliomas, it is necessary to review the histologic development of the normal glial cell which is illustrated in Fig. 128.

Bailey and Cushing (1926) formulated the first satisfactory classification of the gliomas, basing it upon their histogenesis. Minor changes have been introduced but, in the main, their classification is satisfactory. As any one tumor may contain cells which are multiplying at different levels of development, one may classify a tumor according to the most primitive type of cell that is seen in any number. This is more logical, as many different tumors may show a preponderance of one type of cell.

The relative incidence in a series of 210 gliomas of the central nervous system from the Montreal Neurological Institute is shown in Table 11.

TABLE 11

	%
Astrocytoma	26.10
Glioblastoma multiforme	24.76
Medulloblastoma	13.33
Astroblastoma	6.19
Ependymoma	13.33
Spongioblastoma polare	5.23
Oligodendroglioma {	
Oligodendroblastoma }	3.80
Pinealoma	.95
Neuroepithelioma*	.47
Case of astrocytoma and glioblastoma multiforme (case of triple tumor)	.47
Unclassified	9.52

*Includes two neuroepitheliomas of the peripheral nervous system.

Astrocytomas may occur at any level of the central nervous system and at almost any age. The greatest incidence is in the second, third, fourth, and fifth decades, with the average for the cerebral hemisphere 33.2 years, for the cerebellar hemisphere 34.2 years, for midline

cerebellar cases 9.5 years, and for the spinal cord 43 years.

They are slow in growth. Preoperative symptoms average 30 months but may go on for years. Similarly the postoperative survival may be years and there may be no recurrence.

This large group has been subclassified (of some practical importance) into at least three types

1. Piloid
2. Gemistocytic
3. Diffusum

Into the piloid group are placed all the tumors in which the dominant cell is a fiber-

forming astrocyte. It is true that there are some which are forming few fibers

It is found that this tumor is the one which frequently forms cysts (in 3 out of 10 cerebral cases and 11 of 14 located in the cerebellum). The piloid astrocytoma may occur in the cerebrum, cerebellum, and spinal cord and is rather evenly distributed over six decades. It has the longest prognosis. A 20-year or longer postoperative survival may be hoped for with adequate removal. Every effort should be made at total ablation. The average age for this group when in the cerebrum is 34 years

The gemistocytic type is composed of piloid swollen astrocytes with a large amount

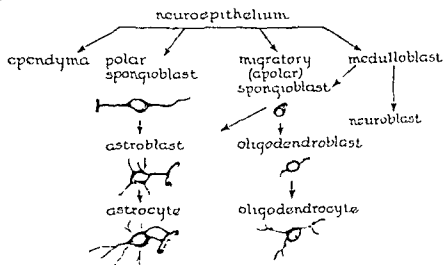


Fig 128 —Development of glia (After Penfield, 1931.)



Fig 129 —Cystic astrocytoma showing mural nodule and ventricular shift



Fig 130 —Gemistocytic astrocytoma showing invasion of gray and white matter and widening of convolutions.

of cytoplasm but few rather large, short processes and a minimum of fibers. This tumor occurs almost wholly in the cerebral hemisphere. The average age incidence is 39, and the majority occur in the fourth and fifth decades of life. There may be a long preoperative and postoperative history, but generally less than for the piloid group. The tumor is likely to be rather fast growing. A 10-year survival is uncommon. Large cysts are uncommon but medium-sized intraneoplastic cysts occur in about one third of the cases.

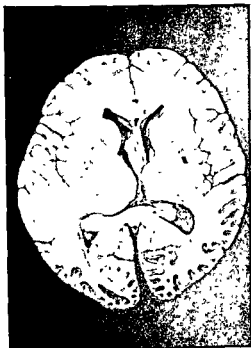


Fig 131—Astrocytoma diffusum Shows diffuse invasion of cerebral hemisphere

The astrocytoma diffusum is so named because of its penetrating characteristics. It is formed of small stellate astrocytes which may be called protoplasmic astrocytes. They tend to wander through the gray matter, passing ganglion cells without destroying them. Sometimes groups form into more solid areas, but these may not be large. Cells pass considerable distances beneath the pia. In the depth of the white matter they may infiltrate and here tend to form fibers. The average age incidence in the above series was 34.5 years, but the majority occur toward the fifth decade in the late thirties. The preoperative symptomatology is shorter, on the average, but many cases go

years before recognition, generally with epilepsy. Because of its infiltrating character, it is difficult to find and recognize and to delimit it at operation. A 10-year postoperative survival is not uncommon.

The surface of the brain may look somewhat whiter than normal, but convolutions are often well preserved. X-rays show merely a uniform shift. Post-mortem examination shows a general slight enlargement of the whole hemisphere. This tumor does not form cysts of any gross size and it occurs in the cerebrum. Unlike the protoplasmic astrocytoma of Bailey, it has not been found in the cerebellum. Some of these tumors can be removed *en bloc*, but sometimes it is wiser to leave them and use x-ray therapy.

The next large group of gliomas is the glioblastoma multiforme. This is a most malignant type generally found in the cerebrum, with an age incidence of 41.2 years and generally occurring in the fourth, fifth, and sixth decades. The preoperative history is, on the average, about six months and frequently less. The postoperative history is short unless a very radical removal is made, in which case one occasionally obtains a longer survival.

The cell type is probably the small polar spongioblast, but the tumor is generally composed of a variety of cells representing different stages of development. Malignant necrosis is common and endothelial budding of vessels and mitoses are frequent. Cysts may occur within the growth but are medium in size and likely to contain thick necrotic material.

There are different ways of dealing with these neoplasms. One is biopsy verification and let alone, especially if speech centers are involved. Another is radical removal. The author believes that radical removal is justified with lobectomy. One such patient has now survived six years. All procedures may be followed with x-ray therapy if desired.

The third large group is the medulloblastoma group, first so classified by Bailey and Cushing (1925). These are primarily tumors of children in the first two decades, though curiously the average age is 19 years, as a few are seen in adults. They occur exclusively in the cerebellum, generally coming from the midline structure, the vermis, or the lateral recess of the 4th ventricle. They are highly malig-

nant and eventually seed out into the cerebrospinal fluid and sometimes metastasize by way of cerebrospinal fluid down the spinal cord and over the brain. They have a short pre-operative history, on the average 6 months, and the postoperative survival varies from a few months to several years. Radical removal followed by x-ray therapy is generally recommended. In many cases of removal, recurrence has been delayed for 2-5 years, and in rare cases patients have been known to survive for many years. The tumor is very sensitive to x-ray, and equally satisfactory results are reported by some where the patient is treated with x-ray therapy alone.

one was reported to arise from the 4th ventricle (Cushing). It has been known to arise from a peripheral nerve. Relatively good results have occasionally been obtained in cerebral cases, especially in children.

One rarely encounters tumors in which the type cell belongs to the neuroblastic development. The ganglioglioma may arise at any level of the nervous system, in the cerebral hemisphere, and in the cerebellum. It is to be expected in association with the spinal ganglia as a paravertebral tumor. It is relatively benign and, if completely removed, is not likely to recur. Neuroblastoma is composed of more

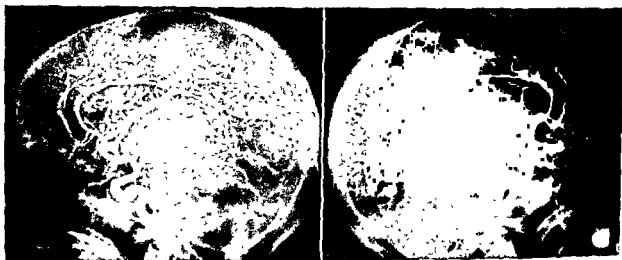


Fig 132 —A, Arteriogram shows elevation of middle cerebral arteries by glioblastoma multiforme containing abnormal vascular network
B, Arteriogram of the opposite normal side for comparison

The remaining gliomas cannot be considered in detail here. They are less frequent. Some are relatively benign. They all have their own special biologic characteristics.

The astroblastoma is somewhat more malignant than the astrocytoma. Ependymoma is moderately benign and arises in proximity to a ventricle or the neural canal. This is also typical for spongioblastoma polare. Oligodendroglioma is generally found in the cerebrum. It is rather malignant, with an average age incidence of 40 years. Pinealoma is found generally in younger individuals. It may be associated with *pubertas praecox*.

Neuroepithelioma is highly malignant and occurs occasionally in the cerebral hemisphere;

immature type cells and has been found in the cerebral hemisphere and midbrain.

Meningioma

The meningiomas form the next major group of brain tumors (13.4% in the series of Cushing, 14% in the series of Montreal Neurological Institute). As the name implies, they take origin from the meninges, and though generally attached to the dura, it is thought that they arise from meningeal cell rests. For example, the author has frequently noted their attachment at the site of a pachionian body. Different varieties have been described by Cushing but common is the meningeal fibroblastoma. The type cell is the

meningeal cell which is mesodermal and may form fibroglial fibers, reticulin, and collagen

These tumors grow to huge size before giving rise to symptoms. They merely displace brain but at their point of attachment invade dura locally and even the cranium, thickening it and passing through it. One can then see an elevation of the scalp. X-ray may reveal an area of bony thickening or endostosis and an increase in size and number of vascular channels. They arise typically at various points along the midsagittal line adjacent to the falx cerebri. They do occur along the line of the fissure of Sylvius. They commonly arise from various regions at the base: lesser wing of the sphenoid bone, dorsum sellae, tuberculum sellae, olfactory groove, floor of the middle fossa, petrous ridge, posterior fossa, foramen magnum, spinal dura, and meninges.

If removed in toto, including involved skull and dura if necessary, they do not recur. Sometimes they show sarcomatous tendencies, in which case they may recur unless radically removed. An osteolytic lesion of the bone may signal such a lesion. They are technically difficult to remove due to general increase of vascularity of the whole region, and it may be necessary to interfere with the great blood sinuses. Arteriography may be of assistance to the surgeon in revealing the pattern of the blood supply.

Perineurial Fibroblastomas

The perineurial fibroblastomas form another group (8.7% in the series of Cushing, 5% in the series of Montreal Neurological Institute). They are benign tumors which arise from perineurium and are found attached to cranial or spinal nerve roots and less often to a peripheral nerve. They do not recur when removed completely. A common type is the acoustic neuroma that occurs so frequently in connection with the 8th nerve as to derive its name from that characteristic. As the tumor enlarges in the cerebellopontine angle of the posterior fossa, it compresses the anterolateral aspect of the pons and the cerebellar hemisphere. Paralysis of the 8th nerve occurs very early, followed by the 7th nerve. The 5th cranial nerve is often involved and less frequently the 9th, 10th, and 11th nerves. The

symptoms and signs can be worked out on the basis of the anatomic structures affected.

Acoustic neuromas characteristically arise from the intracranial portion of the nerve at and within the entrance to the porus acusticus, which is usually seen to be enlarged in x-rays. Acoustic neuromas can be removed completely, in which case they will not recur. The removal generally presupposes section of the facial nerve which leaves the patient with a disfiguring abnormality. This can be largely overcome by suitable nerve transplantation. The most satisfactory method is to join the proximal end of the 12th nerve to the distal end of the 7th, and the proximal end of the ansa hypoglossi to the distal end of the 12th cranial nerve.

The next great group are the metastatic tumors (6.6% in the series of Montreal Neurological Institute). These are generally found by the neurosurgeon in the cerebral hemisphere but may occur at any level. The usual source is from the lung.

A solitary secondary tumor of the hemisphere should be removed for relief of headache; the results are often remarkably good. The patient may recover completely for an indefinite period.

Pituitary Tumors

The pituitary adenomas (6.9% in the series of Montreal Neurological Institute) may be divided into the chromophil or acidophilic, the chromophobic, and the basophil or basophilic, which give characteristic syndromes. They all produce certain symptoms in common. Increase of intrasellar pressure causes headache which is usually bitemporal. When the adenoma escapes from the confines of the sella turcica there is relief from headache until the local pressure rises again or until there is an elevation in the general intracranial pressure. When the tumor is sufficiently large, pressure upon the optic chiasm results, first in an upper quadrant bitemporal or bitemporal hemianopia. Various degrees and combinations of field defect can occasionally occur, leading eventually to blindness in one or both eyes, according to the pattern of impingement upon the chiasm and the optic nerves.

Chromophil adenomas are composed mainly of acidophilic or eosinophilic cells containing alpha granules. This type of tumor en-

larges the sella turcica as it grows. It causes disturbances of growth hormones, which lead to gigantism and acromegaly.

Chromophobe adenomas are the most common type. The cells resemble the chromophobe cells of the pituitary. These contain few, if any, acidophil particles. They grow to a large size and soon rupture through the confines of

the sella turcica. They tend to produce symptoms of hypopituitarism.

Basophil adenomas are formed by a growth of the basophilic cells of the pituitary which contain specially staining beta granules. These tumors are generally very small and can be seen on cross section of the pituitary gland. They are associated, however, with very grave

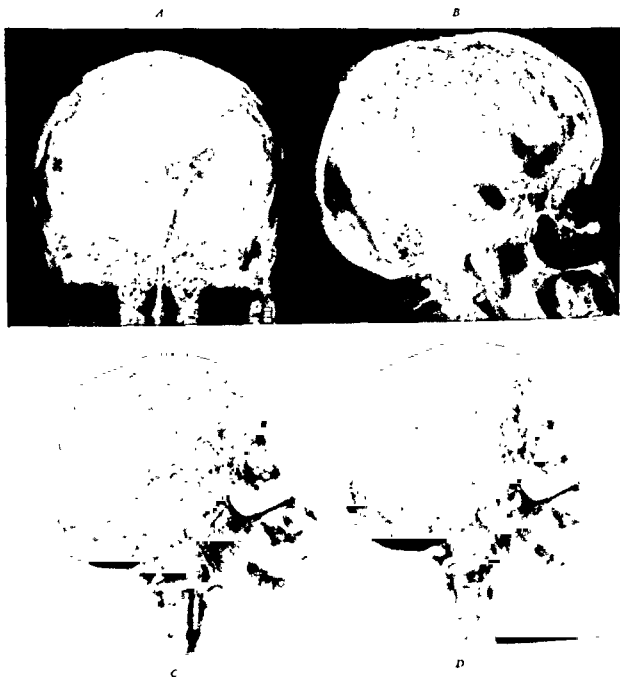


Fig 133 —A, Ventriculogram shows tremendous ventricular shift.
 B, Depression of lateral ventricle and large vascular channels within the skull
 C, Arteriogram shows arterial supply to the tumor and the vascular blush revealing the growth
 D, Vascular blush still visible in subsequent plate.

symptoms known as basophilism, characterized by polycythemia, hypertension, adiposity with pendulous abdomen and striae, hypertrichosis, amenorrhea.

Adenocarcinoma of the pituitary is a rare growth; biopsy and attempted removal are made.

When adenomas of the pituitary grow large enough to cause signs of compression of the optic nerves, operation is indicated. Before this, x-ray therapy can be employed if the indications are evident. The acidophilic tumors are generally considered to be the most radiosensitive and can be held in check to a considerable degree by roentgentherapy. Hormonal therapy has to be instituted as necessary.

failure of temperature regulation, somnolence, and disturbances of vision. They are slow in growth. Removal is very difficult and frequently one has to be satisfied with mere emptying of a cyst or with partial removal. Hypophysectomy in the treatment of cancer of the breast is described in Chapter 14.

Miscellaneous Tumors

Other rare congenital tumors of the brain are cholesteatomas, dermoids, teratomas, and lipomas. They occur in fairly characteristic locations and can generally be removed completely or at least partially.

Blood vessel tumors (4% of cases) include hemangioblastomas and hemangiomas. The

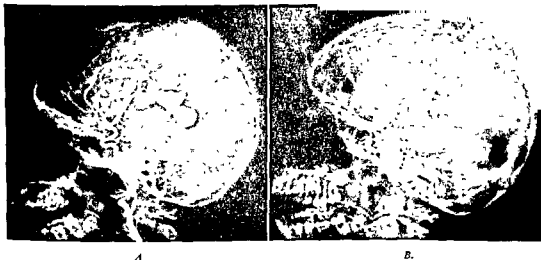


Fig. 134—A, Large arteriovenous angiomatic formation fed by main branches of the middle cerebral artery.

B, X-ray taken approximately 4 seconds later.

Epitheliomas of the hypophyseal duct arise from cell rests along the tract of the primitive craniopharyngeal duct or hypophyseal duct. They are composed primarily of epithelial cells in various types of formation with cystic spaces large or small which contain cholesterol crystals. There is usually a deposition of calcium, and the growths are often seen by x-ray. The symptoms are caused primarily from a disturbance of pituitary function and pressure upon the optic chiasm and hypothalamus. These consist of disturbances of growth and nutrition and metabolism, hypophyseal cachexia or Simmonds' disease, dwarfism, sexual infantilism, adiposogenital dystrophy, diabetes insipidus,

former are in gross appearance not unlike a glioma but on section are found to be formed of angioblastic or reticulo-endothelial elements. They are frequently associated with cyst and usually are located in the cerebellum. They are benign and if removed completely do not recur. Hemangiomas are difficult to differentiate from congenital anomalies of blood vessels and are probably such. The basic lesion is generally an arteriovenous connection. Cerebral arteriography is of paramount importance in the diagnosis of these lesions. Many of these formations can be removed with good results. They are prone to cause convulsions, headache, and sometimes hemorrhage.

Papillomas occur rarely from the choroid plexus and can be removed if producing symptoms. They occur in about 0.5% of cases.

Sarcoma of the dura is not common. *Meningiomas* sometimes take on sarcomatous tendencies.

Perithelial sarcoma of the brain (0.7% of cases) is not uncommon and usually occurs in the cerebral hemisphere. The treatment is removal and roentgentherapy.

Melanotic sarcoma may arise from melanoblasts in the leptomeninges.

Tuberculomas can be successfully removed if they produce symptoms. In some countries they are common.

Syphilomas rarely occur in this country and are prevented now by chemotherapy.

Echinococci cysts are common in some countries, they are treated surgically

THE VERTEBRAL COLUMN AND THE SPINAL CORD

The Vertebral Column

The vertebral column consists of 33 segments: 7 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 4 or 5 coccygeal. Both sacral and coccygeal segments have become fused into single bony masses known as the sacrum and coccyx, respectively. The movable vertebrae are separated by the fibrocartilaginous discs. They consist of a body and a laminal arch supported by two pedicles. The apex of the laminal arch protrudes as a spinous process.

The transverse processes jut laterally as extensions of the laminal arch and pedicle. There is a superior and an inferior articular facet on either side of a vertebra which affords a certain amount of movement; this varies for each and every vertebra. The vertebrae lie in series one above the other, their flattened superior and inferior surfaces separated by discs of fibrocartilage which allow some movement and also afford a resilient cushioning effect. The thoracic vertebrae, in addition, possess one or two facets for the head of the corresponding rib.

The vertebral bodies are held together by the strong annulus fibrosus of each disc and by the anterior and posterior spinal ligaments. Further, the vertebral spines are joined by strong interspinous and supraspinous ligaments.

The laminal arches are bound together by the elastic ligamenta flava.

When viewed from the side, the vertebral column exhibits a double S-shaped curve which is convex forward in the cervical and again in the lumbar regions and is concave forward in the dorsal and in the sacral regions.

The cervical vertebrae are the smallest and most movable. The upper two, the atlas and the axis, have a very special articular connection with the base of the skull and with each other, which permits the necessary movements of rotation in the transverse and the antero-posterior directions. Between the atlas and the axis, besides the arthrodial diarthrosis between the vertebral arches on each side, there is a special rotatory diarthrosis between the dens of the axis and the articular facet on the posterior surface of the anterior arch of the atlas. Several special ligaments bind the vertebrae together.

As the cervical vertebrae possess the greatest mobility, dislocation most readily occurs in this region. In the dorsal region there is greater rigidity, as it is supported by the thoracic cage. In the lumbar region there is moderate movement, and a common level for fracture is at the dorsolumbar junction.

The vertebral canal lies beneath the laminal arches and the intervening ligamentum subflavum. The floor is formed by the posterior spinal ligament which lies upon the posterior aspect of the vertebral bodies and the annular capsule of the intervertebral discs. The canal is narrowest in the dorsal region.

Related to each pedicle is a canal through which passes the corresponding nerve root. One root, the first, passes between the skull and the atlas. This means that the 1st cervical root passes from the vertebral canal above the first vertebra. Seven cervical vertebrae are described, but there are 8 pairs of cervical nerves, so that the 8th passes out below the 7th vertebra and the 1st thoracic nerve passes below the 1st thoracic vertebra, and all nerves below this level make their exit below the vertebra from which they are named.

The Spinal Cord

The cord is enclosed by three membranes. The inner consists of the pia mater spinalis, which is said to be somewhat thicker than the

which covers the brain but the function and anatomic relationships are similar. It carries within it the blood vessels for the spinal medulla. The arachnoid is a delicate membrane continuous with that covering the brain, and between it and the pia mater is the subarachnoid space. The inner layer of the cranial dura extends down to form the outer tough covering of the spinal cord.

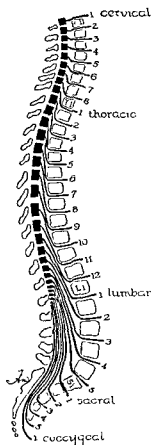


Fig. 135—Relative position of spinal nerve segments to vertebrae

As the nerve roots leave the vertebral canal, they carry a covering of these membranes which fuses with the connective tissue sheaths of the peripheral nerves on leaving the intervertebral foramina. Between each pair of nerve roots there is the dentate ligament which arises from the pia mater along the lateral side of the spinal cord. Below each root a sickle-shaped slip of this ligament passes laterally to form a pointed attachment to the dura. This occurs between each pair of nerves down to include the 1st lumbar.

The blood supply to the spinal medulla comes from the anterior and posterior spinal arteries and from spinal branches passing through the intervertebral foramina from the vertebral arteries and from the intercostal and the lumbar arteries. The venous drainage is by way of six longitudinal channels which form a plexus in the pia mater. This empties into the internal vertebral venous network which, in turn, empties into the vertebral, intercostal, and lumbar veins and into the cranial venous sinuses and plexus above.

Until the third month of fetal life the spinal portion of the medullary tube runs the full length of the vertebral canal. From there on, however, a discrepancy in rate of growth occurs, so that at birth the end of the spinal cord only reaches as far as the 1st lumbar vertebra. Because of this, nerve roots within the vertebral canal take a progressively increasing downward inclination. The lumbosacral nerves tail off below the conus of the spinal cord in what is known as the cauda equina. Puncture of the spinal subarachnoid space may be safely performed in the lumbar region below the level of the spinal cord.

In considering spinal localization, the relative position of the nerve segments and the spinal roots in relation to the vertebral column is of greatest importance. Thus the 5th lumbar nerve segment in the spinal cord lies at the level of the body of the 12th dorsal vertebra, but the nerve root runs all the way down to pass out of the vertebral canal between the 5th lumbar and the 1st sacral vertebra. A smaller discrepancy will occur as one comes to each succeeding higher level. The 1st lumbar nerve segment probably lies at the level of the body of the 10th dorsal vertebra; the 8th dorsal over the 6th dorsal vertebra, the 1st dorsal over the 7th cervical; the 4th cervical beneath the 3rd cervical, and so on. It must also be remembered that the vertebral spines in the dorsal region overlap considerably.

The paired nerve roots leave the spinal cord at regular intervals. Each pair of nerves which includes sensory and motor nerves, in addition to autonomic fibers in certain regions, supplies a certain area of skin and muscle. Such a region of supply is known as a spinal dermatome or nerve segment. The segmental innervation

of muscles is recorded in standard textbooks of anatomy.

The cutaneous areas supplied by each nerve segment have been mapped in considerable detail in man by Head and by Foerster; in the monkey by Sherrington, who employed the method of "remaining sensibility" in which three consecutive nerve roots above and three below a single intact root are sectioned. The area supplied by the remaining root can then be outlined and is the maximal for the intact root. The segmental dermatomal distribution in

for the appreciation of hot and cold sensations which are conducted in separate though closely packed laminae. Light touch is thought to pass largely by way of the contralateral ventral spinothalamic tract. Certain grades of touch also pass by the posterior columns.

Musculotendinous sensory impulses, as tested by joint position, two-point discrimination, appreciation of weight, figure writing, vibration sense, and such discriminatory functions, pass via the posterior columns of Gall and Burdach, or the fasciculi gracilis and cuneatus.

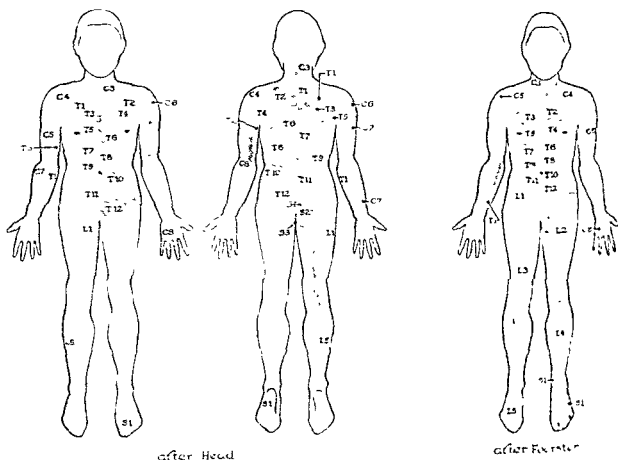


Fig 136—Segmental dermatomal distribution

man was studied in considerable detail by Head in patients with herpetic eruptions. Foerster recorded the areas of vasodilation after antidromal stimulation of the posterior roots and also mapped the dermatomal distribution by the method of remaining sensibility in clinical cases.

At this point, one may merely mention that conduction of painful sensory impulses passes up the spinal cord by way of the contralateral lateral spinothalamic tracts. This is true also

The autonomic pathways are not well known. The lateral corticospinal tract, subserving motor impulses, lies in the lateral funiculus immediately posterior to the rubrospinal tract. Impulses to the cerebellum from muscle, tendon, and joints pass by way of the dorsal and ventral spinocerebellar tracts.

Anatomic Localization

In spinal surgery it is of first importance to know the exact level at which the lesion is

situated, which is determined by neurologic examination.

The sensory level below which there is loss or reduction of sensation to painful stimuli may be determined. The sensation for hot and cold should be tested when more detailed examination is required; and in certain spinal lesions, a dissociation of involvement may be found. Similar testing of musculotendinous sensation must be made to determine the level of loss of function.

Autonomic disturbance may be judged by palpating the skin of the patient. The skin may be dry below the level of involvement because of the absence of sweating in the denervated areas, whereas above, it may feel moist. The presence of sweating may be demonstrated by placing the patient in a warm chamber and painting the skin with a preparation of iodine, and dusting starch powder over this. A blue coloration will occur in the presence of moisture. Other tests have been devised, and a very satisfactory method is relative measurement of skin resistance with the dermograph.

Motor power must be tested and relative weakness and paralysis listed, not forgetting the trunk musculature, for example, the intercostal muscles and the rectus abdominis. Spasticity, flaccidity, and atrophy have to be adjudged. In addition, the tendon reflexes, both deep and superficial, are examined. Thus, with proper examination, one may determine the level of the lesion by evaluating

- 1 Sensory levels
 - (a) pain, heat, cold, light touch
 - (b) muscle, tendon, joint sensation, i.e., position sense, two-point discrimination, figure writing, vibration sense
- 2 Motor levels
 - (a) muscle power
 - (b) muscle atrophy
 - (c) muscle tonus
- 3 Coordination by suitable tests
4. Autonomic levels
 - (a) palpation of sweating level
 - (b) sweating tests, chemical
 - (c) dermograph
- 5 Reflex level
 - (a) deep reflexes
 - (b) superficial reflexes

It must be remembered that damage to the spinal cord will cause a transitory abolition of deep reflexes below the level of the lesion. The plantar reflexes may vary according to the completeness of damage. This period is known as the period of spinal shock. In the lower animals it may last but a few hours or days but in man persists for several weeks. It is said to remain longer if infection or cachexia is present.

It may be mentioned here that if the spinal cord were hemisectioned, the motor paralysis by virtue of the crossed pyramidal fibers would be ipsilateral, as would be the loss of discriminatory sensation, but the loss to the modalities of pain, heat, and cold would be on the opposite side due to the crossing of the fibers below the lesion. This hypothetical type of neurologic manifestation is called the *Brown-Séquard syndrome*. It is not usual to have such a sharply defined lesion clinically, but frequently modifications of it are seen.

In addition to the above, which may give a fairly accurate level, there are some special signs which are of great importance and afford a regional localization.

- 1 Horner's syndrome
- 2 Paralysis of the diaphragm
- 3 Disturbance of bladder function and weakness of the anal sphincter

Horner's syndrome is dependent upon interruption of the autonomic sympathetic pathway at any point from the hypothalamus to the muscle controlling the pupil of the eye. The autonomic fibers pass out via the 8th cervical root and upward by way of the cervical sympathetic chain. The syndrome consists of miosis, enophthalmos, and slight ptosis. It is most common to encounter this sign in tumors at the level of C8.

It must be remembered that the diaphragm derives its main central nerve supply from the phrenic nerves, which emerge from or with the 3rd, 4th, and 5th cervical roots. It may also be mentioned here that the function of the intercostal muscles can be tested on simple palpation, and a motor level can be demonstrated, if present, in this way. The abdominal muscles can be palpated during the contraction. The umbilicus will move upward if muscle segments below it are weakened, i.e., Beevor's sign.

The bladder is supplied by sympathetic fibers from lumbar roots 1, 2, 3, (4) and by parasympathetic fibers from roots S 2, 3, 4. The parasympathetic fibers cause contraction

voluntary control is possible. Various effects upon bladder function are produced by injury to the spinal cord. This includes a phase of shock resembling spinal shock on the somatic

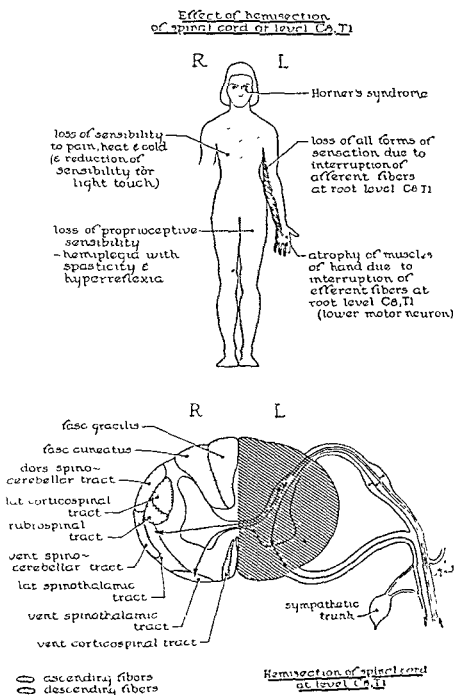


Fig. 137—Brown-Séquard syndrome

of the detrusor muscle and the pudendal nerve relaxes the external sphincter. Cerebral autonomic fibers pass upward and downward in the spinal cord, but these tracts are not as yet well known. Normally a certain amount of

side in which bladder tonus may at first be lost and subsequently increased to produce a hyperactive bladder of small capacity and of frequent emptying. At this juncture, it may be well to mention that ideally bladder function

in the paraplegic patient can best be treated in the initial stages, at least, by tidal drainage, which is an automatic syphonage system that rhythmically empties the bladder. After a period of treatment, it is felt that the bladder capacity will be greater than without this automatic device. In wartime it may be necessary to resort to suprapubic cystostomy.

A block below the thoracic level is likely to show reduction of respiratory oscillations, but fluctuations occur on pressure over the abdomen or on straining. A block at the lumbar level will show none.

In addition to the level of the lesion one may judge as to whether the lesion is intramedullary, intradural, or extradural, both by

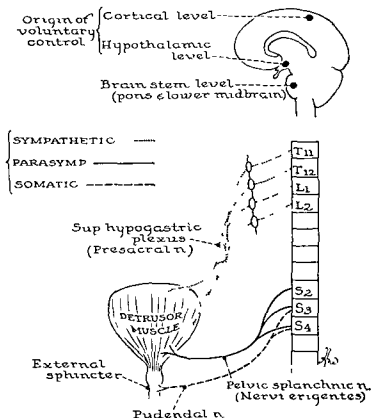


Fig 138—Pathways responsible for normal micturition. Diagram shows sympathetic, parasympathetic, and somatic nerve supplies of the bladder. Note that the parasympathetic fibers carry the impulses which are responsible for contraction of the detrusor muscle and that the pudendal nerve relaxes the external sphincter. The role of the sympathetic system in the act of micturition is somewhat indefinite.

Lumbar Puncture.—Localization of the lesion may be made very accurately by neurologic examination, which can be checked by lumbar puncture studies and by myelography. One may in fact determine by spinal manometric test not only whether a subarachnoid block is present but also to some extent the level at which it exists. Thus on jugular compression a block in the cervical, thoracic, or lumbar region will show an absence of response in the manometer below the block. A

block below the thoracic level is likely to show reduction of respiratory oscillations, but fluctuations occur on pressure over the abdomen or on straining. A block at the lumbar level will show none. In addition to the level of the lesion one may judge as to whether the lesion is intramedullary, intradural, or extradural, both by the history and by the neurologic examination.

An intramedullary lesion is generally painless, but an extradural and especially an extradural one will cause pain first. There will be more gradation of sensory loss and dissociation of modality in respect of pain, temperature, and touch sensation in the intramedullary type.

The vertebral canal can be considered to be divided to some extent by the dentate ligament into anterior and posterior compartments. Some

idea as to whether it lies anteriorly or posteriorly in the intervertebral canal can be adjudged by the neurologic examination. Thus a spinal lesion should be described as having its upper level at such and such a spinal nerve segment, and its lower level, which is more difficult to determine, estimated.

Lumbar puncture manometric study will give evidence of absolute or relative block. This will aid in the differential diagnosis regarding presence of an expanding lesion, adhesions, etc. Due to the realization that Pantopaque can be easily withdrawn at the end of the procedure, this test is generally combined or supplanted by myelography. The site of blockage can then be seen under the fluoroscope and upon x-ray films.

as spina bifida anterior. When a similar occurrence occurs in the skull, it is known as cranium bifidum. Experimental work, however, produced evidence to the effect that defects occur when an embryo is subjected to abnormal environmental conditions. It is believed that they might occur from faulty implantation as a result of endometritis in pregnancy.

These bony anomalies are generally associated with failure of the meninx to differentiate into its three characteristic layers. Logically and grossly it retains an embryonic type of structure, and in some cases the meningeal-like tissue is thick and spongy so that the pressure of the cerebrospinal fluid forces the meninges bulge through the skeletal



Fig. 139 Spina bifida myelocoele showing probable attachment of cauda equina beneath center of thin dermal covering.

Spina Bifida and Cranium Bifidum

Lesions Arising on the Basis of a Malformation.—The neural tube closes toward the end of the third week of intrauterine life in the human fetus. Mesoblastic tissue growing between the covering ectoderm and the separated neural tube forms the membranous and bony coverings of the entire nervous system. By the 11th week the vertebral canal is covered, roofed by the neural arches from the 1st cervical to the 3rd or 4th sacral vertebral level. Occasionally there is failure in the process of closure of the laminae, which results in an anomaly called spina bifida. Rarely this occurs anteriorly through the body of the vertebra, usually the sacrum, in which case it is known

in the form of a fluid-filled sac known as a meningocele. Often nerve roots and free spinal cord may be drawn into the sac, in which case it is then called a myelomeningocele. Various degrees of developmental failure of the nervous elements are seen in these cases. In the case of herniation of brain tissue in association with cranium bifidum the anomaly is called an encephalomeningocele or encephalocele. In severe spinal defects there is frequently and primarily maldevelopment of the myeloid element. The neural tube itself may have failed to close in which case there may be an associated failure of the meninges and vertebral coverings to close. Usually the laminae are widely open and the vert

pedicles are considerably separated. The meninges are thin and the skin like transparent parchment. One can see the undeveloped neural element or primitive spinal cord running sagittally as a double band along the inner side of the transparent covering. This represents the most complete type of defect, *spina bifida* with *myelocoele*, and is generally not suitable for surgery. It is sometimes called *rachischisis completa*.



Fig 140—Case of *spina bifida occulta* showing subcutaneous pad of fat and scar tissue dissected free, revealing stalk which enters vertebral canal

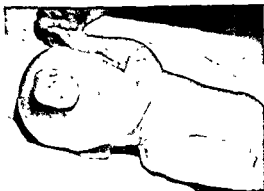


Fig 141—Cranium bifidum with meningocele

In the other extreme, very simple defects may occur through the laminal arch and in rare cases are hardly more than a notching of the laminal arch. In these there is generally a thin stalk which runs out from the level of the dura

into the subcutaneous tissues. The end of this is often enveloped in a ball of fatty and fibrous tissue which bulges beneath normal-appearing skin. This type is called *spina bifida occulta*.

Between these extreme examples are the *meningocele* and the *myelomeningocele* described above.

In rare cases there is an epithelial tract that runs from the skin surface to and into the dural sac and is continuous with an intraspinal epidermoid or dermoid cyst. This condition is recognizable as a button of thickened, discolored skin, usually in the midlumbar region, with a small, central dimple which marks the external opening of the dermal tract. The area may be surrounded by a light growth of hair. These lesions occasionally occur in the skull, usually involving the posterior fossae.

Spina bifida occurs occasionally in more than one member of a family and occasionally in successive generations. A family history was admitted in 65% of cases by Ingraham, et al. Occasionally more than one *meningocele* presents in the same patient.

Surgical cases of *spina bifida* and *cranium bifidum* may be classified as follows:

- 1 *Spina bifida occulta*
- 2 *Spina bifida* with *meningocele*
- 3 *Spina bifida* with *myelomeningocele* or *myelocoele*
- 4 *Spina bifida* with *syringomyelomeningocele*
- 5 *Cranium bifidum* with *meningocele*, *encephalocele*, or *meningoencephalocele*
- 6 *Spina bifida* associated with epithelial sinus and dermoid

Symptoms and Signs

In the case of *spina bifida occulta* there may be no neurologic defect. On the other hand, there may be weakness and wasting of the muscles of one leg and sensory loss and disturbance of bladder function. The signs probably arise as a result of traction on the spinal roots or spinal cord as a result of anchorage by the filum terminale or nervous and fibrous attachments. There is, of course, no emergency about operating upon this type, and patients may not be referred for treatment until adolescence, when complications have become more

Actual muscle weakness may be present as can be demonstrated by asking the patient to pull or push against resistance while testing dorsiflexion and plantar flexion of the foot and of the toes. A useful test is to ask the patient to stand on his heels and on his toes; this will reveal weakness of the flexor muscles on the one hand and of the extensor muscles on the other. The ankle jerk is generally hypoaffective or absent in an L5 to S1 herniation, but less likely to be lost in an L4 to L5 protrusion. The knee jerk is usually reduced in an L3 to L4 rupture. One may see fibrillation or fasciculation in some of the involved muscles.

The Lasègue sign is generally positive when there is clinical evidence of disc disease in the lower lumbar region. When the thigh is flexed at the hip, pain occurs when the leg is extended on the thigh, supposedly due to stretching of the sciatic nerve or a part thereof which causes radiation of pain. In some cases there occurs only pain in the lower back and hip. On the contrary, flexion of the hip alone without extension at the knee, will not cause pain. This is helpful in making a differentiation between a spinal lesion such as a ruptured disc and a lesion about the hip joints. The sign can be intensified by dorsiflexion of the foot. In exceptional cases when the test is negative, the Lasègue sign can be modified by the raising of both legs together. This practically always gives a positive result. Tenderness over the lower lumbar spine in the gluteal region, posterior thigh, calf, and the Achilles tendon can usually be elicited.

A useful test is to allow the patient to lie on his back with legs hanging over the end of the table or side of the bed. This generally causes increase of pain which can be relieved by raising one or other or both legs to the horizontal. Deviation of the legs to the right and to the left may also then accentuate the pain.

In the standing position it will generally become obvious that the patient has some degree of scoliosis, tilting of the pelvis, lumbar muscular spasm, and/or reduced lordosis and may be unable to straighten up. The patient protects the leg on the affected side by flexing the knee. In acute conditions this is sometimes quite extreme. Bending forward may increase

the scoliosis and muscular spasm, with limitation of movement which will be obvious and frequently causes kinking and locking. Certain movements produce low back pain with some degree of sciatic radiation. Bending backward and to the left and to the right will generally cause some of the above subjective and objective phenomena.



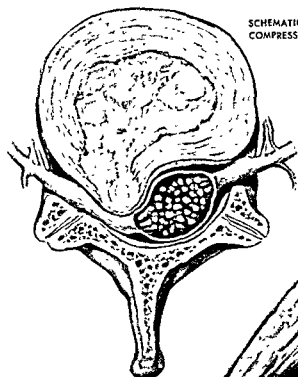
Fig. 142—A, Myelogram shows almost complete block at the level of L1-5 interspace.

B, Shows marked spurring at the 3rd, 4th, and 5th interspaces.

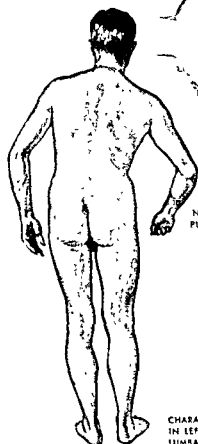
C, Myelographic defect shows protrusion at level L1-5 on the right.

D, Myelogram shows protrusion at L1-S1 on right and evidence of bilateral protrusions at the L1-5 level.

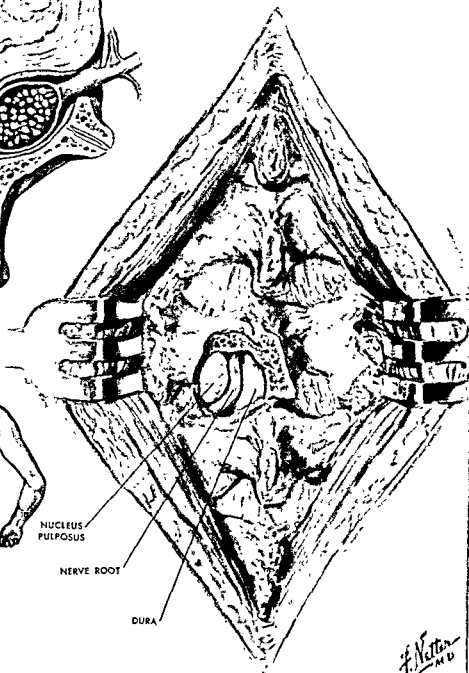
Radiologic Examination.—Simple x-rays at the required level may show narrowing of the disc shape with some osteophytic reaction. Increase of protein is often found in the cerebrospinal fluid. Roentgenologic examination with the aid of radiopaque substances is desirable when possible. An air or an oxygen myelogram had for a time some popularity but has been superseded by Lipiodol, which can be aspirated



SCHEMATIC CROSS-SECTION SHOWING
COMPRESSION OF NERVE ROOT



CHARACTERISTIC POSTURE
IN LEFT SIDED, LOWER
LUMBAR DISC HERNIATION



SURGICAL EXPOSURE OF LOWER LUMBAR HERNIATION



from the spine following the study, and more recently by Pantopaque, which is thinner and can be aspirated more easily. Up to 9 ml. of Pantopaque is injected, and the needle remains in situ during fluoroscopy and the taking of films. At the completion of these studies the material is removed by aspiration. The material should be run as high as the thoracic region and can be carried on to the cervical if indicated.

Diagnosis.—Clinical examination and the myelogram should be in agreement, and when this is the case, operation can be considered. When they do not coincide, the question of

as well as the more uncommon granulomas. It is not necessary to prolong the list of other possibilities.

Treatment.—Treatment resolves itself into nonoperative and operative. In *nonoperative* treatment bed rest alone can effect marked improvement. Plaster jacket for some six weeks, possibly in a position of slight flexion, or in any event in a comfortable position, may cause a temporary cure. It is, however, probably not much better than simple bed rest. Application of heat, massage, and exercises may be of value. If conservative measures fail, operative treatment may be indicated.

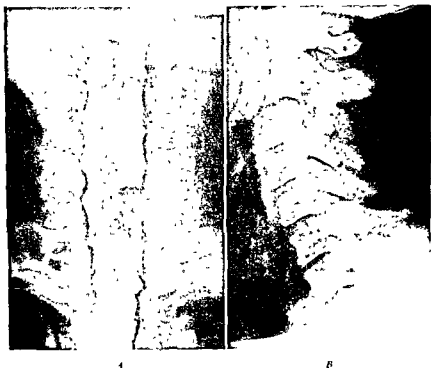


Fig. 143—A, Myelogram shows defect at the C6-7 level on the right side, suggesting large herniation of the intervertebral disc.

B, X-ray shows narrowing of the intervertebral space and slight spurring at the C6-7 level.

operation has to be left to the experience of the individual surgeon. In differential diagnosis one has to consider spondylolisthesis which, of course, can be diagnosed with the x-ray and sometimes even on palpation. It may also be associated with a disc herniation. Tumor of the cauda equina, metastatic lesions, and chordoma of the vertebral column may confuse the picture. A tumor of the sciatic nerve is a rare possibility. Tuberculosis must be considered,

The question as to whether the disc should be removed and whether simultaneous fusion should be carried out has been a source of discussion between neurosurgeons and orthopedists. In the author's opinion spinal fusion is not necessary as long as the disc has been radically removed. It is possible that some degree of soft tissue fusion will take place if the disc is radically removed, and it has been shown by Cone and Rabinovitch that when the

disc is completely removed, including the cartilaginous plate in animals, actual bone fusion takes place. Because of the amount of separation between the human vertebral bodies, bony fusion, in the author's opinion, is not likely to occur. It is necessary to remove sufficient bone from the laminal edges for good exposure and especially from the posterior aspect of at least the entrance to the nerve root canal in order to decompress the nerve root satisfactorily. It is important to explore adequately for the disc as on some occasions it may point more medially or more laterally and in rare instances lie between the nerve root and the dura further caudal than one would expect, and occasionally it migrates a short distance up or down the vertebral canal.

In carefully selected cases the results are satisfactory in at least 80% of patients. In some the leg pain disappears while they may be left for a period of time with a feeling of weakness, stiffness, or occasional backache. This, no doubt, may be due to mechanical factors but, frequently, merely to muscular spasm. It can usually be cured by suitable exercises. In the case of lumbar discectomy the author allows the patient bathroom privileges on the day following operation but otherwise prefers to keep the patient in bed for 8-12 days.

Cervical Discs

Diagnosis.—The herniated *cervical disc* presents a special problem. The signs and symptoms are similar, except for the location, to those found in the lower lumbar region, giving rise to attacks of neck pain with radiation down the arm, sometimes with numbness and a feeling of paresthesia and of weakness with atrophy. The most common site for the hypaesthesia is over the radial distribution, generally root C6 or C7, coming from a disc at the level of C5 to C6 and C6 to C7, respectively. Tilting the head passively to one side and jerking it slightly will frequently elicit pain in the side of the neck which may or may not radiate. There is often a point of tenderness over the transverse process of C6. X-rays of the cervical spine show a narrowing of the disc with some spurring of the bone. The normal cervical curvature is altered, and limitation of movement can be seen by po-

sitional x-rays. The diagnosis is further confirmed by myelography.

Treatment.—The results in cervical disc removal are very satisfactory. Operation in this area is more hazardous than in the lumbar region because of the proximity of the cervical spinal cord. For this reason conservative treatment should be given a very adequate trial before operative interference is considered. A collar of felt or plaster can be worn to support the chin. Bed rest for a short while and linear extension by halter traction can also be tried. It is highly important that the nerve root be adequately decompressed in the procedure of cervical discectomy, which should be undertaken only by an expert in the surgery of the central nervous system. The results in this type of operation are highly satisfactory.

Thoracic Discs

Herniations at other levels are generally non-surgical and may fail to be recognized, as they do not cause pain due to the relative position of the root in respect to the disc. However, on some occasions, they do, in which case symptoms and signs will correspond to the level involved. In the *thoracic region* they may cause compression of the spinal cord with resulting degrees of paralysis and loss of bladder function. When such is the case and when the localization is exact, operative removal is indicated. The level again can be checked by myelography. However, one must guard against artefacts of myelography, especially in the thoracic region. Operation in the thoracic region is particularly difficult. Some cases of cervical and thoracic disc disease have simulated multiple sclerosis.

Fracture-Dislocation of the Spine With Neurologic Involvement

The general plan of procedure has been to send the patient as rapidly as possible by ambulance or airplane to a hospital that is sufficiently well equipped and is best suited for dealing with these cases. This is true both in military and in civilian practice.

The diagnosis must be suspected at the time and at the site of the accident by rapid examination of the patient. The history, the clinical signs of pain or tenderness, spasm, paresthesia, anesthesia, and paralysis should deter-

mine the diagnosis. If the patient is unconscious one has to rely upon the immediate history of witnesses and the objective signs. Palpation of the cervical spine may reveal loss of proper alignment, ecchymosis and swelling may be present over the dorsolumbar spine.

During transportation, if conscious, the patient must remain flat on his back for cervical fracture and his head must be braced or held between sandbags or kept in linear extension. For lumbodorsal fracture, lying prone on a stretcher may be desirable, preserving normal dorsolumbar curves, or supine on a firmer surface if the patient is conscious. Much will depend upon the state of respiration, as naturally a good airway is essential. The unconscious patient is transported more safely lying prone.

On arrival at hospital, neurologic examination, ecchymosis, swelling, sensory level, paralysis, etc. should at once give an idea of the site of the fracturing. These will give an indication of the most important areas to be x-rayed. Distention of the bladder must be controlled by catheterization. Further treatment will depend on the type of fracturing, the neurologic signs, and the presence or absence of complicating factors such as multiple injuries, etc.

If cervical fracture-dislocation has occurred, one must consider the following surgical procedures:

Gradual reduction of the dislocation by linear traction of the spine. This can be carried out by one of three methods:

1. Halter or chin strap traction
2. Skull traction by chrome steel wire passed through burr holes or trephine holes
3. Skull traction with tongs
 - a. Crutchfield
 - b. Barton-Cone

The first method is not satisfactory. It is painful and cannot be sustained long. The second is unnecessarily complicated but is a good method. The third is simple and is satisfactory and comfortable. With these last two methods the patient can be properly nursed and turned safely, and an operation can be performed while the patient is in traction.

After reduction by the skull traction one must decide whether an open operation is de-

sirable. It is preferable to explore and fuse in any case where complete dislocation has occurred or where multiple fractures have occurred, usually of the body and of the laminal arches. Such an operation is made while the traction apparatus is in place. The operation consists of (a) *exploration* and (b) *reduction*, which is often complete when the surgical wound is exposed. One may if necessary open the dura mater a short distance to view the condition of the cord and the vertebral canal for encroachment by bone or fibrocartilage. The dura is then repaired. One can elevate any bone which presses in from the laminal arch.



Fig. 144—Skull traction by linear extension, using the Cone Barton ice tongs.

When the wound is satisfactory, an internal fixation is made by a fusion operation, usually using parallel grafts of bone from the tibia or the ribs. Os purum has also been tried and homologous bone from the bone bank is now commonly used. After the stitches are removed, a suitable plaster cast can be applied and the patient allowed up.

Similar procedures are carried out for dorsolumbar fractures, but here linear skeletal traction is difficult. In simple cases of fracturing of the body without neurologic signs, the usual

reduction by hyperextension can be used, but when the fracture is more complicated, includes the pedicles and arch, and especially if neurologic signs are present, this maneuver is dangerous as it may cause a narrowing of the vertebral canal. In these cases, open operation, exploration, decompression, repair of dura, and replacement of roots of cauda equina, when necessary, should be carried out. This may be

done with shoulder and foot traction apparatus applied during the operation and with some degree of hyperextension to correct the bony deformity. Cone has developed special apparatus for this purpose. In addition, a spinal fusion is employed to hasten recovery by strengthening the spine. The operative method of treatment is preferable in most of these cases, but if this is not easily possible, the

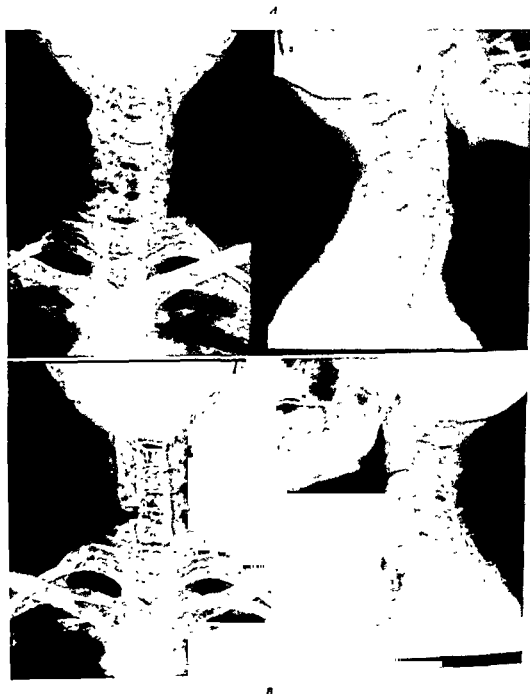


Fig. 145—*A*, X-rays demonstrate fracture-dislocations at the level C1-2.
B, Spinal fusion after reduction by skeletal traction of fracture-dislocation at the level C1-2.

absolute indications for operation might include a condition in which a narrowing of the vertebral canal has occurred, such as by a depression or displacement of a portion of the laminal arch or body, which has caused a degree of paralysis. Satisfactory x-rays are important in making this decision. This can be

checked by lumbar puncture and a Queckenstedt test to determine whether a spinal block is present. The absolute indications to justify operation for such cases cannot be properly defined in so short a space as too many variables are possible, and procedure will depend on the findings in each particular case.



Fig. 146—*A*, Severe fracture-dislocation of the first lumbar and adjacent vertebrae.
B, Spinal fusion with parallel tibial bone grafts after exploration and decompression under skeletal traction.

Of greatest importance is the attention to the urinary bladder. This should not be allowed to become distended. Initial catheterizations should be done at suitable times, followed as soon as possible after operation by the establishment of an automatic bladder. It may require many weeks before this can be established. In the meantime, tidal drainage is employed, but if this is not possible (as it requires considerable care on the part of the surgeon) one must rely upon catheterization and, if necessary, suprapubic cystostomy. After a certain time it should be possible to stop continuous catheterization and employ manual pressure every 2-3 hours, at regular intervals catheterizing for the residual. In this way the patient may develop a reflex emptying bladder. If it is not at first successful, this can be tried again after another week or so.

For the various grades of paraplegia the problem of late convalescence and rehabilitation has become more hopeful in the last few years due to the research of Munro, the School for the Crippled and Disabled in New York, and of various Centers for paraplegia in Canada and the United States which have been developed since World War II. The patient ideally should be sent to a rehabilitation center, but this is not essential. He must learn to strengthen the muscles of the shoulder girdle so that he will eventually be able to use crutches and perhaps learn to walk in splints. He can learn a gainful occupation, learn to drive an automobile, have a wheel chair, etc., and especially to look after himself, his bladder, his bowels, and to dress himself. In the words of Helen Barton, "he must learn to live again," so that he will become self-reliant and self-supporting.

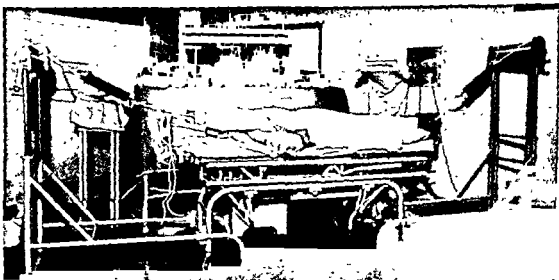


Fig. 117—The Cone operating table with traction apparatus applied

Of equal importance is the prevention of bedsores by sufficient turning and massage of the patient over the important areas. If they develop, further care is necessary. Plastic surgeons may prefer to repair with skin flaps. If this is not desirable, they can be healed by proper cleansing, débridement, and nursing care.

Spasticity may become a troublesome complaint. It may be minimized by adequate physiotherapy. More radical methods of dealing with this symptom will not be discussed

Infection of the Spine

Tuberculous infection of the spine is more properly treated as an orthopedic condition and is discussed in that section. Only when signs of paraplegia develop from pressure of tuberculous granulation tissue or pus is the neurosurgeon involved. In such cases a decompressive laminectomy and spinal fusion are indicated.

Nontuberculous and acute pyogenic involvement of the spine may occur as an osteomyelitis, in which case it may have arisen as a

complication of a surgical procedure. Exposure and packing with antibiotics are usually necessary. In the case of spontaneous infection, it has been possible to effect a cure with antibiotics alone without operation. Rare chronic infections such as blastomycosis occasionally occur.

Another form of spinal infection takes place as an epidural abscess. This condition arises spontaneously, apparently as a result of a metastatic infection lodging in the epidural tissues. This gives rise to general signs of acute infection to which are added spasticity of spinal muscles and consequently some arching of the back, together with marked tenderness and pain which may be girdlelike. Any movement causes great pain. If unrelieved, spinal compression occurs with paraplegia. The treatment is open operation, packing, and the use of antibiotics.

Tumors of the Vertebral Column

Neoplasms of the vertebral column will cause pain and eventual compression of the spinal cord. Some may be listed as follows:

A. Benign

- 1 Benign giant cell tumor
- 2 Chondroma
- 3 Osteoma
- 4 Hemangioma
- 5 Myeloma

B. Malignant

- Primary—1 Osteogenic sarcoma
 2 Lymphosarcoma
 Secondary—1 Chordoma
 2 Metastatic carcinoma

Each cannot be discussed in detail. Suffice it to say that the ideal treatment for *benign giant cell tumor* is as complete removal as possible followed by deep x-ray therapy. In the *primary tumors*, decompressive operation, biopsy, and attempted removal are desirable, followed by deep x-ray therapy.

The remaining tumors are removed so far as possible when they produce symptoms or signs.

Paget's disease is sometimes associated with sarcoma. This sometimes calls for decompression and removal. Decompression is also indicated when simple compression occurs in *Paget's disease*. X-ray treatment is very effective in some cases of sarcoma of the spine.

Secondary carcinoma of the spine becomes a neurosurgical problem when it causes pain or paraplegia. Pain can be relieved by suitable spinohalamic tract section, paraplegia can be prevented by decompressive laminectomy, with partial removal of the neoplasm, and the segmental pain can be relieved by simultaneous extradural root section.

Tumors Arising Within the Vertebral Canal

Tumors arising within the vertebral canal may be classified in the same manner as brain tumors. They fall naturally into a group of tumors arising from the meningeal coverings of the spinal cord and the nerve roots, and another which grows from and within the spinal cord itself. It may be pointed out that there is also a group of extradural tumors that are listed above under tumors of the vertebral column. In addition to these conditions already mentioned, Hodgkin's disease should be added; the lymphogranulomatous tissue usually lies in the epidural space with some attachment to the outer layer of the dura. Surgical removal may be necessary.

Epidural tuberculous granulation tissue may be encountered and is generally treated by antibiotics and orthopedic measures.

Parasitic cysts should also be mentioned, though rare in this country.

Tumors Arising From the Meningeal Coverings.—The *meningeal fibroblastoma* is a benign tumor which arises from an attachment to the dura. This neoplasm probably originates from arachnoidal cells within the dura. It is usually reddish yellow, discrete, firm, attached to the dura, finely lobulated, and encapsulated. Meningiomas occur mostly in the dorsal region. This tumor must be removed completely with the involved dura. If this is done, there will seldom be a recurrence. This may be technically difficult. The author generally lays in a fascial graft or a split dural graft. Some leave the dura open.

Perineurial fibroblastoma arises from the epineurium and perineurium of a nerve root, usually the posterior root. These tumors are generally intradural. They are yellow, firm, discrete and benign and must be removed completely and the nerve root probably sacri-

ficed. The nerve fibers spread out over the capsule of the growth and can be dissected free. If removal is complete, there will be no recurrence.

A *neurofibroma* has the general appearance of the above though it is somewhat whiter and firmer and the nerve fibers pass into the mass rather than through its capsule. There will be no recurrence if removed completely. There may be multiple *neurofibromas* and other evidence of von Recklinghausen's disease.

results, but in the more malignant types, and if paralysis is severe, the result will only be relative. *Medulloblastoma* of the cerebellum commonly metastasizes to the spinal meninges.

Syringomyelia is sometimes associated with tumor, in which case it is preferable to call it tumor with cyst, granted the cyst may traverse a considerable distance and even the full length of the cord. Here the treatment is as for tumor. *Syringomyelia* proper occurs without tumor and is usually thought to be asso-



Fig. 148—Stages of removal in case of perineurial fibroblastoma at the level of L2. Note next attachment to right of retractor.

Tumors of the Spinal Cord.—Tumors of the spinal cord itself consist mainly of the gliomas. The more common are the astrocytomas and the ependymomas and occasionally glioblastoma multiforme and spongioblastoma polare. These tumors call for exploration. They can be removed with very satisfactory

results. In some cases exploration and incision of the cyst are indicated to relieve signs of pressure and possibly pain. *Syringomyelia* is associated in some cases with a congenital anomaly at the craniospinal junction in which the odontoid process is tilted backward and impinges on the anterior surface

of the spinal cord and medulla. Decompressive operations have been performed for this condition. In some the condition is confused with a dilated neural canal which is known as a hydromyelia of the spinal cord.

Other Rare Conditions.—Other rare conditions include *lipoma* of the cord, which is generally irremovable without causing much disturbance of function. *Spinal dermoids* are difficult to remove, but with careful dissection complete removal is often possible and prognosis may be good.

Improvement results if the area of adhesion is small and delicate, arachnoiditis circumscripta, but often this process is extensive, and it is difficult to obtain regression of signs even with meticulous dissection.

PERIPHERAL NERVES

The nerve supply of the upper extremity is derived from the anterior rami of the 5th, 6th, 7th and 8th cervical and the 1st dorsal nerve roots. In the so-called pre-fixed plexus there is a contribution from the 4th cervical

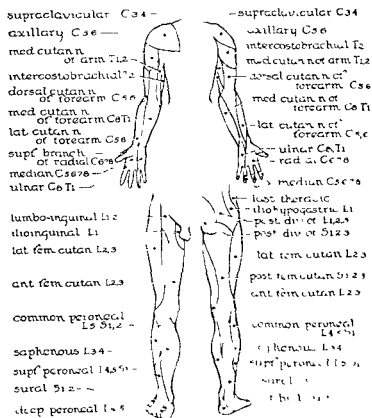


Fig. 149.—Peripheral nerve sensory skin areas

Tuberculomas of the spinal cord rarely come to operation.

Angiomas of the spinal cord may be encountered.

Subarachnoid parasitic cysts should also be mentioned though rare in this country.

Adhesive arachnoiditis is a rather uncommon condition in which a dense layer of adhesions is discovered involving the pia arachnoid membrane. Sometimes postoperative im-

provement results if the area of adhesion is small and delicate, arachnoiditis circumscripta, but often this process is extensive, and it is difficult to obtain regression of signs even with meticulous dissection.

The anterior spinal nerve roots are composed of the axonal processes of nerve cells situated in the anterior columns of the gray

matter of the spinal cord from which they emerge as rootlets from the region of the anterolateral sulcus. The posterior spinal roots are formed by the proximal axonal processes of cells in the ganglia of the posterior spinal root which lie at the level of the intervertebral foramina. The anterior and the posterior roots pierce the dura separately. Thence they share a common sheath which includes the spinal ganglion of the posterior root. The distal processes of the posterior root ganglia and of the anterior roots continue to share a common sheath and become, by definition, spinal nerves as they leave the intervertebral foramina.

Due to regrouping in the various nerve plexuses the distribution of the peripheral nerve fields will necessarily differ from that of the sensory root dermatomes. An approximation to the general representation is given in Fig. 150.

Posterior Cervical Plexus

The posterior cervical plexus as distinct from the cervical plexus is constituted by communicating branches from the posterior rami of the 1st, 2nd, and the 3rd, and possibly 4th cervical nerves. The greater occipital nerve

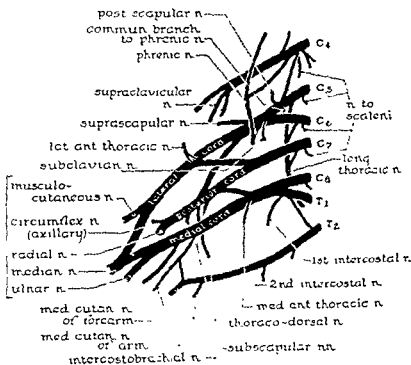


Fig. 150—Brachial plexus

On leaving the intervertebral foramina the spinal nerves divide into primary posterior and anterior branches. The posterior rami innervate the skin of the head and trunk posteriorly and the longitudinal muscles of the spinal axis. The anterior branches supply the whole lateral and anterior parts of the body, including the limbs, and form the great plexuses. The spinal nerves each are joined by at least one gray ramus from the paravertebral sympathetic ganglionic chain. Each anterior root, from the 1st dorsal to the 2nd and 3rd lumbar segments, supplies one white ramus communicans to the sympathetic chain with efferent fibers.

arising from the posterior ramus of the 2nd cervical nerve supplies sensory fibers for the posteromedial half of the occipital scalp. The 3rd occipital nerve supplies the skin more laterally and comes from the 3rd posterior ramus. The 1st cervical nerve does not generally contain sensory fibers, and motor branches are of little surgical importance.

The occipital nerves are sometimes injected with Novocain or alcohol in cases of occipital neuralgia. Seldom is this necessary. Generally these cases are psychogenic. The greater occipital nerve can be injected most readily 1" lateral to the greater occipital protuberance at the

level of the superior nuchal line. It can be localized more accurately by faradic stimulation.

Cervical Plexus

The cervical plexus is formed by the anterior rami of the upper four cervical nerves. The sensory fibers pass by way of the lesser occipital nerves (C2, 3), great auricular nerve, nervus cutaneous colli, and descending supraclavicular branches (C3, 4) to supply the skin of the lateral third of the occipital scalp, the side of the neck, and the area of skin overlying the angle of the mandible bounded above by the distribution of the trigeminal nerve. The peripheral nerve fields and the dermatomes which they supply are of importance, principally in diagnosis. Thus, in a cervical fracture dislocation at the C2, C3 level one may find an anesthesia in the area supplied by the lesser occipital and the greater auricular nerves.

From a surgical point of view the most important motor branch arising in part from the level of the cervical plexus is the *phrenic nerve* which contains fibers from the 3rd, 4th, and 5th anterior rami of the cervical spinal nerves. Passing obliquely across the scalenus anticus muscle the nerve must be identified and isolated in the operation for scalenotomy. Phrenicotomy with avulsion of the phrenic nerve has been used in the treatment of tuberculosis in order to collapse the lung by immobilizing the diaphragm. Regeneration seems to take place in a few months. Paravertebral growths or direct trauma may affect the phrenic nerve, and examination should include both observation and fluoroscopic examination of the diaphragm.

Brachial Plexus

The brachial plexus is formed from the anterior rami of the 5th, 6th, 7th, 8th cervical and the 1st dorsal nerves. In some cases the 4th cervical nerve contributes a branch and in other instances the 2nd dorsal sends a branch.

The nerves undergo a reassortment as they pass downward and laterally in the posterior triangle of the neck toward the first rib over which they pass into the axilla. Three primary cords are formed. The *upper cord* is formed by union of the 5th and 6th nerves. The *middle cord* is represented by the 7th alone

and the *lower cord* by the 8th cervical and 1st dorsal nerves. At the same time the nerves divide into anterior and posterior trunks. Regrouping results in the formation of three secondary cords. The *lateral cord* is formed from the anterior trunk of the 5th, 6th, and 7th nerves, a *medial cord* from the anterior trunk of the 8th cervical and the 1st dorsal (part), and a *posterior cord*, to which all of the posterior trunks from the 5th to the 1st dorsal, inclusive, belong.

The secondary cords pass over the first rib in the subclavian groove surrounding the superior, posterior, and inferior aspects of the subclavian artery between the scalenus anticus and the scalenus medius muscles.

Anatomic localization is important in the case of lesions in the neighborhood of the brachial plexus and is made possible by a knowledge of the special anatomy of the region.

Supraclavicular lesions with nervous involvement at and proximal to the level of the primary cords are likely to involve certain nerves to muscles of the shoulder girdle and trunk and may be looked upon as supraclavicular.

Supraclavicular branches may be listed as follows (Cunningham):

Anterior branches:

- 1 Nerves to scalenus anterior and longus colli
- *2 Communicating nerve to join the phrenic nerve
- †3 Subclavian nerve

Posterior branches

- 1 Nerves to scalenus medius and scalenus posterior
- *2 Dorsal scapular nerve
- *3 Long thoracic nerve
- †4 Suprascapular nerve

A lesion of the brachial plexus at a lower point will allow the above branches to escape. A lesion further distal toward the secondary cords will develop the aspect of a multiple peripheral nerve lesion. The *lateral anterior* and the *medial anterior thoracic nerves*, the *subscapular nerves*, and the *thoracodorsal nerve* arise from the secondary cords and may be involved.

*From anterior rami of the plexus

†Level of formation of secondary cords

The following branches arise from the infraclavicular portion of the brachial plexus (Cunningham):

Anterior branches from the lateral cord:

1. Lateral anterior thoracic
2. Median (lateral head)
3. Musculocutaneous

Anterior branches from the medial cord:

1. Medial anterior thoracic
2. Median (medial head)
3. Ulnar
4. Medial cutaneous nerve of forearm
5. Medial cutaneous nerve of arm

Posterior branches from the posterior cord:

1. Axillary nerve
2. Radial nerve
3. Two subscapular nerves
4. Thoracodorsal nerve

Lesions below the level of the secondary cords will have the characteristic distribution of peripheral nerves to the arm and forearm.

Surgical Lesions.—These may be divided anatomically into those affecting:

1. Spinal roots and primary cords and branches adjacent to the vertebral column
2. The supraclavicular portion at the point of maximal mingling in the posterior triangle of the neck
3. The infraclavicular portion at the level of the secondary cords and branches

Diagnosis and anatomic localization must be made by examination of the particular muscles involved and sensory loss and sympathetic nervous involvement.

Lesions may be traumatic, neoplastic, inflammatory, and trophic.

Lesions of the three primary cords or trunks may be divided into three types:

1. The upper cervical or the Duchenne-Erb type
2. The middle type
3. The lower or the Duchenne-Aran type

The *upper radicular* or *Duchenne-Erb* type results from a lesion of the anterior rami of the 5th and 6th cervical roots or the upper primary cord to the brachial plexus. If the damage is close to the vertebral foramina, the dorsal scapular nerve, the long thoracic nerve and branches to the scalene muscles, and the

longus colli may be affected. If the breach occurs farther distal, these branches are spared whereas the suprascapular nerve and the subclavian nerve may be involved, together with the subscapular nerves and the lateral anterior thoracic nerves. Peripheral nerves to the arm involved may include the axillary and part of the radial fibers from the lateral head of the median and the musculocutaneous.

Muscles in the arm and forearm which derive their total or main supply from cervical nerve roots C5 and C6 are the biceps, brachialis, brachioradialis, pronator teres, flexor carpi radialis, palmaris longus, supinator. According to level of section there may be, in addition, paralysis of the rhomboidei, levator scapulae, and serratus magnus. There may be palsy of the supraspinatus, infraspinatus, teres minor, and deltoid.

The resulting deformity consists of adduction of the arm, internal rotation, extension at elbow and pronation. Conversely the patient cannot flex the arm at the elbow, supinate the forearm, externally rotate or abduct the arm. The characteristic deformity and atrophy will be diagnostic. The arm hangs with the palm of the hand rotated backward.

A lesion of the *middle primary cord*, which is composed of the 7th cervical nerve, causes principally extensor paralysis of the upper extremity in addition to paralysis of certain muscles about the shoulder and trunk. The deformity in the forearm is similar to that of a radial nerve lesion except that the brachio radialis may escape. Muscles involved are latissimus dorsi, subscapularis, teres major, triceps (part), the muscles supplied by the radial nerve, and, in addition, the nerve to the coracobrachialis. The type of deformity and treatment are similar to those for the musculospiral nerve (Stookey).

The *lower radicular palsy* (*Duchenne-Aran*) occurs with damage to the 8th cervical and 1st thoracic nerves. This gives rise to a paralysis of muscles supplied by the ulnar nerve and the medial head of the median nerve. Muscles involved are flexor carpi ulnaris, flexor digitorum profundus, flexor digitorum sublimis, flexor pollicis longus, and the intrinsic muscles of the hand. Abductor pollicis and opponens pollicis may escape since they may receive fibers from C5, C6 roots (Stookey).

The result is atrophy of the muscles along the ulnar side of the forearm and marked wasting of the small muscles of the hand. Paralysis of adduction and weakness of flexion at the wrist occur with paralysis of flexion, adduction and abduction of the fingers, extension of the distal two phalanges, opponens action, and abduction of the fifth digit. There will be a cutaneous sensory loss for C8 and D1. In addition, a Horner's syndrome may result from implication of the sympathetic rami



Fig 151—Case of lower radicular palsy from traction injury. Complete paralysis of the muscles of the hand and loss of sensation along the medial side of the limb. Horner's syndrome was present.

In trauma various combinations of the above syndromes may occur. A certain lesion, however, may simulate an upper, middle, or a lower cervical lesion, a supraclavicular or infraclavicular syndrome. In the adult civilian, involvement of the upper roots is most common in injuries which forcefully separate neck from shoulder, as of a weight falling upon the shoulder, thus stretching and tearing the upper roots and nerves forming the plexus. It occurs commonly from birth injury.

The lower roots are less frequently involved but may be injured at birth if the arm is delivered in a vertical position or in an adult who catches an object with his hand to break

a fall. In injuries that cause stretching, nerve fibers may be torn from a distance, even from the spinal cord itself. Thus satisfactory suturing may be impossible. In wartime, injuries from missiles are common. Such may include severance, contusion, and concussion of the nerves.

Radial Nerve.—The radial nerve derives its fibers from the posterior divisions of C5, 6, 7, 8, and D1. Injury to the radial nerve in the arm causes paralysis of brachioradialis, triceps, extensors of wrist, fingers, and thumb. Thus paralysis is associated with wristdrop and loss of extension at the elbow and at the metacarpophalangeal joints of the thumb and fingers. This deformity should be corrected with cock-up splints. In addition, a characteristic sensory loss will be observed.

It may be injured in the axilla by fractures and dislocations of the humerus, by the pressure of a crutch, or by abnormal compression during sleep. It is frequently damaged in fracture of the shaft of the humerus and in gun shot wounds and other direct injuries.

Median Nerve.—The median nerve takes its origin from the 5th, 6th, 7th, and 8th cervical and the 1st dorsal roots by way of the outer and the inner heads. Muscles supplied are opponens pollicis, the outer head of the flexor pollicis brevis, abductor pollicis brevis, flexor digitorum sublimis, flexor pollicis longus, flexor carpi radialis, palmaris longus, pronator quadratus, pronator teres, lateral two lumbricals.

Disability results from involvement of flexion at wrist, loss of flexion of the thumb and index finger, paralysis of flexion at the proximal interphalangeal joints of all digits, loss of opponens action, and weakness of abduction of the thumb and of pronation of the forearm. Sensory loss occurs over palm and volar surface of thumb, index, middle finger and lateral half of the fourth finger and dorsal surfaces of terminal phalanx of index and middle fingers.

Ulnar Nerve.—The ulnar nerve is derived from C8, D1 by way of the inner cord of the brachial plexus. Muscles involved include flexor carpi ulnaris, inner mass of flexor digitorum profundus with the 3rd and 4th lumbricals, adductor pollicis, flexor pollicis brevis (deep part), volar and dorsal interossei, the

opponens and abductor digiti quinti. The result of paralysis is a flattened, narrow hand due to wasting of the small muscles, with the fourth, fifth, and, to some extent, the third fingers extended at the metacarpophalangeal joints and flexed at the proximal phalangeal joints, giving rise to a "clawhand" deformity. In addition, there is evidence of wasting along the ulnar side of the forearm, and the hand is in a position of radial deviation. Flexion and adduction of the wrist are weakened. Flexion at the metacarpophalangeal joints of fourth and fifth and, to some extent, of the third digit is lost. Flexion at the terminal

phalanges and extension at the phalangeal joints of these digits is also affected. Adduction and abduction of the fingers are lost. There is inability to abduct and oppose with the fifth digit and to adduct with the thumb.

Circumflex Nerve.—The circumflex nerve arises from the 5th and 6th cervical nerves. It supplies the deltoid and the teres minor muscles, the shoulder joint with sensory fibers, and the dermis over the deltoid region. It may be involved by crutch palsy and fracture-dislocation of the head of the humerus. A lesion of this nerve results in the appearance of flattening of the shoulder, due to atrophy

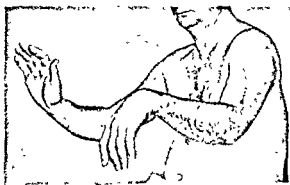


Fig. 152—Wrist drop from radial nerve palsy, the result of fracture of the humerus



Fig. 153—Exposure in middle third of the arm, showing extensive thickening of radial nerve, which has been freed from dense adhesions in the neighborhood of a fracture of the humerus which had been plated. Return of function occurred in a matter of weeks. Same case as seen in Fig. 152.



Fig 154—*A*, Case of typical deformity resulting from injury to the ulnar nerve immediately above the elbow

B, Atrophy of the small muscles of the hand and inability to adduct or abduct the fingers

C, Atrophy and inability to extend the 4th and 5th digits at the phalangeal joints

D, Patient fails to oppose the 5th digit to the thumb

of the deltoid muscle. The peripheral disability is due to loss of abduction of the arm.

Musculocutaneous Nerve.—The musculocutaneous nerve supplies the biceps and the brachialis muscles and transmits sensory fibers to the cutaneous areas over the radial side of the forearm and hand. The nerve to the coracobrachialis muscle is generally associated with it. Loss of flexion at the elbow is the result of involvement of this nerve. In certain positions some flexion is possible through the action of the brachioradialis. Injury may be from fracture or gunshot wound and compression.



Fig 135—Neuroma in continuity Ulnar nerve at the wrist.

Neuroma Formation

When a nerve has been left unsutured, the growing axonal processes from the central end cannot make contact with the lower segment and consequently grow into a fusiform swelling mixed with proliferating connective tissue from the nerve sheaths. This is known as a *neuroma*. Some thickening of the traumatized end of the lower segment also occurs from

mesodermal proliferation. When the nerve has not been anatomically severed one obtains a *neuroma in continuity*. The isolated processes in the peripheral segment undergo rapid degeneration, leaving the sheath of Schwann intact. When the two ends of the freshly cut nerve are brought into light apposition, the axon processes from above are directed by some chemotactic mechanism to enter the sheaths of Schwann below and down to reinnervate the part. Many fibers do not succeed in entering the degenerated nerve, but grow outside it, or enter the wrong sheaths, and lose their function.

Primary Suture of Nerves

Injury to peripheral nerves must be diagnosed through adequate history and physical findings in the light of an adequate knowledge of anatomy. In traumatic severance of a nerve,

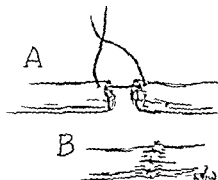


Fig 136—Nerve suture

immediate suture should be done. The severed ends of the nerve may be cut clean with a very sharp blade and are laid close together in a normal position. Suturing should be carried out so that the upper point on the circumference of the cut central end corresponds as nearly as possible with the corresponding point on the upper pole of the distal segment. In this way the best possible approximation of the funiculi which may be seen within the cross section of the nerve may be hoped for. The severed ends are drawn together by interrupted sutures, which are passed through the nerve sheath.

It is possible to rotate the nerve, as necessary, with these sutures. Sutures are placed at equidistant points through the sheath round the circumference of the nerve. A large nerve

may require several and a small nerve two or three sutures. The two ends of the nerve sheath are drawn together so that they are in contact. The nerve fibers under these optimal conditions should barely touch.

The suture material is very fine silk or tantalum. Specially prepared sutures fused to needles are available. It is important to handle the nerve gently and as little as possible. Such manipulations as are necessary must be carried out by manipulating the sheath. In suturing, only the sheath should be held by the forceps. In early suture a satisfactory result can be expected. If the practitioner does not feel able to suture the nerve at the time of the accident, the wound may be débrided and allowed to heal and the patient sent along later for a secondary suture.

Secondary Suturing of Nerve

When a nerve lesion has gone unrecognized or when exploration has been deliberately delayed in order to watch for possible spontaneous recovery, which has not materialized in a reasonable time, a secondary suture can be made. This will entail excision of the neuroma and suturing. The extent of the neuroma on the proximal end and of the scarring in the distal segment may be judged by inspection and gentle palpation. If the two ends of the severed nerve are far apart it may be difficult to bridge the gap. In this instance the distance may be shortened by flexing the neighboring joint such as the elbow or the knee joint. Following suture the joint must be maintained in flexion by plaster fixation. After 1-2 weeks the cast may be bivalved. Passive exercise can be carried on every day, and a second cast may be applied with greater extension. After three weeks it is possible for the joint to be fully extended. It is important that passive exercise be given from the start to those parts not limited by the cast, for example, to the fingers and wrist when the elbow is fixed by a cast.

A common method to effect shortening of the distance to be bridged is that of transposition of the nerve. The ulnar nerve may be transplanted anterior to the medial condyle of the humerus. The long end of the nerve is passed beneath the flexor digitorum sublimis and pronator teres muscles and then su-

tured. In making such a transplant one must consider the branches which arise at the level of transposition. In this case the branches to the flexor carpi ulnaris and to the flexor digitorum profundus may have to be sacrificed. It may be possible to bridge the gap merely by transposing the nerve anterior to the condyle and the flexor muscles. In this instance the branches to the flexor carpi ulnaris and to the flexor digitorum profundus can be spared.

Signs of returning function should be noted: return of sensation by testing light touch and pain sense, recovery of muscle tone and power by palpation, observation of the finest movements, and by disappearance of wasting. Return of function can be more precisely evaluated by the technique of electromyography when equipment is available.

Treatment of Lesions of Brachial Plexus

Traumatic lesions of the brachial plexus are difficult to treat and are likely to be discouraging in end results. Accurate diagnosis and localization are essential to satisfactory procedure. In civilian practice trauma to various parts of the brachial plexus generally occurs without open laceration. In this instance history and careful neurologic examination have to be relied upon for diagnosis. It is usual to allow a certain time to elapse to decide whether regeneration is going to occur. If obvious severance has taken place, exploration and repair should be done at once. In some instances signs of recovery may appear without necessity of exploration. Nonoperative treatment consists of affording relaxation to the nerve trunks by elevation of the shoulder girdle, with flexion at the elbow. This can be accomplished by some form of airplane splint. In simple cases a sling may be sufficient. In lower plexus lesions corrective splinting has to be devised. Suitable support and physiotherapy should be instituted to prevent muscular fixation at the shoulder.

In birth injury (Erb's) it is usual for recovery to take place over weeks and months without operation. But diagnosis must be accurate. Sufficient immobilization and relaxation to the shoulder must be maintained. This can be accomplished by securing the child's wrist to a soft cap or to the top of the bed.

Supraclavicular Compression Syndrome.—See Chapter 33, *Peripheral Vascular Diseases*.

Cervical Rib.—In order to understand this syndrome it is necessary to visualize the particular anatomic relationships that exist at the exit of the supraclavicular triangle. Looking from the side, the anterior and the middle scalenus muscles have the shape of a tent, through the door of which pass the secondary cords of the brachial plexus and the subclavian artery. The threshold is normally formed by the subclavian groove of the first rib.

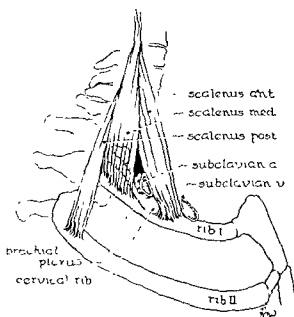


Fig 157—Supraclavicular compression syndrome—cervical rib

The subclavian artery passes over the rib in the angle immediately behind the attachment of the anterior scalene muscle. Arranged about its posterior, inferior, and superior surfaces are the secondary cords of the brachial plexus. The medial cord, containing the elements of the ulnar nerve and the contribution of the medial segment to the median nerve, lies upon the first rib immediately behind the artery. Above it in order lie the posterior cord and the lateral cord. Immediately posterior to the cords of the plexus is the scalenus medius muscle. Normally there is just sufficient room for the roots and artery to pass; in fact, some muscular individuals are able to obliterate their pulse by contracting or tightening their scalene muscles.

In some individuals the muscles appear to be so well developed and so placed as to cause unusual compression at the exit of the supraclavicular triangle, thus causing symptoms and signs of nerve and of arterial compression. This was recognized by Adson as a syndrome many years after similar symptoms and signs were known to occur in the presence of cervical ribs. When a cervical rib is present the above portal of exit is even more crowded. A complete cervical rib will run all the way forward and will join the first rib just beneath or just behind the artery. It usually ends in a protruding bony hump which may lie beneath the artery and nerve or just behind them. This may exert just enough extra pressure to cause symptoms. In addition, the extra



Fig 158—Wasting of interosseous muscles. Case of cervical rib with involvement of medial secondary cord of brachial plexus

rib tends to hold the scalenus medius rigidly forward. In this way the nerves and artery are subject to unusual compression. In incompletely formed ribs there may be a continuing band to the first rib, though this must be uncommon. In other cases where only a short rib or none is visible, the tendinous band or muscular edge of the scalenus medius muscle may give pressure on the nerves.

Thus the secondary cords of the brachial plexus may be compressed in a variety of ways, each of which may produce a slightly different clinical pattern. By its musculotendinous edge the scalenus medius may play an important part in the compression syndrome to affect principally the posterior secondary cord. On the other hand, the usual ulnar involvement occurs because these fibers pass directly over

the first rib or the end of the cervical rib and are more easily damaged in the above conditions

Clinical cases may be divided into groups as follows, according to the manner in which the structures are affected

1. Patients with nerve pain
2. Patients with hypesthesia—usually ulnar, rarely radial
3. Patients with muscular wasting and weakness, usually ulnar
4. Patients with circulatory disturbances, even leading to gangrene
5. Patients with autonomic disturbances, sweating, acrocyanosis

From an anatomic point of view one might classify these cases as follows

1. Cases in which there is no cervical rib, condition presumably due to muscular hypertrophy (scalenus anticus) or the general anatomic arrangement
2. Cases due to compression by the musculotendinous border of the scalenus medius
3. Cases of cervical rib, complete, incomplete, and rudimentary with band

Treatment in clearly defined examples is generally operative. Simple section of scalenus anticus may be successful for all types. However, modifications are necessary in certain cases. The author has used the following techniques in the different types and advocates removal of the extra rib when present

1. Section of scalenus anticus
2. Section of sharp musculotendinous border of scalenus medius along with section of scalenus anticus
3. Removal of cervical rib, including the mass at its point of junction with the first rib, beneath the artery and nerve cords, with section of the scalenus anticus
4. Section of rudimentary band

Lumbosacral Plexus

The lumbar plexus is formed by the anterior branches of the first three lumbar nerves and a portion of the fourth. Occasionally there is a branch from the 12th thoracic

The 1st lumbar nerve gives rise to the iliohypogastric and the ilio-inguinal nerves. The genitofemoral arises from branches of the 1st and 2nd lumbar nerves. The lateral femoral

cutaneous nerve of the thigh arises from the 2nd and 3rd lumbar nerves. Irritation of this nerve in the inguinal region from external pressure and scarring may cause pain along the anterior surface of the thigh. This condition is called *meralgia paraesthetica*. It can be relieved by injection or resection of the nerve or by stopping the cause.

The obturator nerve is formed from the anterior portions of the 2nd, 3rd, and 4th lumbar nerves. It is sometimes injured in pregnancy. It is sometimes sectioned in order to relieve adductor spasm.

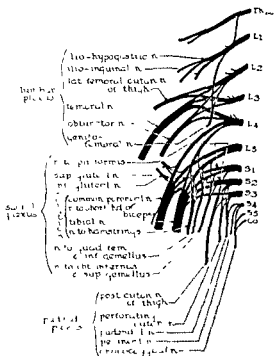


Fig. 159—Lumbar, sacral, and pudendal plexuses

The femoral nerve is formed from the posterior aspect of the 2nd, 3rd, and 4th lumbar nerves. This nerve supplies the quadriceps femoris, sartorius, pectineus, and the iliopsoas muscles.

Paralysis causes inability to flex the thigh and to extend the knee. Sensory involvement gives loss over anteromedial aspect of thigh and leg. Particular branches supply the hip and the knee joint. The femoral nerve is rarely injured but may be involved in a pathologic process, especially in the paravertebral region

The sacral plexus arises from the anterior rami of a part of the 4th lumbar nerve, the 5th lumbar, the 1st sacral, and portions of the 2nd and 3rd sacral nerves. Branches from the sacral plexus are divided into anterior (ventral) and posterior (dorsal).

They may be listed as follows (Cunningham)

Anterior branches:

1. Tibial nerve
2. Nerves to the hamstring muscles
3. Nerves to the quadratus femoris
4. Nerves to the gemelli
5. Nerves to the obturator internus
6. Articular branches (hip)

Posterior branches

1. Common peroneal nerve
2. Nerves to the short head of the biceps
3. Nerves to the piriformis muscle
4. Superior gluteal nerve
5. Inferior gluteal nerve
6. Articular branches (knee)

The *sciatic nerve* is composed of the following:

1. Nerve to the hamstring muscles
2. The tibial nerve
3. The common peroneal nerve
4. The nerve to the short head of the biceps muscle

The *sciatic nerve* is derived from the descending branch of the 4th and of the ventral and dorsal divisions of the 5th lumbar, the 1st, 2nd, and 3rd sacral roots.

The *common peroneal* and the *tibial nerves* are the principal terminal branches. The common peroneal divides into three terminal branches:

1. Recurrent tibial
2. Deep peroneal
3. Superficial peroneal

The first supplies the proximal fibers of tibialis anticus muscle, the tibiofibular articulation, and the knee joint. The deep peroneal nerve supplies the tibialis anticus, extensor hallucis longus, extensor digitorum longus, peroneus tertius, extensor digitorum brevis; the 1st, 2nd, and 3rd interosseous muscles, articular branches to the ankle and the small joints of the foot, and the dorsal cutaneous area at the base of the great and second toes

and their adjacent surfaces. The superficial peroneal nerve supplies the peroneus longus and peroneus brevis muscles and the skin over the dorsum of the foot and a portion of the toes.

The tibial nerve supplies branches to the two heads of gastrocnemius, plantaris, soleus, popliteus, tibialis posterior, flexor digitorum longus, flexor hallucis longus, flexor digitorum brevis, flexor hallucis brevis, abductor hallucis, quadratus plantae, abductor digiti quinti, flexor digiti quinti brevis, interossei, adductor hallucis, and lumbrical muscles. Cutaneous fibers supply the plantar surface of the foot and contribute the main supply to the sural nerve for the posterolateral portion of the lower leg, heel, and lateral border of the foot, ankle, and tarsal joints. In addition, fibers from the tibial nerve supply branches to the knee joint, the tibiofibular joint, and the tarsal and metatarsal joints.

The *sciatic nerve* may be involved in neoplastic growth in the pelvis, at the sciatic notch, and along its course. Some possibilities may be listed: aneurysm at the sciatic notch, gravid uterus, sarcoma of the upper end of the femur, and trauma, as in forced manipulation for dislocation of the hip joint, contusion by fracture, open wounding, and gunshot wounds.

Complete involvement of the sciatic nerve causes paralysis of all muscles in the leg and the foot and partial paralysis of the muscles of the thigh and produces anesthesia of the foot and the lateral surface of the leg. The general practical result will include a steppage type of gait with complete footdrop. Autonomic disturbances with lack of sweating and edema of the foot also may be noted.

The common peroneal nerve is more vulnerable than the tibial. In addition to the usual forms of trauma and gunshot injuries, damage occurs as a result of pressure on the nerve, for example, in crossing the legs and pressure from casts. It results in a paralysis of dorsiflexion and eversion of the foot, extension of the toes at proximal phalangeal joints, and anesthesia of the skin of the lateral surface of the leg and dorsum of the foot.

In paralysis of the common peroneal nerve, homologue of the radial nerve in the arm, the knee is lifted and the foot swung forward

in such a manner as to clear the ground. The patient cannot dorsiflex his foot and toes from the ground.

The tibial nerve is not frequently injured except in war casualties. Trauma results in paralysis of plantar flexion of the foot and toes, of flexion and separation of the toes, and in sensory loss over the sole of the foot and the dorsal surfaces of the terminal phalanges of the toes.

In paralysis of the tibial nerve, homologue of the median and ulnar nerves in the arm, the subject cannot raise the heel from the ground or stand on the toes

The various lesions are again treated by primary suture when possible or by resection of the neuroma and suturing. Appliances to protect the paralyzed muscles from prolonged overstretching and the institution of properly supervised physiotherapy are of the greatest importance at all stages of treatment

Pudendal plexus (3rd subdivision of lumbosacral plexus) derives its fibers from anterior rami of first three sacral nerves and all of the anterior rami of the 4th and 5th sacral and coccygeal nerves. Muscles supplied by nerves from this plexus are the levator ani, coccygeus, external sphincter, transversus perinei superficialis and profundus, ischio-cavernosus, bulbocavernosus, sphincter urethrae membranaceae. Cutaneous nerves supply the perianal region, perineum and genitalia, and the posterior aspect of the thigh. Autonomic fibers are dealt with elsewhere

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Chapter 12

Diseases of the Face, Mouth, and Neck

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Diseases of the face, mouth, and neck should ordinarily be detected early in their course and treatment instituted before serious or permanent damage has resulted. Unfortunately, this is not always the case. Infections and neoplasms in these locations are not infrequently allowed to progress to an advanced stage and may even end fatally before being recognized. Lesions in this region should be immediately apparent in most cases either to the eye or the touch or both. These two senses constitute the most valuable means of detecting abnormalities of this region, and with the aid of a few simple instruments such as laryngeal and nasopharyngeal mirrors, headlight, tongue depressors, and finger cots, the examination can be thorough and complete. Many cases of cancer are missed in their early stages simply because the examiner did not carry out a thorough and systematic examination of this region, including the base of the tongue, tonsillar region, pharynx, larynx, and nasopharynx. More important, he may neglect to carry out a careful digital palpation of the various structures in the neck and oral cavity. It is surprising how often a metastatic cancer in the neck, or a cancer in some part of the oral cavity, can be better felt than seen. Therefore each student should practice on every patient the methods of executing a thorough examination of this region until he becomes adept and develops confidence in his examination. Only then will serious lesions in this region be detected and treated at an early stage.

THE FACE

The discussion of congenital anomalies and traumatic lesions of the face, including burns and scalds, has been omitted because these are included in Chapter 10, Plastic Surgery.

INFECTIONS

The face is prone to all infections that attack the skin in general. We can divide infections of the face into two main groups: the acute and the chronic.

Acute Infections

Acute infections of the face include the following conditions: furuncles, carbuncles, cellulitis, erysipelas, and impetigo contagiosa.

A furuncle or boil is a painful circumscribed inflammation of the skin and subcutaneous tissue caused by the *Staphylococcus pyogenes*. The bacteria enter through the hair follicles or sudoriferous glands. Furuncles are particularly common on the upper lip and in the nasal region. In any serious infection of the face there may be associated edema of the face and eyelids. The main danger of a facial infection is that, if virulent, and if the patient's resistance is low, the infection may spread along the course of the facial, angular, and ophthalmic veins to produce a thrombosis of the cavernous sinus, with marked morbidity and mortality.

A carbuncle generally starts as a furuncle and may occur anywhere on the face. The

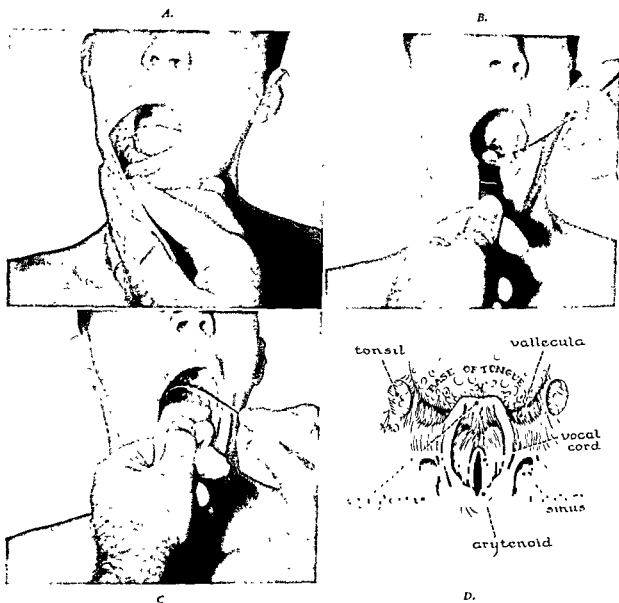


Fig 160 — Inspection of oral cavity, pharynx, and larynx

A Inspection should include a systematic examination of the lips, teeth, alveolar ridges, buccal mucosa, Stensen's duct orifice, hard and soft palates, the floor of the mouth, tongue, tonsils, pharynx, and larynx

B, Indirect nasopharyngoscopy The tongue is depressed with a retractor, and the patient is asked to breathe through both his mouth and nose. One should see the nasal septum in the midline and the middle and inferior turbinates and eustachian tube orifice on either side

C, Indirect laryngoscopy The examiner gently pulls the tongue forward, and the patient is asked to breathe through the mouth

D, Diagram showing the main structures to be visualized in the larynx and hypopharynx, as viewed in *C*

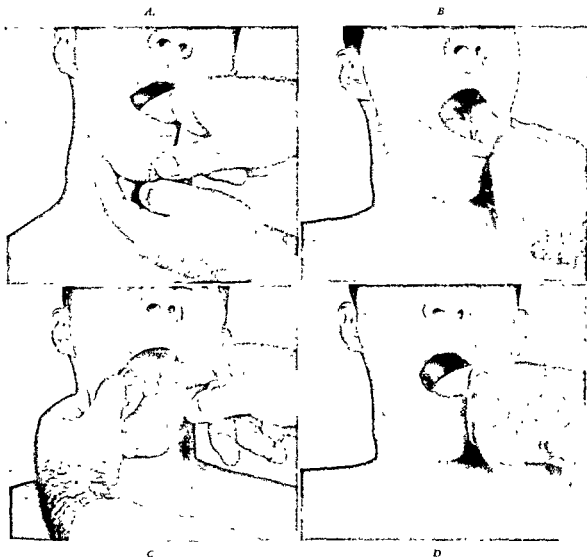


Fig 161 —Palpation of the oral cavity

A, Bimanual palpation of the floor of the mouth and of the submaxillary and submental regions

B, Palpation of the buccal mucosa, cheek, and distal portion of Stensen's duct

C, Palpation of the anterior and lateral portions of the tongue

D, Palpation of the posterior portion or base of the tongue, the tonsillar region, and the hypopharynx

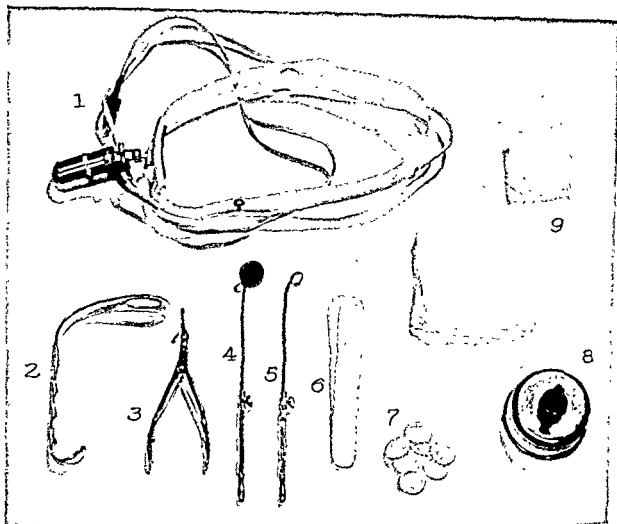


Fig 162.—Simple equipment necessary to carry out a thorough examination of the head and neck 1, headlight, 2, tongue depressor, 3, nasal speculum, 4, laryngeal mirror, 5, nasopharyngeal mirror, 6, wooden spatula, 7, rubber finger cots, 8, alcohol lamp, 9, gauze

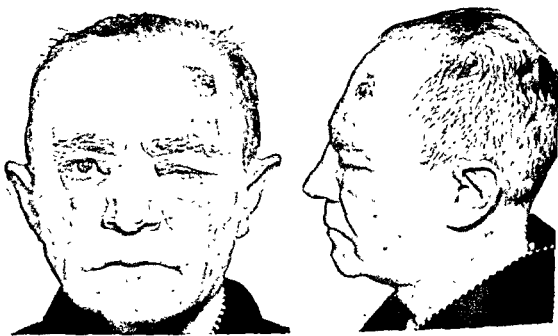


Fig 163.—Carbuncle on forehead with secondary swelling about orbit and eyelids

causative organism is again the *Staph. pyogenes* which is of enhanced virulence, and there is an associated systemic factor, such as lowered resistance in the patient or the presence of diabetes. The lesion is characterized by an inflammation of the subcutaneous tissues and multiple areas of necrosis of the skin which rupture to produce sinuses that discharge pus. There is pain with tenderness and redness of the area and marked constitutional symptoms. Enlargement and inflammation of regional lymph nodes accompany the infection. More rarely, invasion of the blood stream may occur with septicemia and distant metastatic abscesses.

Cellulitis is an inflammation of the loose subcutaneous tissue characterized by a diffuse and spreading induration. Such a lesion may respond to therapy, with restoration of the tissues to their normal state, or the process may undergo suppuration and localize to form an abscess.

Erysipelas is an acute inflammation of the skin and occasionally of the mucous membrane, caused by a *hemolytic streptococcus*. According to Boyd, the site of the inflammation is the face or scalp in 90% of the cases, and the infecting organism is probably derived from the nose and nasal sinuses. It is characterized by an intense local redness and tenderness of the skin and by marked constitutional symptoms. The margins of the affected area of skin are elevated and somewhat indurated, vesicles or blebs may be present. In the more severe cases, the subcutaneous tissues are involved and suppuration may take place.

Treatment of Acute Infections—The danger of acute facial infections has been reduced since the introduction of the antibiotics. However, such infections should still be treated by the physician with great respect. In general, in dealing with these pyogenic infections, conservative measures are preferred, and surgical intervention is used only when there is definite abscess formation. At first hot moist fomentations containing a saturated solution of magnesium sulfate are applied as nearly continuously as is practical, and their application is continued until a fluctuant center develops or until the process has subsided into a subacute phase. Minor incisions to drain softened abscesses may be necessary. The concomitant use of a suitable antibiotic is essential.

Impetigo contagiosa is a troublesome pustular skin infection caused by staphylococci. It is contagious at all ages but especially so in children. The infection can run through a school or a children's ward, attacking many. It first appears as small vesicles on the face or body or about a wound. These vesicles soon become pustular, and the pustules rupture and coalesce with other sores to form crusted patches. If untreated the lesions become widespread.

In treatment, great care is taken to prevent spread of infection to other areas by protecting the sores from scratching and handling. Personal contact with others and the common use of towels must be avoided, and careful destruction of dressings is necessary. The crusts should be softened and removed by hot saline soaks, and a detergent such as pHisoderm with G-11 should be used several times daily. After removal of the crusts the involved skin should be treated by applications of bacitracin or neomycin ointment or with 3% ammoniated mercury ointment. Systemic antibiotics are usually unnecessary and, furthermore, may cause sensitization. This may deprive the patient of a valuable form of systemic treatment at a later date. In more resistant cases of impetigo that do not respond satisfactorily to local therapy, the systemic use of one of the wide-spectrum antibiotics may be necessary.

Chronic Infections

Chronic infections of the face include acne, syphilis, tuberculosis, actinomycosis, and blastomycosis and are, in comparison with acute infections, rather infrequent.

Acne generally begins at the age of puberty and is common in both sexes. The lesions are commonly found on the face but are also seen on the back and chest. It is a chronic inflammatory disease of the sebaceous glands, most frequently due to a staphylococcus. The acne may be aggravated by improper cleansing, cosmetics, or the squeezing and pressing of pimples or comedones. The inflamed glands form small red pustules which may progress to furuncles and carbuncles.

Actinomycosis and tuberculosis are fully discussed in the sections on the Neck.

Cutaneous blastomycosis is a chronic inflammatory infection of the skin characterized

by the formation of various-sized wartlike patches having papulopustular nodules. The disease is caused by a yeast organism, and the majority of cases occur in men over the age of 40 years. There is a systemic form of the disease which is generally fatal. The traditional method of treatment is a saturated solution of potassium iodide as outlined for actinomycosis. The use of stilbamidine intravenously in a dose of 150 mg daily for 12 days is promising. In resistant cases x-ray therapy is of benefit.

NEOPLASMS

Benign Tumors

Sebaceous Cysts

The most frequent sites of sebaceous cysts on the body are, respectively, the scalp, the face, and the neck, in which areas sebaceous glands are most numerous. The cysts are caused by obstruction to the duct of a sebaceous gland and therefore contain a mixture of desquamative epithelium and sebum. While the cysts may appear in any part of the face or scalp, they are particularly common behind the ears. Sebaceous cysts of the scalp are called *wens*. The cysts vary in size from 4-5 mm to 5-6 cm in diameter and are in close contact with the overlying skin. When not infected, they have a relatively tough cyst wall. As the cyst enlarges the skin is stretched, and infection may supervene to form a red, tender, and fluctuant abscess or a persistent discharging sinus. Rarely, a cyst may open to the skin surface, where the sebaceous secretion becomes dried as a *cutaneous horn*. Such cutaneous horns may develop malignant changes in the base of the cyst.

Treatment.—Sebaceous cysts should be removed for cosmetic reasons, because they may become the sites of recurring infections and because a small number develop malignant changes. Small clean cysts are best excised and closed by primary suture. When removing a sebaceous cyst, it is desirable to remove a small ellipse of attached skin with the cyst. This facilitates the dissection and lessens the danger of breaking into the cyst wall. Malignant cutaneous horns should be widely excised; if benign, more conservative excision of the horn and underlying cyst will be sufficient.

An infected sebaceous cyst should be treated by incision, drainage, and antibiotics. Complete excision is postponed until the infection has completely subsided.

Senile Keratosis

This is a degenerative disease of the skin most frequently seen on the forehead, face, or neck of elderly people. The lesion begins as a small, superficial, brownish discoloration resembling a freckle. Later it becomes raised, wartlike in appearance, and covered with a greasy scale. The scale tends to drop off, leaving a superficial, moist scab which is soon recovered by a growth of brownish scales. This lesion is definitely precancerous, and if there is a history of progressive increase in size, the lesion should be widely excised.

Dermoid Cysts

(See section on Congenital Tumors of the Neck.)

Hemangioma

(See Chapter 33, Peripheral Vascular Diseases.)

Moles or Benign Pigmented Neri

Nevus means mark or blemish and this term is often used to designate a birthmark. A so-called *mole* is a nevus which may consist of a localized pigment deposit. Moles are common about the face and neck. They may be skin colored, hairy, or show varying degrees of pigmentation. It is estimated that 90% of people possess pigmented moles. The number per person averages about 20 but may range from 2 or 3 to several hundred visible moles. While moles are benign, they are important because they can and do become malignant. It is estimated that anywhere from 30-65% of malignant melanomas arise from pre-existing benign nevi. Such melanomas are considered to be among the most malignant lesions found in man. Chronic irritation, infection, and trauma seem to play a definite role in the transformation of benign moles into malignant melanomas. It is significant that pigmented moles very rarely become malignant before the age of puberty. Such prepubertal lesions may clinically and histologically resemble a true malignant

melanoma seen in the adult. They do not, however, behave as such but rather as perfectly benign lesions.

The prophylactic removal of certain moles is completely justified, and this should be done before the age of puberty because of the increased malignant tendency after that age. Lesions to be removed are those situated in locations subject to chronic irritation by clothing or occupation, those which are darkly pigmented, and those which show any evidence of irritation or increase in size. Ackerman found the incidence of malignant change to be highest in the flat, hairless, slightly elevated, light brown mole.

The only treatment of a mole is a wide, complete excision. Nevi are radioresistant and should never be treated by methods of irradiation. The use of electrocautery or electrodesiccation as a form of treatment should be condemned because it is not generally wide or deep enough to remove all of the neval cells, and if part of the tumor remains, the irritation of this procedure has been shown to cause the cells to grow more rapidly and even to become malignant.

Malignant Tumors

Cancer of the skin is the most common form of cancer. Mortality statistics do not show this preponderance, because so many of these cases can be cured. The exposed skin surfaces of the face, ears, neck, and hands which are open to the sun's rays are the most common sites of skin cancer. Farmers and sailors who have been subject to sun and weather for years very often develop skin cancer. Persons with fair skin more frequently suffer from senile keratoses and skin cancer than those of darker complexion. Skin atrophy, skin tuberculosis, and x-ray dermatitis are precancerous conditions.

Basal Cell Carcinoma (Rodent Ulcer)

The face is the most common site of basal cell carcinoma, and basal cell carcinoma is the most common malignant tumor of the face. These tumors, like epidermoid cancer, tend to occur in the older age groups. They may appear in any part of the face but most often above the mouth and below the hair line and

about the ears. At the onset the tumor appears as a pearly nodule in the skin, and this nodule in the course of time breaks down to form an ulcer with rolled pearly edges. The ulcer enlarges slowly and persistently, and it may finally invade deeply, destroying underlying bone and cartilage, eventually causing hideous deformities. Despite its invasive character, a basal cell cancer rarely metastasizes.

Treatment.—Destruction of senile keratoses by electrocoagulation or excision will prevent the onset of many basal cell cancers. Because of its slow growth, the basal cell cancer is easily controlled in its early stages by adequate x-ray or radium treatment or by surgical excision.



Fig. 164—Basal cell carcinoma in skin of elderly white man. Note the raised, rolled edges.

The requisite for successful treatment is destruction of all tumor cells. The cure rates of surgery and irradiation are equally satisfactory if properly applied at a reasonably early stage.

Epidermoid Cancer

Epidermoid cancers frequently begin in hyperkeratoses of the skin of the cheeks, malar and temporal areas, and ears. They are less common than basal cell cancers on the forehead, eyelids, and nose. The epidermoid or squamous cell cancer usually begins as a warty area, which later ulcerates. It tends to grow rapidly. The edges of the ulcer are indurated, undermined, and infected. These cancers may metastasize to regional lymph nodes, though only a small proportion of such tumors of the face metastasize at an early stage.

Treatment.—The primary tumor may be treated by excision or by irradiation. Preference should be given to x-ray therapy if excision will produce large deformities about the eyes, mouth, or nose. Tumors that recur after irradiation should be treated surgically. Metastatic cervical lymph nodes are best treated surgically by a complete radical neck dissection of the involved side.

Malignant Melanoma

Malignant melanomas comprise 1-2% of the malignant tumors of the body and about 20% of the cutaneous tumors. In general, melanomas of the face, neck, and scalp comprise about 30% of all melanomas. It is one of the most lethal of all neoplastic diseases, with the average 5-year survival time of all cases being around 20%.



Fig. 165—Malignant melanoma on cheek of 65-year-old white woman. Note the size of the lesion and the central ulceration.

All moles which show any of the following changes should be immediately suspected of having undergone malignant transformation: (1) increase in size, (2) change in color, especially an increase in pigmentation; (3) pain, irritation, itching, or discomfort; (4) infection or ulceration; (5) bleeding or increase in vascularity; (6) the appearance of satellite pigmented areas adjacent to the original lesion.

Malignant melanomas metastasize through the lymphatics to the regional lymph nodes or by way of the blood stream to distant organs.

It is the latter which generally defeats efforts to cure. Prior to carrying out any therapeutic therapy one should have proof of diagnosis either by means of an incisional biopsy, in the case of a larger lesion, or a punch biopsy in a smaller one.

Surgery provides the only efficient method of treatment of malignant melanoma. Melanomas are radioresistant, and this method of therapy should be used only as a palliative measure or for psychologic reasons. Extreme desiccation is to be condemned as being completely inadequate, and hormonal therapy, including the use of estrogens, and pituitary irradiation, castration, bilateral nalectomy, and even a hypophysectomy, have been valueless.

The surgical therapy will generally consist of a wide local excision of the primary tumor together with a resection of the regional lymph nodes. In melanomas of the face or neck, in addition to wide excision of the primary lesion, a radical neck dissection on the involved side should also be carried out even if the lymph nodes are not clinically palpable. In a large percentage of these cases, metastatic malignant melanoma cells will be found within the lymphatics on histologic examination.

THE LIPS

Macrocheilia

Macrocheilia, hypertrophy of the lips, is a congenital condition, analogous to macroglossia. It is due to a lymphangioma or hemangioma accompanied by a connective tissue overgrowth. The lower lip is more frequently involved than the upper lip. The lip is firm, thickened, enlarged, and everted, causing considerable deformity. It is best treated by wedge excision of the excess tissue.

Herpes Labialis

Herpes of the lip generally follows the common cold, or exposure to sun. Cold sores begin as numerous small vesicles which later coalesce to form pustules, followed by a dry crust. The herpes should be completely healed in 1-3 weeks, unless secondarily infected. The disease is due to a virus infection. Herpes and ulcers of the lips which

not heal, under treatment, in 2-3 weeks should be regarded with suspicion. A biopsy should be taken in order to rule out the possibility of an early carcinoma.

In the early stages treatment by mild astringents, such as Witch Hazel, may be used. Bland ointments to soften the crusts should be applied later. Local application of a mild antibiotic ointment may be used with benefit.

Other Benign Lesions

The lips are subject to multiple benign conditions which are important because they may simulate an early carcinoma or are themselves precancerous in nature. They include the following conditions: hyperkeratosis, warts, papillomas, cracks and fissures, leukoplakia, and syphilis.

Hyperkeratosis is a condition characterized by the presence of fine flaky scales on the lip. The lip may bleed slightly when the scales are removed. It may be localized to a small area or may involve the entire lip. Hyperkeratosis is a premalignant condition seen mainly in people such as farmers and sailors exposed to excessive amounts of sunshine. If allowed to progress, the lesion will frequently become thickened and indurated and undergo malignant changes.

The treatment of hyperkeratosis in the early stages consists of the use of bland mild ointments to the involved area. In more resistant cases the use of low-voltage superficial x-ray therapy is of value. In more extensive and advanced cases, a mucosal stripping operation of the entire lower lip is carried out. The lip is reconstructed by advancing the mucous membrane on the inside of the lower lip and suturing it to the skin margin.

Warts and papillomas are very similar in their appearance and may be seen on the upper or lower lip. They, too, may undergo malignant changes, and for that reason they should be excised.

Cracks and fissures of the lips may be persistent and annoying, the crack tending to split open on smiling or stretching the lips. Cancer of the lip may begin in one of these fissures. If the crack or ulcer does not heal promptly on treatment by bland ointments, the fissure should be excised, and the specimen should

be examined by the pathologist. Chapped lips are associated with cold weather, the use of cosmetics, and the habit of licking the lips. Ointments should be used to keep the lips soft. Cracks about the corners of the mouth may indicate congenital or late syphilis or a vitamin B deficiency.

Leukoplakia of the lip, as elsewhere in the mucous membrane of the oral cavity, is a precancerous condition. It appears as a thick, white patch on the vermilion border of the lip similar to leukoplakia elsewhere. Histologically,

rete pegs and round cell infiltration beneath the dermis. If carcinoma ensues, the lesion becomes firm and indurated and may bleed when the surface is scraped. A biopsy must be taken in all suspicious cases. The treatment of early leukoplakia will include strict attention to the oral hygiene, elimination of infective foci in the mouth, cessation of excessive smoking, and an adequate intake of vitamin B.

Syphilis The lip is the most common site of extragenital chancre. This lip lesion is similar in appearance to a chancre elsewhere. There may be great edema of the lip, with enlargement of the regional lymph nodes. A chancre is more often found on the upper lip. Definite diagnosis is made by a search for spirochetes, by a biopsy if necessary, and by the prompt cure of the lip under antisyphilitic treatment. Treatment of the syphilis is all that is needed.

Benign tumors of the lips, such as *warts*, *polyps*, and *hemangiomas*, should be treated by the usual methods. Occasionally such lesions resemble cancer in appearance.

Cancer of the Lips (Epithelioma)

Only those cancers of the lip arising from the vermilion border or the mucocutaneous junction should be included in this category. The cancers which originate in the mucous membrane and spread to the lip grow more rapidly, metastasize earlier, and have a more serious prognosis. Cancer of the lip is a common tumor, and it comprises about 30% of oral cancers. At least 95% of lip cancers appear on the lower lip; less than 5% occur on the upper lip. Only about 3% of lip cancers

DISEASES OF THE FACE, MOUTH, AND NECK

are found in women, as shown in most statistical surveys. The majority of lip cancers are found in men over 60 years of age who have worked for years in the sun. Pipe smoking is not a proved factor in the production of labial cancer. About 40% of carcinomas occurring on the lip originate in a precancerous lesion.

Cancer of the lip may show itself as a raised, indurated lesion with a small central ulcer, as a deep hard ulcer, or as a raised warty tumor. This cancer tends to spread along the surface of the lip, rather than to penetrate early into its depth. More than half the lower lip may be attacked by the neoplasm, without deep penetration. Microscopically, the tumor is a typical squamous cell carcinoma, and the malignancy are well differentiated Grade I or II.

Metastases from lip cancer occur less frequently than in other forms of oral cancer. Even in the presence of large primary cancers there may be no lymph node metastases. Of all patients with lip cancers, between 40-45% show cervical lymph node metastases on admission or develop such metastases later. Disistant metastases are very rare. When metastases do occur, they are found most often in the submaxillary lymph nodes.

Treatment should not be given until a positive diagnosis has been established by the pathologist's examination of a biopsy specimen.

Treatment.—A small carcinomatous lesion of the lip (less than 1.5 cm in diameter) can be handled almost equally well by surgery or by x-ray therapy. Surgery will consist of a V-shaped excision, taking an adequate margin of tissue around the growth. In larger lesions treated surgically, the use of full-thickness pedicled flaps of the cheek or of the upper lip may be required in order to reconstruct the lower lip. The cosmetic result in many of these cases is surprisingly good. In the more superficial spreading cancer of the lip, the use of x-ray therapy is preferable. The local cancer can be controlled with little deformity by this means. Cervical metastases are best handled by a radical neck dissection. Prophylactic neck dissection to anticipate metastases in cases where none are clinically apparent is not indicated. If operation for neck metastases is inadvisable for any reason, the lymph node metastases may

be retarded by thorough x-ray therapy. In the absence of cervical adenopathy, prophylactic x-ray treatment is not advised.

THE MOUTH AND TONGUE

The mouth is lined by squamous epithelium and this epithelium is constantly exposed to minor irritation and injury. The rich blood supply of the mouth is one factor that makes its tissues resistant to the bacteria which are always present. Wounds heal promptly if serious infection is not present.

Malformations

Malformations of the mouth include cleft palate, absence of the tongue, bifid tongue, tongue-tie, macroglossia, and fissured or geographic tongue. Malformations of the jaws and irregularities of the teeth are common and varied. In more severe cases surgical treatment may be required, but most patients can be greatly improved by the orthodontist if treatment is begun at a sufficiently early age.

Tongue-tie or ankyloglossia, an abnormal fixation of the tongue, may be congenital or acquired. The congenital variety is usually caused by a short frenum. This may interfere with nursing in the infant, or it may produce speech defects in older children. Simple incision of the frenum relieves the symptoms if carried out early, and this should be done before the child begins to talk.

The term *macroglossia* refers to a chronic enlargement of the tongue, which may be congenital in origin or acquired. The congenital type may be due to a diffuse lymphangioma or hemangioma of the tongue, or it may result from a congenital arteriovenous fistula between the lingual artery and vein. The acquired lesions result from acute inflammations, as well as from various types of tumors and cancers of the tongue. An extensive cancer of the base of the tongue will occasionally manifest itself first as massive edema of the anterior two thirds of this organ.

The treatment of the congenital variety of macroglossia is usually unsatisfactory, while the acquired variety generally lends itself to antibiotic therapy, to surgery or to radiation therapy, or, in some cases, to a combination of all three methods.

Injuries

Wounds of the mouth and tongue are frequent, often caused by biting of the tongue. Bleeding may be severe and prolonged. Fractures of the jaws are usually compounded into the mouth, and this may lead to secondary infection. Teeth are often broken or completely avulsed during oral injuries.

Larger bleeding vessels should be ligated, and wounds should be sutured. Loosened teeth do not necessarily require extraction. Fractures of the jaws should be immobilized in the best possible position. If sufficient teeth are present, this is best accomplished by wiring the teeth together to splint the fracture until healing is well under way. The use of antibiotics assists in controlling infection.

Burns and scalds of the mouth and tongue, unless very deep, produce only superficial destruction of the mucous membrane and papillae. They are very painful. Deeper burns may lead to the formation of ulcers which heal with a scar. Strong acids or alkalis usually cause more destruction in the base of the tongue and the pharynx than in the anterior part of the tongue and mouth.

Inflammations and Infections

Infections in the mouth are very common, with associated spread to the regional lymph nodes or cellulitis of the surrounding soft tissues. Dental abscesses are frequently the primary site of the infection.

Stomatitis

The term *stomatitis* is applied to inflammation of the mucous membranes of the mouth. The mucous membrane becomes red, swollen, and tender, and there may be marked swelling of the face and lips. Many etiologic factors have been suggested, including vitamin deficiency, fevers, local infections from pyorrhea, or dental caries. Treatment with heavy metals such as mercury may produce stomatitis. Frequent mouthwashes, antibiotics, the removal of tartar from neglected teeth, and the care of infected teeth aid in the cure.

Aphthous stomatitis or canker appears in children or older persons as pinhead sized red or yellow spots on the edges of the tongue or

floor of the mouth. Small, very painful ulcers may form. The lesions disappear in a few days, but new crops may appear. It is said to be due to a virus. Cauterization of the cankers with a silver nitrate stick hastens recovery and relieves pain.

Vincent's Angina (Trench Mouth)

This is an ulcerative form of stomatitis due to an infection with a spirochete and a fusiform bacillus. It involves the oral mucous membrane. Smears should show the characteristic infecting organisms. Treatment includes attention to infection about teeth and gums, mouthwashes of dilute potassium permanganate or sodium perborate, and bland diet. In severe cases one may use systemic procaine penicillin 300,000-600,000 units daily.

Glossitis

Glossitis, or inflammation of the tongue, appears in several forms, and not infrequently it is associated with stomatitis or Vincent's angina. We generally recognize the following types of glossitis.

Acute superficial glossitis is characterized by burning pain, redness, and atrophy of the papillae. It may be associated with some other systemic disease such as uremia, diabetes, Plummer-Vinson syndrome, oral sepsis, or alcoholism. One must find and treat the underlying cause.

Acute parenchymatous glossitis is a deep inflammation of the tongue which arises from penetrating or incised wounds or accidental bite of the tongue. The onset of inflammation is sudden, the tongue becomes painful and swollen, and it may protrude from the open mouth. Edema may be so severe as to interfere with respiration. Suppuration is not the rule. Treatment includes warm mouthwashes and antibiotics. If an abscess forms, it must be incised. In rare cases a tracheostomy is necessary.

Chronic glossitis occurs mainly along the side of the middle third of the tongue or on the dorsum. Ulceration may or may not be present. In some cases, especially in the milder forms of chronic inflammation, there is a combination of atrophy and hypertrophy, so that while the papillae may be fewer in number, those which remain are increased in length and

thickness. The hypertrophy may be so marked in some areas that the long papillae fall apart in rows, leaving irregular fissurelike lines 3-5 mm. deep. There is no break in the mucosa and no real fissure. Scattered patches of leukoplakia are usually present. The Wassermann reaction may or may not be positive.

Treatment of this condition may be difficult. One must pay very strict attention to oral hygiene, including the correction of dental caries and other foci of infection. Large doses of vitamin B complex must be given; alcohol and tobacco should be discontinued; mouthwashes and antibiotics may also prove valuable. If the Wassermann reaction is positive, antisyphilitic therapy must be given.

Glossitis migrans or geographic tongue. The exact etiology of this condition is not known. It occurs especially on the anterior two thirds of the tongue and is characterized by round localized areas of atrophy of the papillae, leaving a smooth red glistening surface with a white margin around it. These areas tend to enlarge and fuse to form irregular outlines simulating a country on a map. New patches are constantly appearing in different locations as the older ones fade. The treatment is similar to that outlined for chronic glossitis.

Median rhomboid glossitis is characteristically located on the dorsum of the middle third of the tongue just in front of the V of the circumvallate papillae. It is a smooth, reddish brown lesion associated with local atrophy of the papillae. It is generally painless, and the consistency is not unlike that of the rest of the tongue. Its exact etiology is not known and the lesion is ordinarily self-limiting. Occasionally it may give rise to mild discomfort, in which case it should be surgically excised.

Leukoplakia

Leukoplakia is characterized by proliferation and thickening of the mucous membrane of the tongue or mouth, without evidence of neoplastic changes. The mucous membrane is white, thick, and smooth. In later stages it may be warty or fissured. In itself leukoplakia is usually symptomless, but it should always be regarded as potentially dangerous. Cancer develops in patches of leukoplakia much more commonly than in normal mucous membrane.

Leukoplakia is seen more often in people over 40 years of age. The etiology is unknown, but chronic irritation, syphilis, poor mouth hygiene, smoking, tobacco chewing, and avitaminosis may play a part in its production.

Treatment is unsatisfactory. Oral hygiene should be improved; smoking and intake of spicy foods and alcohol should be curtailed. Syphilis, if present, must be treated. Large doses of vitamins A and B must be given, especially riboflavin. Recurrence of the leukoplakia is to be expected after treatment by excision, cauterization, or irradiation. Patients with leukoplakia should be observed regularly so that warts, ulcers, and beginning cancers can be treated at the earliest opportunity.

Benign Ulcers of the Tongue

Simple ulcers of the mouth and tongue may be caused by jagged teeth, badly fitting dentures, other trauma, or from unknown causes. They should be treated by removal of any known cause, by bland mouthwashes, or by painting with dilute solution of silver nitrate. Ulcers that fail to show signs of healing in two weeks should be biopsied. A diet high in vitamins should be prescribed.

Syphilis

Syphilis of the tongue is relatively rare today. One may see it as a *primary chancre* which differs little from a chancre elsewhere. It is generally present near the tip of the tongue. The regional lymph nodes in the submental and submandibular triangle are usually enlarged.

Mucous patches of secondary syphilis may also occur in the mouth, and in this form the infection is readily transferred to other individuals by kissing or by the use of drinking or eating utensils.

The *tertiary form of syphilis* may appear on the tongue, either in the form of a gumma or of a syphilitic glossitis. The former is often found near the midline as a soft to firm nodule which may later ulcerate. The gummatous chancre is painless and has a typical punched-out appearance, the edges are soft and overhanging, and it is usually associated with a dirty discharge of slough and pus. In the

syphilitic glossitis, the tongue has a smooth, glazed appearance associated with a moderate amount of leukoplakia and atrophy of the papillae and later ulcerates. Visible scarring may be apparent, due to the underlying fibrosis.

The blood Wassermann and biopsy will confirm the diagnosis. Specific systemic treatment will cure the local lesion.

Tuberculosis

Tuberculosis of the tongue is almost invariably accompanied by other manifestations of the disease in the larynx or lungs. The tuberculous ulcer is generally found on the tip or dorsum. The ulcer is superficial, with sloping, soft edges and a pale necrotic base, and is characteristically painful. Diagnosis may be confirmed by biopsy. Treatment of the general disease will heal the local ulcer, but excision may hasten healing and relieve pain.



Fig 166—Tuberculosis of tongue. Lesion is characteristically painful and situated on dorsum of tongue. Must exclude cancer by biopsy.

Actinomycosis

Primary actinomycosis appears rarely as a firm, painful nodule in the depths of the tongue. The nodule breaks down to form a necrotic ulcer or numerous sinuses discharging the typical sulfur granules on the dorsum of the tongue. The cervical lymph nodes are not typically enlarged. Carious teeth and pyorrhea are usually present. When the ulcer is excised the fungus may be seen in the stained sections. Excision of the ulcer, extraction of diseased teeth, and massive doses of penicillin should cure the disease.

Inflammation of the Teeth, Gums, and Jaws

Pyorrhea Alveolaris

The deposits of decayed food and tartar about the teeth at their junction with the gums allows infection to occur under the gum edges. The gums become red, pus escapes, gums recede, and from this source infection may spread to the floor of the mouth or neck. Treatment consists of cleaning tartar from the teeth and oral hygiene.

Alveolar Abscess

As a result of tooth decay, bacteria may reach the root canal, causing an abscess at the root of a tooth. The abscess may be symptomless, or there may be tenderness on pressure on the tooth. Such an abscess may lie dormant, or it may break through the thin external plate of the alveolus to produce a chronic sinus. In the lower jaw such a discharging sinus usually opens onto the skin of the chin in the midline. Extraction of the tooth generally cures the root abscess. The sinus tract may have to be completely excised.

An acute alveolar abscess may cause severe pain with swelling of the face or neck, redness of the gums, and tenderness on pressure over the affected tooth. The abscess may rupture into the mouth, or it may cause an inflammation in the face, floor of the mouth, or in the neck. Osteomyelitis of the jaw frequently results if treatment is delayed. The use of antibiotics and tooth extraction will bring about a cure.

Extension of the acute inflammation of the mouth to the neck, as a cellulitis or a cervical adenitis, is to be expected. Abscess formation is common. If the inflammation extends into the submaxillary and submental spaces, a Ludwig's angina may develop, with protrusion of the tongue and interference with respiration.

Tumors of the Mouth and Tongue

Benign Tumors of the Tongue

Dermoid cysts are found anteriorly in the midline, on the undersurface of the tongue, in the region of the raphe. As with other dermoid cysts it is lined by squamous epithelium and contains sebaceous material. The cyst can be removed readily through an oral incision.

Granular cell myoblastoma is a benign tumor that arises from embryonal myoblastic tissue. The tongue is the most common site in the body for these tumors. The tumors are characteristically small, measuring 1-2 cm. in diameter. They are firm and may occur along the lateral border or the dorsum of the tongue. Histologically, they are composed of large, rounded, or ovoid cells with acidophilic granular cytoplasm.



Fig. 167—Benign, firm sessile fibroma of tongue

Other Benign Tongue Tumors.—Rarely, innocent tumors such as lipomas, fibromas, and teratomas, containing bone or cartilage, may appear in the tongue. Hemangiomas of the capillary or cavernous types are more common, the cavernous hemangioma sometimes presenting as a pedunculated bluish mass which bleeds easily and freely.

Papillomas and warts are quite common on the tongue and are considered to be premalignant lesions. Some of these papillomas grow to cover a large area of the tongue, suggesting the possibility that cancer has already developed.

All these tumors should be treated by excision, and in all cases the tissue must be examined by a pathologist. An inflammation or hypertrophy of a lingual tonsil may be mistaken for a cancer. This tonsillar tissue is situated on the lateral border of the tongue, at its junction with the anterior pillar of the soft palate.

Benign Tumors of the Mouth

Mucous cysts are small retention cysts, containing thick mucus, which form in the mucous glands of the buccal mucosa and inner side of the lower lip. Complete excision cures the cysts.

Ranula is a cystic swelling situated in the floor of the mouth to one side of the midline. Its origin is not clear, but it appears to be a cystic dilatation of the sublingual salivary gland or of the submaxillary duct. The cyst is usually unilateral, appears in childhood, grows slowly to reach a large size, and may extend to cause swelling in the submaxillary triangle.

Treatment by complete excision is technically difficult because of the thin, delicate cyst wall and the large size of the cyst. Suture of the



Fig. 168—Lingual thyroid located in the region of the foramen cecum.

defect after incomplete removal of the ranula will cause a recurrence. Most of the cysts may be cured by unroofing the cyst and then suturing the cut edges of the cyst to the mucous membrane of the floor of the mouth, in effect a marsupialization.

Mixed cell tumors similar to those of the parotid gland occur anywhere in the mouth, most frequently on the hard palate. They are derived from the minor salivary glands present in the mucous membrane of the mouth. The tumors are well circumscribed and are covered by normal mucous membrane. They grow slowly and painlessly and are almost always

benign. They can be easily shelled out, and they do not recur if completely removed.

Torus palatinus is a hard, smooth nodular growth which measures 2-4 cm. in diameter and occurs at about the center of the hard palate where the two maxillas are fused in the midline. It is a true osteoma of the maxilla and is generally covered with normal mucous membrane. The lesion has no special clinical significance, except that it may be mistaken for a malignant bone tumor or may interfere with the proper fit of a dental plate. It can be surgically excised if necessary.

Torus mandibularis is a similar exostosis occurring on the inside of the lower jaw. It may occur in the midline, or it may be present symmetrically on either side of the midline. No treatment is necessary unless the lesion interferes with lower dentures.



Fig 169—*Torus palatinus*. A benign osteoma of the hard palate

Epulis is a not uncommon tumor of the mouth and is seen most frequently in children and young adults between the ages of 10 and 20 years. It occurs on the alveolar ridges at the gingival margins of the teeth, often where teeth have been extracted, and is said to arise from the alveolar dental periosteum. Such tumors are usually covered with mucous membrane and may have a narrow pedunculated attachment or may be sessile and spread over the gum surface for some distance, hiding the crowns of adjacent teeth. The consistency of the tumor is soft, and it has a red or reddish brown color. The epulis is almost always a

benign tumor which grows slowly and painlessly. Histologically it is composed of many multinucleated giant cells in a dense fibrous stroma.

The treatment is surgical. If not completely removed, the lesion is likely to recur. It may be necessary to extract adjacent teeth, especially if these have been loosened from their sockets by the pressure of the tumor.



Fig 170—Benign giant cell epulis of the gum

Malignant Tumors of the Mouth

Cancer within the mouth is common, and it may involve the tongue, cheek, gum margins, floor of the mouth, palate, or tonsils. Cancers in these different areas of the mouth vary somewhat in rapidity of growth, malignancy, and tendency to form metastases. In all cases the disease is so malignant and dangerous that the need for early diagnosis and prompt, adequate treatment cannot be overemphasized.

Cancer of the mouth accounts for about 4% of all cancer in man, and occurs in the male four or five times more commonly than in the female. The neoplasm, usually occurring after 40 years of age, is most common between the ages of 50 and 70 years.

Although the etiology of cancer is unknown, intraoral cancer is generally found in association with poor mouth hygiene, rough carious teeth, badly fitting dentures, leukoplakia, and syphilis. There is often a history of excessive smoking or of tobacco chewing. Chronic anamiasis with glossitis and stomatitis may also be present.

About 90% of mouth cancers are of the epidermoid type, arising from the squamous epithelium which lines the mouth and covers the tongue. Adenocarcinoma may arise from mucous or minor salivary glands, and such cancers are most frequently found in the palate. Melanocarcinomas infrequently appear in the mouth, usually on the palate and alveolar ridge. Lymphosarcomas may originate about the base of the tongue, tonsils, and oropharynx. Most of these tumors tend to metastasize to regional lymph nodes.

A. Cancer of the Tongue

Cancer of the tongue is the most frequent type of intraoral cancer. It comprises approximately 25% of all malignant tumors originating from within the mouth. For clinical reasons we generally divide the tongue into two main areas—the mobile anterior two thirds of the tongue extends from the tip to the vallate papillae, and the base of the tongue extends backward from this point to the hyoid bone. The squamous epithelium which covers the tongue is firmly adherent to the underlying muscle. The lymphatics of the anterior part of the tongue drain to the submental and submaxillary triangles and to the internal jugular chain of lymph nodes. The lymphatics of the base of the tongue, in common with the oropharynx, drain to the subdigastic lymph node and to the lower internal jugular chain. There are lymphatic communications across the midline of the tongue, especially at the tip and base of the organ, so that cancers of the tip, dorsum, or base of the tongue frequently metastasize to both sides of the neck. However, when the growth is confined to the lateral margin of the tongue, unilateral spread only is to be expected. Cancer of the anterior two thirds of the tongue is predominantly a disease of males, occurring five to six times more commonly in males than in females. Cancer of the base or posterior third of the tongue affects both sexes almost equally. It is interesting that the majority of tongue cancers, approximately 60%, occurs on the lateral edges of the middle third of the tongue, about 30% occurs on the posterior third, while the remainder occurs on the anterior third or dorsum of the tongue.

Cancer of the tongue begins as an indurated area or a fissure in a patch of leukoplakia, as a papilloma, or as a small ulcer. The earliest lesions look so innocent that cancer is often not suspected. Central necrosis and ulceration of the cancer occur early. Severe pain is not usually a symptom in a small tongue cancer, and this fact is responsible for the delay in diagnosis of cancer of the base of the tongue. The cancer begins most often on the lateral margins or undersurface of the middle third of the tongue. The primary lesion, if untreated or uncontrolled, does not long remain superficial but spreads as a deep burrowing indurated ulcer. In the later stages severe pain, in *section, tongue fixation, salivation, cervical metastases, and terminal hemorrhage* present a horrible picture.

Examination of the tongue should include palpation, as well as inspection, as the area of induration may be more extensive than might appear from inspection alone. Palpation is especially useful in examination of tumors of the base of the tongue.

Cervical metastases appear early in tongue cancer, and they are present in about 40% of patients when first examined. Cervical metastases can be expected at some stage in 65-70% of cases under observation or treatment. Distant metastases are considered rather uncommon, but autopsies frequently demonstrate their presence.

The appearance of the lesion, its progressive growth, the age of the patient, leukoplakia and enlarged cervical lymph glands may make positive diagnosis of cancer easy. Vincent's angina, traumatic ulcer, papilloma, tuberculosis, or gumma must be considered in the differential diagnosis. Biopsy, which should be done early, is necessary to settle the diagnosis. A positive Wassermann reaction does not eliminate the probability of cancer, and, indeed, mouth cancer is much more common in syphilis than in the general group of the population. All suspicious tongue lesions should be regarded as cancer until proved innocent by biopsy.

Treatment.—Much may be accomplished by prophylaxis to prevent oral cancer. Removal of small areas of leukoplakia, dental care, and the treatment of syphilis are indicated.

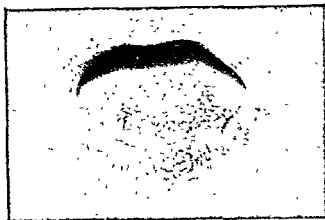


Plate 2.—Cancer of the Lip.



Plate 3.—Leukoplakia of the Tongue.

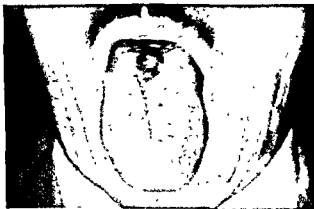


Plate 4.—Median Rhomboid Glossitis.

*Courtesy Martin Hayes, Cancer of the Head and Neck,
Mouth Cancer and the Dentist, American Cancer Society, Inc.*



Plate 5.—Cancer of the Tongue.



Plate 6.—Cancer of Undersurface of Tongue and Floor of Mouth.

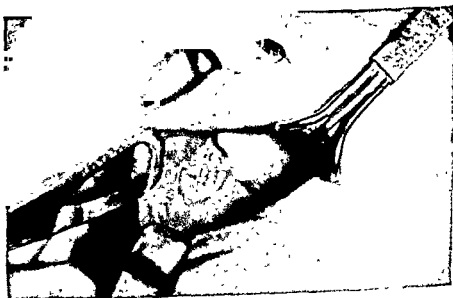


Plate 7.—Cancer of Buccal Mucosa.

Courtesy Martin, Hayer. Cancer of the Head and Neck. Mouth Cancer and the Dentist, American Cancer Society, Inc.

When tongue cancer has begun, treatment includes attack on the local lesion and on the metastases. In the advanced cases only palliative treatment is advisable.

Surgery and irradiation are the two principal methods employed in the treatment of cancer of the tongue. Whatever method is used will depend to a large extent on the experience and skill of the attending consultant, the size and location of the lesion in the tongue, the presence or absence of cervical metastases, and the general condition of the patient.

Radiation therapy is suitable for (1) patients who refuse surgical procedures, (2) aged and debilitated patients who are poor operative risks, (3) patients with small to moderate-sized growths on the tongue without evidence of cervical lymphatic spread, and (4) patients with tumors involving the base of the tongue.

The main methods of radiation therapy consist in the use of the following procedures either alone or in combination with one another: (1) external x-ray, (2) peroral x-ray therapy using a suitable sized cone, and (3) interstitial irradiation using radon seeds or radium needles.

The reason that cancers of the base of the tongue tend to be treated by means of irradiation alone is that such lesions are relatively inaccessible for surgery. Furthermore, they are generally more anaplastic and therefore more radiosensitive than lesions situated on the anterior two thirds of this organ.

The cases most suitable for surgery are as follows:

1. Small cancers measuring up to 3 cm in diameter. Such lesions situated on the lateral border or on the tip of the tongue can be removed quite adequately by a V excision, including a wide margin of normal-appearing tongue tissue on either side.

2. All cancers of the tongue in which there is evidence of metastases to the cervical lymph nodes. For such patients, the proper treatment includes complete destruction of the primary growth simultaneously with removal of the lymphatic tissue and lymph nodes draining the tumor area. The operation will consist of a radical neck dissection, together with a resection of the primary lesion on the tongue, generally carried out in continuity. This is done in one of two ways:

(a) The neck dissection is performed and the mandible, floor of mouth, and half of the tongue are removed *en bloc*.

(b) Here the neck dissection is completed, and the floor of the mouth and tongue muscles are loosened in the neck. The neck wound is closed, and the hemiglossectomy is executed through the mouth. The resected neck tissues are pulled up through the mouth, or the tongue is pulled down into the neck so that there is actually an *en bloc* dissection without removing the jaw. This is the "pull-through" operation of Ward.

3. All cases where the mandible is involved by growth. The use of radiation therapy can result in marked osteonecrosis of the mandible with considerable amount of morbidity and with little chance of controlling the cancer in the bone.

4. Failure or recurrence following radiation therapy will certainly require radical surgical attempts in an effort to control the disease. Hemiglossectomy and even total glossectomy may be required for patients who fail to respond to radiation or in those in which the tumor recurs after thorough irradiation.

As we have already implied, the most effective treatment for metastatic cervical lymph nodes from a primary cancer in the tongue is a complete radical neck dissection. In a small percentage of cases where there are metastases to both sides of the neck, a bilateral radical neck dissection will be required. The neck dissection may be carried out as a later and separate procedure, where the primary is already controlled, or as an associated procedure as mentioned previously. There is no uniform agreement regarding the advisability of carrying out a prophylactic neck dissection in cancer of the tongue. The decision in any given case will depend upon the clinical setting which includes many factors such as the age of the patient, the size and location of the primary tumor, the ability of the patient to attend frequent follow-up visits, and the histologic grade of the lesion.

B. Other Malignant Tumors of the Mouth

Other malignant tumors in the mouth may arise from the floor of the mouth, the upper or lower alveolar ridge, the buccal mucosa, or

the hard or soft palate. The lymphatic drainage from these areas is primarily to the sub-mandibular and the upper cervical lymph nodes.

The majority of the cancers are epidermoid in type arising from the surface epithelium. Occasionally one finds an adenocarcinoma arising from the minor salivary gland tissue present within the oral mucous membrane. On very rare occasions a malignant melanoma may be seen arising either from the alveolar ridge or from the hard palate.

Oral cancers generally begin in an area of leukoplakia either as a small warty lesion, as a larger fungating tumor, or as an ulcerating and infiltrating neoplasm. A biopsy will readily establish the diagnosis.

Treatment.—The treatment in any given case will depend on several factors, including the age and general condition of the patient, the location and extent of the primary tumor, the presence or absence of cervical lymph node metastases, and the proximity of the lesion to bone or to its actual involvement of bone. In many of the smaller lesions, radiation therapy or surgery will yield equally excellent results. In larger tumors adjacent to or involving bone and in cases where there is established lymphatic metastasis in the neck, radical surgery is to be preferred. This will consist of wide excision of the primary tumor combined with a partial resection of the upper or lower jaw, as the case may be, and a radical dissection of the lymphatic tissue of the involved side of the neck.

THE PHARYNX

The pharynx is a cylindrically shaped tube that is wide above and narrow below. It extends from the base of the skull above to the level of the 6th cervical vertebra below. The pharynx is generally divided into three parts:

- 1 The nasopharynx extends upward above the level of the soft palate to the base of the skull

- 2 The oropharynx is that part of the pharynx between the soft palate above and the tip of the epiglottis below

- 3 The hypopharynx is that part of the pharynx that extends below the tip of the epiglottis and includes the pyriform sinus on

either side. Anteriorly the hypopharynx communicates with the larynx, while posteriorly it becomes continuous with the esophagus

Inflammatory Lesions

Tonsillitis

This may be acute or chronic in type. In the acute type the symptoms are those of sore throat, difficulty in swallowing, chills, fever which may go up to 103°-104° F., and general malaise. The responsible organisms are usually hemolytic streptococci. The tonsil itself

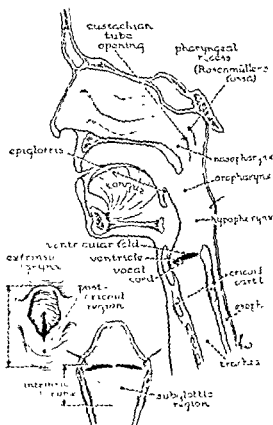


Fig. 171.—Anatomy of the pharynx and larynx

generally becomes enlarged and appears red and swollen, and there may be grayish white patches of exudate on the surface or within the tonsillar crypts. The upper jugular lymph nodes which drain the tonsil become acutely enlarged, painful, and quite tender.

The treatment of acute tonsillitis is conservative and consists in the use of large doses of a suitable antibiotic and the use of warm saline or soda bicarbonate mouthwashes, together with the use of aspirin compound to control the pain and lower the temperature

Peritonsillar abscess or quinsy, a serious complication of acute tonsillitis, consists in suppuration outside the capsule of the tonsil and, in most cases, is situated in the tissue of the soft palate above the tonsil. The symptoms are those of acute tonsillitis, only more severe. Examination will readily show that the palate on the affected side is congested and bulging, and the uvula may be quite swollen, edematous, and pushed across toward the opposite side. In addition to the methods recommended above for the treatment of acute tonsillitis, one must perform surgical incision and drainage of the abscess. This is usually followed by immediate relief of symptoms and rapid recovery.

Retropharyngeal Abscess

Two forms of retropharyngeal abscess are generally recognized—the acute and the chronic. The acute abscess is due to pyogenic infection in the retropharyngeal lymph nodes, which occurs most often in children but can be seen occasionally in adults and is generally secondary to some focus of infection in the mouth or throat. Suppuration of the lymph node ensues with subsequent abscess formation.

The acute type may also be seen as a complication following gastroscopy and esophagoscopy when a periesophageal infection results from damage to the mucosa of the upper

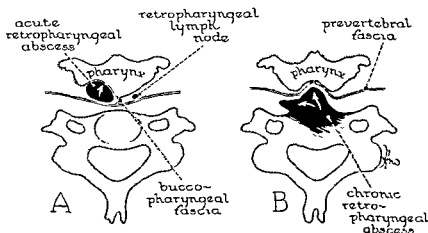


Fig. 172—A, Acute retropharyngeal abscess situated anterior to the prevertebral fascia and to one side or other of the midline.

B, Tuberculous retropharyngeal abscess secondary to tuberculosis of a cervical vertebra. The abscess is situated behind the prevertebral fascia and may bulge the pharynx in the midline.

Chronic tonsillitis is a condition which occurs predominantly in children and is associated with hypertrophy of the adenoids. It generally manifests itself by a chronic hyperemia of the pillars. The tonsil at times may be small, while at other times it is larger than normal. It is usually smooth and pale in appearance. The upper cervical lymph nodes are frequently palpably enlarged.

Treatment, consisting of tonsillectomy, is necessary when (1) the tonsils are large enough to cause respiratory or speech difficulty, (2) there are recurrent sore throats, and (3) there are systemic infections traceable to a focus in the tonsil.

Esophagus. The usual site of injury is in the posterocervical region where the thin esophageal wall may be compressed between the lower cervical vertebral bodies and the rigid endoscope. This is particularly likely to occur in patients with anterior bony spurs due to osteoarthritis. The injury, which may be a simple bruising of the wall or an actual pressure necrosis, results in a perforation of the wall and a resultant retropharyngeal or retroesophageal infection.

The acute abscess occurs in front of the prevertebral fascia and behind the buccopharyngeal fascia. Such an abscess tends to remain on one side or the other of the midline because of the attachment of the buccopharyngeal fascia

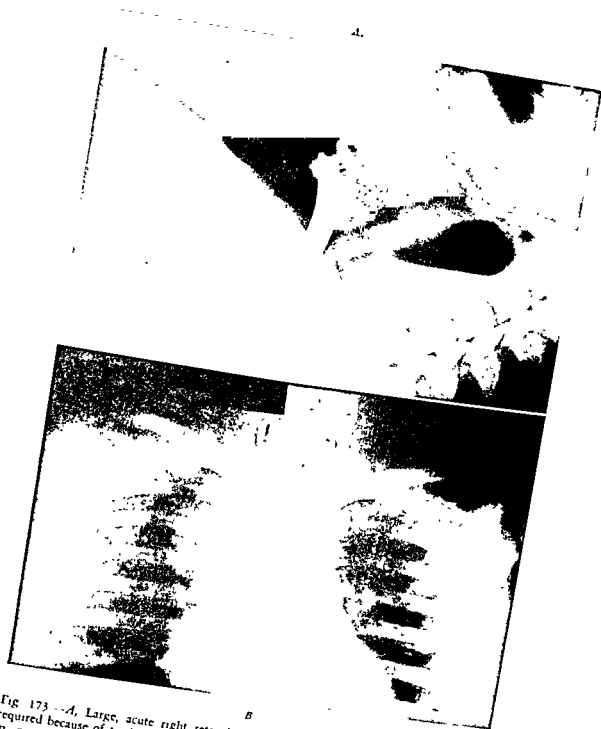


Fig. 173 --A, Large, acute right retropharyngeal abscess. Note the air fluid level. Tracheotomy required because of tracheal compression.
 B, Secondary abscess in the right superior mediastinum, caused by downward extension of the infection between the second and third layers of the deep cervical fascia. Both abscesses were found to be in direct communication at operation and were drained through a single incision in the right neck along the anterior border of the sternomastoid muscle. The middle space was entered by incising the second layer of deep fascia anterior and medial to the carotid sheath.

to the prevertebral fascia in the midline. This abscess, lying between the second and third layers of the deep cervical fascia, if left untreated, will track down to form a secondary collection in the superior mediastinum.

The *symptoms* to be expected are fever, swelling in the pharynx, difficulty in breathing, supraclavicular tenderness, and more or less toxic reactions. Clinical swelling of the neck may develop but is not an early feature. X-ray of the neck may show the presence of gas within the soft tissues, and this is almost pathognomonic of either a frank tear or a slow leak from a minute hole in the esophagus.

Treatment.—Before treatment is initiated, an x-ray should be taken of the cervical vertebrae to determine whether or not tuberculous disease is present. A history of a previous endoscopic procedure should immediately raise the question of a possible periesophagitis, and x-ray may show the presence of gas and/or fluid in the soft tissues of the neck. For the patient with an acute abscess, immediate conservative therapy should be instituted, giving him large doses of antibiotics and forbidding all fluid and food by mouth. If there is a demonstrable fluctuant abscess in the posterior pharynx, then the patient should be taken to

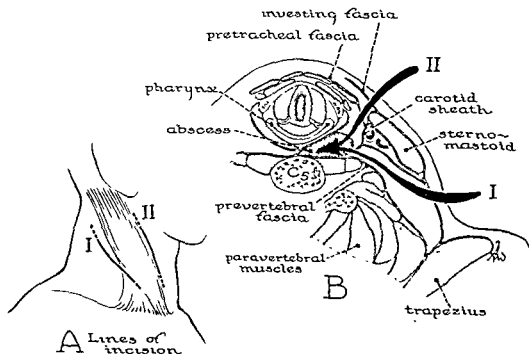


Fig. 174—Lateral cervical approaches to retropharyngeal abscess

A, Lines of incision

B, Arrows indicate the pathway to the focus I, posterior approach, II, anterior approach

The second type of retropharyngeal abscess is the chronic type and is generally due to tuberculosis of cervical vertebrae. This abscess lies behind the prevertebral fascia and may present into the pharynx in the midline, in contrast to the acute abscess which is lateral in position. Such an abscess spreads behind the prevertebral fascia and may point on either side of the neck behind the sternomastoid muscle. Occasionally it may track down behind the fascia into the posterior thorax.

the operating room, where, under endotracheal anesthesia, the fluctuant abscess is opened through the mouth. The patient's head should be lowered and the pharynx well packed off in order to prevent aspiration of pus. A tracheostomy set should be on hand in case of need. Occasionally an abscess may be incised without anesthesia.

Unless there is a frank tear in the esophageal wall, most cases of periesophagitis following endoscopy will respond to conservative treat-

DISEASES OF THE FACE, MOUTH, AND NECK

ment, as outlined above. A minority of these cases, however, may go on to frank suppuration, and then surgical drainage becomes necessary. The usual approach is through a small incision along the posterior border of the sternomastoid muscle, just below the cricoid level. Once the deep cervical fascia has been reached, the finger easily passes medially along the front of the prevertebral fascia. This leads directly to the retropharyngeal space at the level where trauma usually occurs. After sucking out the pus from the abscess cavity, a rubber tube is passed along the track and the skin approximated around the tube. Alternately, where there is evidence of tracking down of the infection into the superior mediastinum, a cervical mediastinotomy becomes necessary, and this is best carried out by a single incision in the neck running along the anterior border of the sternomastoid muscle. The retropharyngeal space is entered by incising the second layer of the deep cervical fascia, anterior and medial to the carotid sheath (Fig 174, A, B).

In the chronic type of retropharyngeal abscess with evidence of bone disease, the abscess should be aspirated through the side of the neck or the pus evacuated by an incision behind the sternomastoid muscle with resuture of the wound. The usual systemic treatment of tuberculosis should then be vigorously instituted.

TUMORS OF THE PHARYNX

Tumors of the Nasopharynx

Benign Nasopharyngeal Fibroma.—This usually occurs in young males aged 6-25 years and is rarely seen after the age of 30 years. Histologically, the lesion is composed of a mixture of vascular and fibrous connective tissue.

The tumors arise from the walls of the nasopharynx and are encapsulated and generally attached by a pedicle at the point of origin. The early symptoms include nasal obstruction and bleeding because of their vascularity. Although benign, these tumors can, because of continued growth and pressure, cause marked displacement of the eyes, cheeks, and surrounding tissue.

Treatment.—Small tumors can be removed through the nares. Medium-sized lesions on a pedicle may be removed by means of a cautery

snare utilizing a combination approach through the nose and through the mouth. Larger tumors are best reached by means of a Ferguson incision on the face, going through the antrum to reach the nasopharynx. If not completely removed, the lesions can and do recur. If residual tumor is left, it should be treated either by x-ray or direct radium application.

Radiation therapy alone is not effective in completely eradicating nasopharyngeal fibroma. Extensive radiation for such benign tumors should not be given because of the danger of early or late osteonecrosis of the base of the skull with secondary meningitis. It has been postulated that these tumors may be related to excessive estrogens, and for that reason many of them have been treated with testosterone propionate. The results with this method of treatment have proved somewhat disappointing.

Cancer of the Nasopharynx.—The majority of malignant neoplasms of the nasopharynx consist of epidermoid carcinoma and lympho-adenocarcinomas arising from the mucous and minor salivary glands. This disease occurs predominantly in males and has a special tendency to occur in the Chinese male. The lymphatics from the nasopharynx drain primarily to the spinal accessory chain situated in the posterior triangle of the neck. The early symptoms of nasopharyngeal cancer may be lacking, and the first evidence may be a large hard lymph node in the posterior triangle of the neck. Later deafness and tinnitus may occur because of encroachment of the disease on the eustachian tube, or there may be nasal obstruction and bleeding. The disease may later erode the base of the skull to involve the 6th cranial nerve and produce paralysis of the external rectus muscle of the eye. The 3rd, 4th, and 5th cranial nerves may be later involved.

Treatment.—Surgery is not indicated in cancer of the nasopharynx because of the relative inaccessibility of the region to an adequate surgical approach. Furthermore, these tumors are usually highly anaplastic and, in general, radiosensitive. The treatment, therefore, is primarily by irradiation, utilizing a combination of external radiation therapy through multiple ports, generally supplemented by intracavitary radium or radon.

Tumors of the Oropharynx

These include lesions arising in the posterior and lateral walls and those arising from the tonsil and tonsillar pillars, but do not include those arising in relation to the base of the tongue or to the extrinsic larynx. The most frequent and important tumors arising in this region are of a malignant nature.

Cancer begins either on the lateral or the posterior wall or both. Early symptoms are lacking, and not infrequently the first symptom is a large metastatic lymph node in the neck. In other cases the patient may complain of a mild irritation in the throat or of slight dysphagia. Later, as the tumor enlarges, dysphagia becomes more marked, difficulty in breathing and hoarseness are still later manifestations.

The majority of the tumors are epidermoid cancers. The treatment is by means of external irradiation given through two or more ports.

Cancer of the Tonsil

Next to cancer of the laryngopharynx, cancer of the tonsil is the common malignant tumor of the upper air passages, and it comprises about 25% of all cancers. Malignant tumors of the tonsil may be carcinomas, lymphoepitheliomas, or lymphosarcomas. The *epidermoid cancer* is the commonest form, and it appears as an ulcerating tumor of the tonsil, spreading to the soft palate and the pillars of the tonsil. This cancer is rather undifferentiated and radiosensitive, and it gives early lymph node metastases.

The *lymphoepithelioma* is usually a smooth tumor, showing little ulceration or spread to the palate. Lymph node metastases appear early, and there may also be extension to the lungs, mediastinum, liver, and bones. This tumor is extremely radiosensitive.

Lymphosarcoma does not ulcerate until late, but it may reach a large size in the tonsil. Enlarged cervical lymph node metastases accompany the tonsillar hypertrophy, and the disease may give a general lymphadenopathy as in lymphosarcoma elsewhere. Here, too, the disease is very radiosensitive.

Treatment—Since the majority of the malignant tumors occurring in the tonsil are highly radiosensitive, it follows that the best form

of treatment is radiation therapy. Even cervical lymph node metastases in cases of lymphoepithelioma and lymphosarcoma may be well handled by means of irradiation. Surgery is generally reserved for recurrences following adequate x-ray treatment. Cases of squamous cell carcinoma of the tonsil may prove to be much more difficult to control by means of irradiation. Primary surgery is especially indicated in cases of squamous cell carcinoma in which there is evidence of cervical lymph node metastases. In such cases the treatment will consist of a radical neck dissection combined with a resection of the ascending portion of the ramus of the mandible together with the tonsillar region in continuity.

Tumors of the Hypopharynx

Here, also, the most frequent and important tumor is cancer. Except for lesions arising on the posterior wall of the hypopharynx, most of the cancers in this region are best considered as lesions of the extrinsic larynx. Cancers of the posterior wall of the hypopharynx usually spread diffusely and tend especially to extend down toward the esophagus. The symptoms include vague discomfort in the throat on swallowing, in the early cases, and frank dysphagia later. Metastases to the deep cervical lymph nodes occur relatively early and at times may be the only symptom.

Treatment may be carried out either by means of external x-ray or by surgery. The Trotter or Wooley operation of resecting the hypopharynx and part of the cervical esophagus may be tried, with reconstruction of the pharynx in several stages by means of skin flaps. Another method of reconstruction consists of utilizing a tubular split thickness skin graft wrapped around a wire mesh or plastic stent. It may or may not be necessary to sacrifice the larynx, depending on the extent of the disease.

THE LARYNX

Benign Tumors

Papillomas of the larynx are the most frequent of all benign growths. They may occur in children as well as in adults and are definitely precancerous in nature. Other benign



Fig 175—Carcinoma of the right vocal cord.



Fig 176—Carcinoma destroying the left vocal cord with subglottic extension

tumors include specific granulomas due to syphilis, tuberculosis and the like, and the non-specific granuloma which is due to chronic irritation or to an inflammatory condition. Fibroma, lipoma, hemangioma, chondroma, and cysts are other rarely encountered benign tumors.

The *symptoms* of benign laryngeal tumors include hoarseness or huskiness of the voice. In larger lesions dyspnea results due to obstruction of the airway.

The *treatment* of benign lesions of the larynx is removal through the mouth by means of the direct laryngoscope and proper forceps.

Malignant Tumors

Cancers of the larynx comprise about 2% of all human cancers. Over 90% of the cases occur in males. We generally divide lesions in this region into two groups:

1. Cancer of the intrinsic larynx, which includes all those arising on the vocal cords, ventricles, and in the subglottic region.

2. Cancer of the extrinsic larynx, which includes those arising on the epiglottis, the aryepiglottic fold, the upper surfaces of the ventricular band, or in the postcricoid region.

The symptoms of laryngeal tumors will depend upon their exact site of origin. Hoarseness is the earliest symptom in cancer of the intrinsic larynx. Later there is dyspnea due to a diminished airway, and finally complete aphasia. Metastases to the cervical lymph nodes occur only late in the disease.



Fig 177—Carcinoma involving the base of the epiglottis

In cancer of the extrinsic larynx, the early symptoms are similar to those mentioned for hypopharyngeal tumors. Hoarseness occurs later as a result of secondary involvement of the glottic region. Metastasis to the cervical lymph nodes is a prominent and early occurrence and in a small group of patients may be the first symptom of an extrinsic laryngeal cancer.

The diagnosis of cancer should always be confirmed by biopsy, usually carried out through a laryngoscope.

The *treatment* for early cancers of the intrinsic larynx confined to the vocal cord can be either by a partial laryngectomy (laryngofissure) or by means of fractionated radiation.

therapy. Once the lesion has extended beyond the confines of one side of the intrinsic larynx and has crossed the midline to the opposite side or has extended down into the subglottic region, the most effective treatment is a total laryngectomy.

Cancers of the extrinsic larynx have, in recent years, been treated primarily by a total laryngectomy, and the results with surgery have been much better than with irradiation. When there are associated metastatic lymph nodes in the neck, a radical neck dissection should be performed in continuity with a total laryngectomy.

Following a total laryngectomy a fairly good esophageal voice can be easily learned by most patients. Others prefer to use an artificial larynx, of which there are several varieties on the market.

CANCER OF THE MAXILLARY SINUS

Cancer of the maxillary sinus occurs in males and females in the ratio of about 2:1 and occurs most frequently in the age group 60-70 years. The most frequent variety of cancer in this area is epidermoid carcinoma. Occasionally adenocarcinoma, lymphosarcoma, or fibrosarcoma occurs.

The lymphatics of the antrum communicate with those of the nasal fossa and they end in the retropharyngeal, submaxillary, and the internal jugular lymph nodes.

Most cancers of the antrum begin in the floor of the sinus near the tooth roots. As it grows, the tumor expands the anterolateral wall and the floor of the sinus, causing swelling in the cheeks, palate, and upper gum with loosening of the bicuspids and anterior molars.

The early symptoms may be a toothache or vague pain or discomfort in the cheek. Nasal obstruction or a bloody discharge from the nose may also occur. Later there is invasion of the anterior or nasal walls of the antrum or of the hard palate with swelling of the cheek, and obliteration of the gingivobuccal gutter. Where the tumor arises on the roof of the antrum, there is early invasion of the floor of the orbit, with displacement of the eye upward and outward.

Metastases rarely occur except during the late stages of the disease when the local

growth has not been controlled. The patient is more likely to die from the local effects of the disease than from metastases.

Diagnosis is made primarily by x-ray examination of the antrum and needle puncture biopsy.

Treatment.—Radiation therapy has been used for many years, but the results and the complications of treatment have been discouraging. More recently the trend has been toward the use of radical surgery. If the growth is confined to the antrum, especially the floor, wide resection of the maxilla can be carried out with little risk and little deformity to the patient. If the floor of the orbit is invaded, an exenteration of the orbit should also be included. The surgical approach for these tumors is generally through a Ferguson incision on the face. The resulting defect is covered with a split-thickness skin graft. X-ray or radium is employed in local recurrences. Electrocoagulation of the tumor followed by radium or x-ray has yielded fairly good results in some centers.

THE JAWS

Fractures

(See pages 248-252)

Infections

Infections within and about the jaws arise, in the majority of cases, from local infection within the teeth or sinuses. In only a very small percentage of cases is the infection blood borne from some distant focus in the body. The periodontal membrane or the pulp canal of the tooth is generally the primary focus of infection, which may then spread by direct extension to involve the bone and adjacent soft tissues. In the majority of cases of osteomyelitis of the jaw, there has been recent extraction of an acutely infected tooth. Other less frequent causes of osteomyelitis of the jaw include compound fracture of the mandible, infection of the maxillary antrum, and mental furuncle with direct extension by the lymphatics into bone, as a blood borne infection from a more distant focus of infection, or it may be a result of radium or x-ray necrosis of bone following irradiation of intraoral cancer.

The signs and symptoms of an acute inflammation of the jaw are those of local pain, cellulitis, and swelling. Associated with this are fever, chills, and leukocytosis.

In the mandible the inflammation may rapidly spread to involve the soft tissues of the submaxillary triangle and upper neck, resulting in a Ludwig's angina with its dangers and complications.

The treatment is, first of all, prophylactic, consisting of the judicious use of radiation therapy in the treatment of head and neck cancer. It is especially important to treat dental sepsis and even to extract all teeth within the field of irradiation prior to commencing treatment. It is wise to provide antibiotic coverage in any case where there is to be a tooth extraction for pulp canal or periodontal infection.

The treatment of an established case of osteomyelitis of the jaw requires the immediate use of large doses of penicillin. Surgery is only performed to drain local soft tissue abscesses or to carry out resection of necrotic bone as demonstrated on the x-ray film.

Tumors

Benign and malignant tumors of the upper and lower jaws are uncommon clinical entities, but they are important because they may interfere with the proper functions of eating and speaking, because of their cosmetic appearance and because if not properly treated they may prove fatal to the patient. Most of the tumors are benign in nature. The classification of tumors of the jaws is quite extensive and complicated. We will discuss in this section only the more important and those most frequently encountered.

Benign Tumors and Cysts

It is not always possible to differentiate between a single or multilocular cyst and a benign tumor. In both conditions there is generally a radiolucent area, or there are areas with irregular outlines. Benign tumors tend to expand the jaw as they grow, and the cortex is thinned out or destroyed by pressure, but it is not invaded as in the case of malignant growth. There is neither periosseous reaction nor soft tissue change in the case

of benign tumors and cysts. The majority of benign lesions are asymptomatic in their early stages. Usually the patient's only complaint is a localized swelling over the jaw. The mandible may be so destroyed that a spontaneous fracture may occur. When the other symptoms appear, they are usually the result of pressure of the tumor on adjacent structures or secondary infection. A vague ache or pain may then be a complaint.

The diagnosis is established by a combination of clinical examination, x-ray study, and biopsy. It is vital to differentiate the benign from the malignant lesions, and while x-ray studies will frequently succeed in this regard, it is a biopsy that will finally establish the exact histologic diagnosis and will thus enable the surgeon to plan his treatment properly.

Odontogenic Cysts.—True cysts arising from dental epithelium are called odontogenic cysts. The cysts contain fluid or a semifluid material and have a definite epithelial lining or membrane. The following types occur.

Dentigerous cysts are the most common cysts of the jaw and are always associated with nonerupted teeth which can be readily seen on an x-ray. In the early stages there are no suggestive symptoms other than nonerupted teeth. Later, as the cyst grows, there is expansion of the jaw, and the cortex becomes so thin that it will dent like a ping pong ball on pressure. The cyst lining is formed from the outer wall of the enamel organ.

In follicular cysts, the first symptom is generally a painless swelling of the jaw, which occurs mainly in young patients. This type differs from a dentigerous cyst in that there are no teeth in the cyst, and the cortex will contain the normal number of teeth. It too is lined by epithelium derived from the dental epithelium.

Radicular cysts almost always occur around the root of a tooth and thus differ from dentigerous or follicular cysts. Occasionally they occur at the lateral surface of the root. The cysts contain a straw-colored watery or thick mucoid fluid. On the x-ray film one will generally see a translucent area of varying size, with well-defined margins at the apex of the tooth. The lesion may be large and involve many teeth or invade the antrum

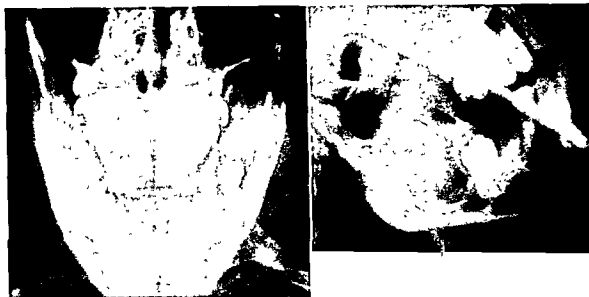


Fig 178—Large dentigerous cyst involving the posterior portion of the left side of mandible
Note the unerupted left first molar tooth

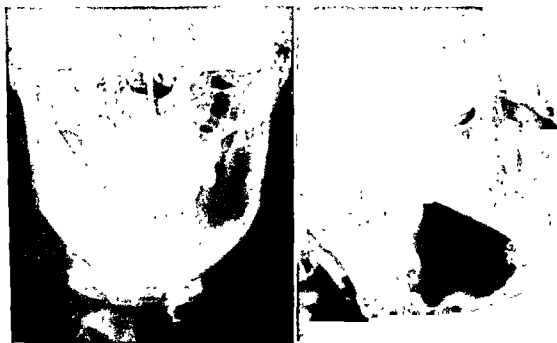


Fig 179—Large cystic type of adamantinoma

Occasionally the cyst may become secondarily infected and rupture into the mouth, discharging a purulent material.

Treatment.—The treatment of all odontogenic cysts is surgical. A mucoperiosteal flap is first raised over the tumor, and the bony wall, if present, is removed. The cysts are then curetted free of their contents and lining membranes and packed open. The mucoperiosteal flap may be partially resutured. The retained tooth in a dentigerous cyst and the involved tooth or teeth in a radicular cyst must be extracted. The packing is removed over a period of several days, and the cavity is allowed to fill with blood and to organize.

Benign Tumors of the Mandible.—Adamantinoma or ameloblastoma arises from the enamel organ epithelium or the ameloblast. The most common symptom is a painless swelling of the jaw which may be continuous for as long as 15-20 years. Because of the lack of pain and the slow insidious growth, the tumors may attain a very great size. They are more frequently found in the mandible than in the maxilla. The tumor is generally irregular in shape and on the roentgenogram may show multilocular cystic cavities. The walls of the cystic cavities may be thinned out and crepitate on palpation. Although the spaces appear cystic, most of them are filled with a solid cellular growth, while the others contain a clear yellow fibrinous or mucoid fluid. While one can frequently suspect the diagnosis of an adamantinoma from careful roentgenologic study, the final diagnosis must always be made by microscopic examination.

Treatment.—It was once felt that these lesions could be treated just as one would treat ordinary cysts of the jaw, that is, by removal of the abnormal epithelium and curettage of the cystlike spaces. This treatment, however, is inadequate and is generally followed by recurrences which will require further treatment or which, in a small number of cases, may become malignant. In small tumors one may carry out a local resection of the mandible, removing a margin of normal bone around the tumor, and yet retain enough of the mandible to prevent fracture and maintain normal bony contour. In larger tumors or in tumors where there is perforation of the bone and extension into soft tissues, one must

carry out a radical resection of the involved mandible. Reconstruction of the mandible by means of bone graft or by various metal prostheses may be used.

Fibrous and Granulomatous Tumors of the Jaw.—This group includes the following lesions: benign giant cell tumor, solitary and generalized osteitis fibrosa, fibrous osteoma or ossifying fibroma, and fibrous dysplasia of bone. These are all destructive central lesions involving the jaws of young people and are considered to be benign. Only rarely do they



Fig. 180.—Benign ossifying fibroma of the mandible.
Note the cystic appearance of the lesion.

become malignant, and seldom do they occur after 30 years of age. Although many of these growths are described as cystic by the radiologist because they appear as radiolucent areas in the bone, the great majority are not truly cystic but fibrous or granulomatous in nature. The cysts, if present, do not have an epithelial lining, as found in cysts of odontogenic origin. The diagnosis is made by means of a careful radiologic study and biopsy. Even radiologically these lesions may appear so much alike that it is impossible to distinguish them with absolute certainty.

These lesions are characterized by a replacement of the central bony structure with a soft tissue which causes marked thinning of the cortex as expansion progresses. In giant cell tumors the growth is largely granulomatous, with many phagocytic and multinucleated giant cells and some fibrous stroma and evidence of hemorrhage. In the localized osteitis fibrosa, the growth is largely fibrous, with some osteoid but no normal bone formation and no giant cells. The serum calcium and phosphorus are normal. The generalized osteitis fibrosa is a manifestation of hyperparathyroidism and is associated with an elevation of serum calcium, a decrease in serum phosphorus, and an increase in the alkaline phosphatase level in the blood. Fibrous osteoma or ossifying fibroma is characterized by a dense fibrous stroma in which there are scattered new bone trabeculae. In fibrous dysplasia there is a proliferation of small spindle cells in a loose connective tissue stroma. In other areas the stroma is composed of dense collagen with little or no cellularity. There may be foci of osteoid formation and also of hyaline cartilage.

Treatment.—Because of the benignancy of these lesions, they should be treated, on the whole, by conservative surgical means. This will consist of simple excision of the growth or curettage of the lesion, with packing of the excavation with bone chips, and occasionally by partial resection of the mandible. In generalized osteitis fibrosa, one must treat the hyperparathyroidism, whether due to an adenoma or hyperplasia of these glands.

Chondroma and Osteochondroma.—Cartilage tumors of the jaws are rare. They occur

in adults and may present with symptoms of pain, swelling, and disability. Radiologically they appear as translucent lesions with or without scattered foci of calcareous tissue. These lesions are capable of recurring or of becoming malignant, and for that reason they should be excised in a radical manner, including a zone of the normal surrounding bone.

Malignant Tumors

Malignant tumors of the jaws may be primary or secondary. The former are by far the most frequent variety. The secondary type may be the result of metastases from cancer of the prostate, thyroid, breast, lung, kidney, gastrointestinal tract, or they may result from the direct extension from a malignant tumor of the lips, tongue, cheek, floor of mouth, or salivary glands.

Primary malignant tumors of the mandible include the following most common varieties: osteogenic sarcoma, chondrosarcoma, fibrosarcoma, Ewing's tumor, multiple myeloma, malignant giant cell tumor, and malignant adamantinoma. No attempt will be made to discuss these tumors as separate entities, since they behave just as they do in other bones.

All these tumors are firm to palpation and cause a dull aching pain early in their onset, followed by swelling and disturbance in mastication. They frequently metastasize early to the lungs and to other distant organs. Early and extensive destruction of bone, especially by the sarcomas, is characteristic and is readily apparent on x-ray examination of the jaw.

Treatment.—Biopsy should be done in all cases in order to establish the exact histologic diagnosis of the malignant lesion. The treatment of choice for primary malignant tumors of the mandible is radical resection of the bone with a wide margin of surrounding tissues. In Ewing's tumor and in multiple myeloma the use of radiation therapy is to be preferred. Reconstructive surgery, as a rule, should be delayed for one year to be reasonably certain that the disease has been locally controlled. The use of rib grafts, iliac crest grafts, metal and plastic bars, and molded mandibular segments have all been used to reconstruct the mandible with varying degrees of success.



Fig. 181.—Palpation of the neck. This should be done with the palmar surfaces of the tips of the fingers and should include an orderly, systematic examination.

A, Palpation of the submental and submaxillary regions.

B, Palpation of the deep cervical lymph nodes along the internal jugular vein.

C, Palpation of the posterior triangle of the neck and supraclavicular region.

THE NECK

There are numerous surgical conditions that occur in the neck. The differential diagnosis of a particular lesion will depend largely on the history and physical examination. The latter includes careful *inspection* and an orderly *palpation* of the various triangles of this region. Local inflammatory areas, visible swellings, sinuses, and fistulous tracks should all be noted.

Palpation is performed with the examiner in front of the patient. The region of the parotid salivary gland is first palpated on each side, swelling of the submandibular salivary gland, if present, is noted and the neck is then searched for enlargement of any lymph nodes. Palpation of the thyroid gland completes the examination (see Chapter 13).

THE CERVICAL FASCIA

Prior to a discussion of the surgical conditions that occur in the neck, the student

should have a thorough understanding of the anatomy of the deep fascia of the neck. The surgical importance of the fascia colli cannot be overemphasized. The cervical fascia is divided into a superficial and a deep layer. The superficial layer of the cervical fascia is thin and attenuated and lies beneath the skin, superficial to the platysma muscle. It is of no particular significance.

The Deep Cervical Fascia.—This fascia is dense and forms a definite structure which invests the principal organs, vessels, and muscles of the neck. It is divided into three layers as follows:

1 The *general investing layer* or the *previsceral fascia*. This layer lies deep to the platysma muscle and completely envelops all the structures in the neck deep to this muscle. It splits to enclose two muscles, the trapezius posteriorly and the sternomastoid anteriorly. It is attached superiorly to the external occipital protuberance, the superior nuchal line, the base of the mastoid process, the zygomatic

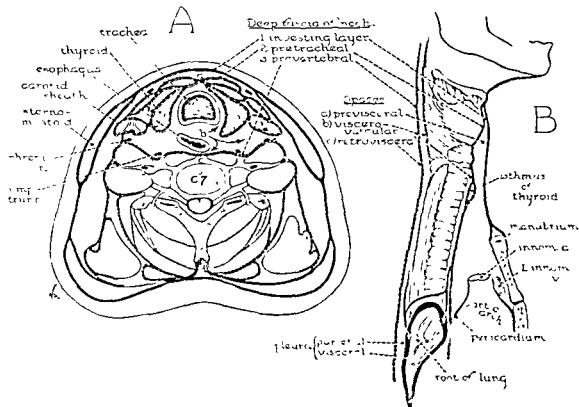


Fig. 182 - The anatomy of the deep fascia of the neck. Note the three layers of fascia and the fact that between the second and third layers there is a free passageway into the superior mediastinum, indicated by the arrows in B.



Fig. 181.—Palpation of the neck. This should be done with the palmar surfaces of the tips of the fingers and should include an orderly, systematic examination

A, Palpation of the submental and submaxillary regions

B, Palpation of the deep cervical lymph nodes along the internal jugular vein

C, Palpation of the posterior triangle of the neck and supraclavicular region

THE NECK

There are numerous surgical conditions that occur in the neck. The differential diagnosis of a particular lesion will depend largely on the history and physical examination. The latter includes careful *inspection* and an orderly *palpation* of the various triangles of this region. Local inflammatory areas, visible swellings, sinuses, and fistulous tracks should all be noted.

Palpation is performed with the examiner in front of the patient. The region of the parotid salivary gland is first palpated on each side, swelling of the submandibular salivary gland, if present, is noted and the neck is then searched for enlargement of any lymph nodes. Palpation of the thyroid gland completes the examination (see Chapter 13).

THE CERVICAL FASCIA

Prior to a discussion of the surgical conditions that occur in the neck, the student

should have a thorough understanding of the anatomy of the deep fascia of the neck. The surgical importance of the fascia colli cannot be overemphasized. The cervical fascia is divided into a superficial and a deep layer. The superficial layer of the cervical fascia is thin and attenuated and lies beneath the skin, superficial to the platysma muscle. It is of no particular significance.

The Deep Cervical Fascia.—This fascia is dense and forms a definite structure which invests the principal organs, vessels, and muscles of the neck. It is divided into three layers as follows.

1. The *general investing layer* or the *previsceral fascia*. This layer lies deep to the platysma muscle and completely envelops all the structures in the neck deep to this muscle. It splits to enclose two muscles, the trapezius posteriorly and the sternomastoid anteriorly. It is attached superiorly to the external occipital protuberance, the superior nuchal line, the base of the mastoid process, the zygomatic

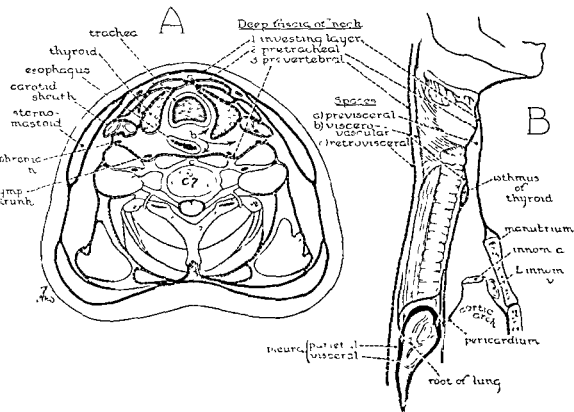


Fig. 182.—The anatomy of the deep fascia of the neck. Note the three layers of fascia and the fact that between the second and third layers there is a free passageway into the superior mediastinum, indicated by the arrows in B.

arch, and the lower border of the mandible. Posteriorly it is attached to the ligamentum nuchae and to the spinous process of the 7th cervical vertebra. It is continuous anteriorly in the midline with the corresponding superficial layer of deep fascia of the opposite side. Below or inferiorly it is attached to the periosteum of the sternum, the clavicle, and the spine of the scapula, and the acromion process. Above, this fascia spreads to enclose the submaxillary and also the parotid salivary gland.

2. The *middle layer* of deep cervical fascia (the *pretracheal fascia* or the *viscerovascular fascia*). This fascial layer is attached to the anterior surface of the trachea. It extends upward in front of the larynx to form the pre-laryngeal fascia and ends above by being attached to the hyoid bone. It gives off a sheath anteriorly to enclose the sternohyoid, sternothyroid, and omohyoid muscles. Inferiorly it envelops the thyroid gland and extends into the mediastinum, where it ends by blending with the fibrous pericardium in the superior mediastinum. This fascia extends laterally with a process given off by the third layer of the deep cervical fascia to completely invest the carotid vessels of the neck. This is known as the carotid sheath. Further laterally, this layer continues as a fascial sheath to cover the scalene muscles and ends by merging with the fascia covering the levator scapulae and the trapezius muscles.

3. The *deep layer* of the cervical fascia (the *prevertebral fascia* or the *retrovisceral fascia*). This fascial layer lies in front of the vertebrae and the prevertebral muscles but behind the buccopharyngeal fascia of the posterior wall of the pharynx and esophagus. It forms the posterior layer of the carotid sheath and runs behind the pharynx, larynx, esophagus, and trachea. Above it is attached to the base of the skull, while inferiorly it extends into the posterior mediastinum where it blends with the endothoracic fascia. Laterally this layer fuses with the fascia covering the scalene muscles and ends by merging with the fascial covering of the trapezius, levator scapulae, and erector spinae muscles. This layer also splits as it extends laterally to form a dome over the apex of the pleura and lung known as Sibson's fascia. *The important point to realize here is that between the middle and*

deep layers of the deep cervical fascia, in the space containing the trachea and esophagus, there is a free passageway into the superior mediastinum.

It is possible for cervical abscesses and infections within the space to track down into the mediastinum, resulting in an acute mediastinitis with or without abscess formation.

In operations on the neck, such as tracheostomy, thyroidectomy, or radical neck dissection, this space is entered, and it becomes possible for air to be sucked down into the mediastinum. This occurs especially in those cases in which there is some obstruction to the airway; the respirations become labored and exaggerated, with an increase in the negative pressure not only in the mediastinum but also in the pleural cavities. This can result in a tension pneumomediastinum, which can lead to a rupture of the mediastinal pleura and a pneumothorax which may be unilateral or bilateral. This complication is an acute surgical emergency and must be recognized and treated as rapidly as possible.

INJURIES

Wounds of the neck are common, the majority of them being due to a suicidal attempt. However, accidental and homicidal wounds also occur. The carotid vessels are protected by the sternomastoid muscle, but deep puncture wounds may easily prove fatal due to hemorrhage. Deep wounds may reach many important structures such as nerves, blood vessels, and air passages. It is interesting that the person attempting suicide generally damages the air passages but rarely touches the contents of the carotid sheath. This is because he holds his head in the hyperextended position, very frequently before a mirror, which projects the trachea and larynx forward. The homicidal wound is more likely to cause damage to the contents of the carotid sheath. The complications of neck wounds are bleeding, infection, pneumonia, and subcutaneous emphysema. Arteriovenous fistulas can result from penetrating wounds of the neck.

Treatment.—First bleeding must be controlled. Packing the wound with gauze can control hemorrhage until the wound can be well exposed in the operating room. The

larynx or trachea may be divided or, if not, may be so badly contused that glottic edema, laryngeal or tracheal necrosis follows. An adequate airway must therefore be maintained, and this may necessitate an emergency tracheotomy.

In cases of laceration of the larynx or trachea, a tracheostomy tube can often be inserted directly through the wound. The neck wound should be débrided in the usual manner and sutured, as should also the lacerated larynx, pharynx, or trachea. A temporary tracheostomy will be necessary, because in such cases one can anticipate edema of the glottis. If the wound is grossly contaminated and there is infection, drainage should be instituted. It is wise to use antibiotics freely to prevent wound infection and also to lessen the danger of respiratory complications.

CERVICAL INFECTIONS

Boils and Carbuncles

Boils and carbuncles on the back of the neck are extremely common. Friction of collars, growth of hair, and the thickness of the skin make this area subject to infection. Here it is hard for infection to burrow to the surface, the furuncles are liable to spread as carbuncles. Boils and carbuncles here should be treated as in other locations.

Acute Infections in the Neck

They are very common and are usually secondary to infections beginning in the scalp, face, mouth, or throat. Secondary lymphadenitis ensues, and the lymph nodes may become enlarged and tender. If infection in a lymph node breaks through the node capsule, it may spread as a cellulitis, contained by the firm fascial planes in the neck. Later there may be abscess formation. It is difficult to decide when an abscess has formed, and there is no advantage in cutting into a node that is still solid. The infection is often so deep that it may be difficult to elicit fluctuation. All that one can feel is, perhaps, a firm indurated mass. By the time there is redness of the skin an abscess has usually formed. Only after a large abscess has formed does the pus break through to the subcutaneous tissues.

Treatment.—Early treatment with antibiotics will often prevent purulent degeneration of lymph nodes, especially if the local abscess in the area is controlled by incision of furuncles or extraction of an abscessed tooth. Cervical abscesses should be incised as soon as they are fluctuant.

Ludwig's Angina

Infection reaching the submaxillary triangle from the mouth, often from an alveolar abscess, may give rise to a spreading cellulitis known as Ludwig's angina. There is a firm and painful swelling in the submaxillary and



Fig. 183.—Ludwig's angina which has been incised (Transverse incision gives less unsightly scar.)

submental regions and in the floor of the mouth, generally with protrusion of the tongue. The inflammation may progress to such an extent as to cause edema of the glottis and death from toxemia and asphyxia. The swelling and inflammation are usually confined to this area by the investing layer of deep cervical fascia.

Treatment.—The use of antibiotics should be instituted just as soon as a diagnosis is made. The patient should be kept under careful observation, and if the edema and swell-

ing appear to be progressive, a curved transverse incision should be made in both submaxillary and submental areas even if no abscess is evacuated. The incision should extend through skin, the platysma muscle, the investing layer of deep fascia, and the geniohyoid and mylohyoid muscles in order to expose completely the submaxillary regions. The wound should be left widely open to ensure free drainage. In rare advanced cases, tracheostomy may be indicated to relieve embarrassed respiration.



Fig. 181—Recurrent tuberculous abscess of the neck

Tuberculosis

The most frequent site of lymph node tuberculosis is in the neck. Tuberculous lymphadenitis is the most common chronic inflammation of these lymph nodes and one of the most common causes of a benign cervical tumor or swelling. Although supervision of milk supply and more rigid control of human tuberculosis have greatly reduced the incidence of cervical lymph node tuberculosis, the disease is still not rare. Early in the disease, one or several movable, discrete, rubbery lymph nodes may be palpated in any of the cervical triangles. In some cases, chains of matted nodes develop which seem to have

no tendency to undergo caseation. In most cases, as the nodes become progressively larger, they tend to caseate and become fixed to the surrounding tissues and to the skin. Late there may be secondary infection, with redness of the skin and spontaneous rupture of the abscess. Following rupture a sinus may persist. The lesions are usually painless until secondary infection appears. Fever, and loss of weight may be entirely absent. While tuberculous of the lymph nodes is generally considered to be a disease of youth, this disease is not infrequently found in those of advanced years, especially in women. X-rays may show calcification in the lymph nodes.

Treatment.—The treatment of tuberculosis has been changing during the past few years, and further changes are likely to occur. Local enlargement of a lymph node in the neck is only one manifestation of tuberculosis that exists elsewhere in the body, but this may not be necessarily so.

A diagnosis having been established, general treatment should be initiated. This includes plenty of rest, sun, and nutritious food. In recent years more or less specific anti-tuberculosis drugs have been developed, and it is not unlikely that still more efficient drugs or antibiotics will be available soon. Among the more effective antituberculosis drugs available today are streptomycin, para-aminosalicylic acid, and isonicotinylhydrazine. It appears that combined drug regimens have proved to be superior to the administration of a single antituberculosis agent in the treatment of this disease. The three best regimens investigated to date are (1) streptomycin combined with daily para-aminosalicylic acid; (2) intermittent streptomycin combined with daily isonicotinylhydrazine; and (3) a combination of daily isonicotinylhydrazine and para-aminosalicylic acid.

Of these three combinations, streptomycin and isonicotinylhydrazine appears to be the most useful. However, the other combinations of drug therapy are also effective and offer certain advantages. As a general rule, the patient should continue to receive the antituberculosis drugs until the disease reaches the inactive stage, or longer. This may sometimes take 8-12 months and, in certain cases, as long

as 18 months. Occasionally it is wise to combine drug therapy with x-ray or surgical measures. In the stage of lymph node proliferation and before caseation, x-ray therapy will often cause regression of the inflammation. In the stage of caseation and before secondary infection, local excision of degenerative lymph nodes is indicated to prevent sinus formation and to promote rapid healing. After secondary infection the abscess should generally be opened and the lymph node remnant curetted. In rare instances, one may be justified in carrying out a radical block dissection of the entire lymph node system on one side of the neck where the nodes are diffusely involved with tuberculosis and have resisted other forms of therapy. However, this should only be performed as a last desperate attempt to rid the patient of the disease.

Actinomycosis

Actinomycosis is a disease affecting man, cattle, and swine and is caused by the ray fungus, *Actinomyces bovis*, which, having lodged in the tissues, produces a granulomatous type of reaction. This reaction is characterized by inflammation, sinus formation, and the discharge of characteristic yellow sulfur-like granules. The majority of cases of human actinomycosis are found in the face and neck. The primary focus of entry is usually in the mouth, around the teeth, and in the crypts of the tonsils. The first sign of neck swelling may follow extraction of a tooth or a tonsillectomy. Abscesses soon form and break down to give rise to discharging sinuses. The fresh pus from a newly opened abscess will often contain the yellow sulfurlike granules of the actinomyces colonies. If untreated, the disease continues to burrow and produce new thick-walled abscesses and sinuses. Gradually the patient becomes thin and weak with further local spread of the disease and extension to other parts of the body, such as the liver, lungs, and brain.

Treatment.—Combined therapy of penicillin and sulfadiazine has proved most effective in many cases. It may be necessary to continue this treatment for several months. Recently the use of stilbamidine intravenously

in doses of 150 mg./day for about 12 days has shown some promise in the treatment of this disease. The use of x-ray therapy to accessible lesions has long been used with results that have been generally satisfactory. A saturated solution of potassium iodide is the traditional and sometimes the only effective means of controlling this disease. One should generally begin with an initial dose of 5 drops in water 3 times a day before meals and increase it by 5 drops daily until signs of intolerance develop, which is usually somewhere between 200 and 300 drops per day. The dosage just below that which produces symptoms is the maintenance dose and should be given for several weeks or several months, depending upon the response of the patient. Probably the most effective method of treatment is surgical. This consists of excising or curetting all diseased or dead tissue, leaving the wound open in order to provide adequate drainage. This should usually be combined with intensive doses of some form of chemotherapy as previously outlined.

TUMORS OF THE NECK

In Table 12 are listed the most common benign and malignant tumors that may be responsible for a so-called "lump in the neck." It is surprising how frequently a patient will present himself to his physician with a slowly enlarging swelling in the neck as his only complaint. It is often very difficult to decide whether the lesion is due to some chronic inflammation, benign tumor, or local or metastatic neoplasm. The commonest error in such cases is the failure of the physician to recognize the probable cancerous nature of most cervical tumors in an adult. Generally speaking, the diagnosis in a given case can be made from the following facts: a careful history which should include the age and sex of the patient, the duration of the tumor, its rate of growth, the presence or absence of pain, tenderness, or local inflammation, history of previous operations or treatment. One should then carry out a very careful physical examination of the lump, its location, consistency, size, the presence or absence of pain or tenderness, whether or not it is single, and so on.

TABLE 12
CLASSIFICATION OF BENIGN AND MALIGNANT TUMORS OF THE NECK

BENIGN TUMORS OF NECK	MALIGNANT TUMORS OF NECK
(A) <i>Inflammatory</i>	1. Metastatic cancer
1. Hyperplasia of cervical lymph node	2. Malignant lymphoma
2. Tuberculosis	(a) Lymphosarcoma
3. Actinomycosis	(b) Hodgkin's disease
4. Boeck's sarcoid	(c) Leukemia
5. Boils and carbuncles	3. Branchiogenic carcinoma
(B) <i>Congenital</i>	4. Sarcoma of soft tissues of neck
6. Thyrocolossal cyst	(a) Fibrosarcoma
7. Branchiogenic cyst	(b) Liposarcoma
8. Dermoid cyst	(c) Malignant schwannoma
9. Cystic hygroma	(d) Rhabdomyosarcoma
(C) <i>Acquired</i>	5. Thyroid carcinoma
10. Carotid body tumor	6. Malignant salivary gland tumors
11. Benign thyroid adenoma	7. Parathyroid carcinoma
12. Benign submaxillary salivary gland tumors	
13. Soft part tumors	
(a) Lipoma	
(b) Fibroma	
(c) Sebaceous cyst	
(d) Schwannoma	
(e) Ganglioneuroma	
14. Parathyroid adenoma	

A careful examination of the mouth, larynx, pharynx, nasopharynx, and tonsillar region for a possible silent primary focus should be performed. One negative examination should not rule out the possibility of a primary lesion in one of those sites, since the primary growth may be so small that it is completely missed. Repeated examinations at regular intervals and by several examiners are necessary before one can be certain that this region is free of disease.

In less obvious and somewhat bizarre swellings, the diagnosis may prove to be most elusive. In such cases one should resort to complete x-ray studies of the chest, gastrointestinal tract, and kidneys. Complete blood and biochemical examinations are necessary. The blood picture may furnish information which would help to distinguish between a primary tumor and acute or chronic inflammatory swellings of the lymph nodes.

A serious and fairly common error in the management of cervical tumors or lymph node enlargements is the immediate excision or incision of a "lump" for biopsy purposes before any of the above diagnostic procedures have been carried out. Such local operative procedures diminish the prospect of cure in cases

of malignant tumors, since the resulting scarring may interfere with the subsequent treatment of this neoplasm either by surgery or with irradiation. In difficult cases aspiration biopsy of a lump in the neck is frequently successful in rendering a diagnosis of an abscess, cyst, benign tumor, or malignant neoplasm. This method is simple to perform and does not interfere with subsequent treatment of the area by whatever means prove necessary. Incisional or excisional biopsy should only be used as a last resort after other diagnostic procedures have failed.

BENIGN TUMORS OF THE NECK

Inflammatory Tumors

This group includes hyperplasia of cervical lymph nodes, pyogenic and tuberculous lymphadenitis, actinomycosis, Boeck's sarcoid, boils, and carbuncles. With the exception of Boeck's sarcoid, these have already been discussed and will therefore not be dealt with further.

Boeck's Sarcoid

Boeck's sarcoid is a chronic, indolent, benign infectious disease of unknown cause, which may involve the lymph nodes, the salivary glands, lungs, or other organs. The

systemic symptoms are usually not severe in the early stages, and they may be so mild that only skin eruption on the face or enlarged lymph nodes may be noted by the patient. When cervical lymph nodes are enlarged, they are rarely more than one inch in diameter. The nodes are discrete and elastic, they never caseate or suppurate, but they are involved in a granulomatous inflammation. Preauricular and postauricular lymph nodes are frequently enlarged. There may be a hypochromic microcytic anemia. When general lymphadenopathy and splenomegaly are present, the disease may

Thyroglossal Cysts and Fistulas

The embryology of this condition is discussed elsewhere. The diagnosis of thyroglossal cysts is generally simple. Most patients will present with a cystic swelling or mass in the anterior neck usually just below the level of the hyoid bone. While they tend to occur in the midline, they are not infrequently found either just to the right or to the left of the midline. Thyroglossal cysts average 3 or 4 cm but may be as large as 10 cm in diameter. They are not painful or tender unless there is secondary infection. A characteristic feature

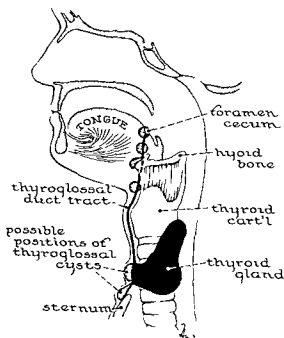


Fig 185—The position taken by the thyroglossal duct in its migration from the foramen cecum to the lower anterior neck. Thyroglossal cysts may occur anywhere along its course. The tract may run anterior to or through the body of the hyoid bone.



Fig 186—Large thyroglossal cyst, characteristically situated in the midline of the neck near the hyoid bone.

be confused with Hodgkin's disease. After a considerable time there is usually spontaneous recovery. A biopsy must be taken to settle the diagnosis. The value of all treatments is doubtful, but x-ray therapy and nitrogen mustard may have some value. Half the patients have enlarged lymph nodes.

Congenital Tumors

This group includes thyroglossal cyst and branchiogenic cyst and fistula, cystic hygroma, and dermoid cysts.

of such cysts is a tendency to move upward on swallowing or on protrusion of the tongue. In about one third of the patients there is a draining sinus instead of a cyst. Such a sinus may have its opening in the anterior neck anywhere from the suprasternal notch to the region of the hyoid bone. The sinus opening results from spontaneous rupture or surgical drainage of an infected cyst.

The treatment is surgical and includes not only excision of the cyst with any external fistulous tract but also tracing and completely

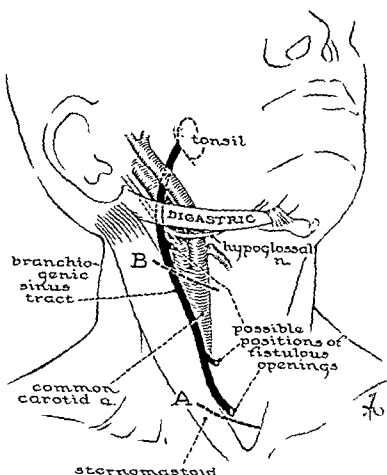


Fig 187--Diagram to show the course and anatomic relations of the branchiogenic sinus tract. The internal opening is generally in the tonsillar region or, in rare instances, in the nasopharynx. A and B illustrate the lines of incision utilized when excising the tract in step-ladder fashion.



Fig 188--Branchiogenic cyst in a young adult, situated in upper lateral neck, adjacent to the anterior border of the sternomastoid muscle.



Fig 189--Infected branchial cyst with abscess formation.

resecting the thyroglossal duct to its point of origin at the base of the tongue. Since it is almost impossible to determine whether the tract runs in front of, behind, or through the substance of the hyoid bone, one must always remove the central portion of the hyoid bone as part of the dissection of the tract, otherwise recurrence may result.

Branchiogenic Cysts and Fistulas

In the embryo there are 5 branchial arches with 4 branchial grooves or clefts between them. The arches develop to form the upper and lower jaws, parts of the hyoid bone, the styloid process, part of the stapes, and most of the thyroid and cricoid cartilages. The branchial clefts and furrows form the external auditory meatus, the eustachian tube and tympanic cavity, the thymus and parathyroids. The major parts of the branchial clefts normally disappear, but they may persist in whole or in part.

The most common remnant of the branchial clefts is in the form of a branchial cyst. Branchial cysts occur with equal frequency in both sexes and usually appear in early adult life in patients 20-30 years of age. They are most frequently found in the upper third of the neck in close association with the anterior border of the sternomastoid muscle. Such cysts vary from 1-10 cm in diameter and give a history of slow steady increase in size. They generally present as smooth, movable, soft, and painless masses. Such cysts may be lined either by squamous or by stratified columnar ciliated epithelium. In those lined by squamous epithelium, the cyst contents is usually a thin, milky fluid, and if columnar epithelium is present, the cyst contents will generally be a thick, transparent, and mucoid fluid. There may be a mixture of both types of epithelium and a combination of both types of fluid. The finding of cholesterol crystals in aspirated material is diagnostic of branchial cysts.

The treatment of such cysts is excision. The use of sclerosing solutions, x-rays, repeated aspirations, or of incision and drainage is only mentioned to be condemned. An oblique incision centered over the most prominent part of the tumor is employed. The cyst should be completely enucleated, and in so

doing one must watch for possible injury to such structures as the mandibular branch of the facial nerve, the carotid vessels, the vagus nerve, the hypoglossal nerve, and the cervical sympathetic chain. The wound is drained from its inferior angle for 24-48 hours.

Branchial Fistulas.—Persistence of a branchial cleft may form a fistula on one or both sides of the neck. This condition again occurs equally often in both sexes, but unlike cysts this condition is generally seen at birth or shortly thereafter, and the majority of the cases are seen before the age of 5 years. The external opening of the fistula is usually found in the lower third of the neck and is usually in close association with the anterior border of the sternomastoid muscle just above the clavicle. Sometimes the fistula may open in the middle or even in the upper third of the neck. If an internal opening is present, it is found near the tonsillar fossa, but the tract may be incomplete. Periodic opening and closing of the fistula may be present, although most of the time, there is some discharge from the external fistula. Secondary infection and abscess formation may occur. Injection of Lipiodol into the fistula is used to show the length of the tract and to demonstrate the internal opening in the pharynx.

Treatment.—Branchial fistulas should be excised except in the very young or in the presence of active inflammation. The operation consists of excising the entire tract from the external skin opening to the entrance of the fistulous tract in the tonsillar region. When the opening is in the lower third of the neck, it may be necessary to use multiple transverse incisions in stepladder fashion, in the lateral part of the neck in order to extirpate the tract. The tract usually passes up between the internal and external carotid arteries to the wall of the pharynx. Its entrance into the pharynx is ligated with fine silk. The wound is drained for 24-48 hours.

Cystic Hygroma

This benign, congenital, multilocular cystic tumor of lymphatic origin presents in the lateral cervical region. According to Goetsch, cystic hygroma of the neck arises from sequestration of the lymphatic tissue derived from



Fig 190—Dermoid cyst of face

the primitive jugular sacs which have failed to join the lymphatic system in the normal manner. While the majority of cases occur in the neck, this lesion is also occasionally seen in the axilla, the retroperitoneal space, and the groin. The tumor is generally seen before the age of 10 years, although about 50% of the cases are noted immediately after birth. The tumor may be large and extend from the mastoid process to the clavicle, or it may be limited to a small area in the posterior triangle of the neck or in the supraclavicular region. The cysts forming the tumor are thin-walled cavities which vary greatly in size and are lined by endothelium and contain a clear watery or straw-colored fluid. The tumor has a soft irregular cystic consistency.

The treatment of cystic hygroma is primarily surgical and consists of wide surgical excision of the lesion. The use of radiation therapy and the injection of sclerosing solutions, although widely used in the past, is of limited value only.

Dermoid Cysts

True dermoid cysts are of congenital origin. They may arise from the embryonic germinal

epithelium or be produced by epithelial remnants caught in the lines of embryonic fusion. Dermoid cysts occur in a number of locations. The most common sites are the following:

1. In the midline beneath the tongue or in the floor of the mouth
2. In the midline of the neck near the hyoid bone
3. In the mid-portion of the lateral aspect of the neck, near the sternomastoid muscle
4. In the submaxillary triangle
5. In the face—most frequently found at the root of the nose, or on the forehead at the outer border of the eyebrow

The cysts vary in size from 2 to 5 or 6 cm in diameter. They are usually oval or circular in shape, rubbery in consistency, and not adherent to the skin, although they may be fixed to the underlying tissue. They are lined by stratified squamous epithelium, and sebaceous glands, sweat glands, and hair follicles may be present in the walls of the capsules. Cysts in the floor of the mouth seldom contain hair, although those elsewhere generally do. They are painless lesions and grow slowly. They can be easily mistaken for thyroglossal or branchial cysts, although if the contents of

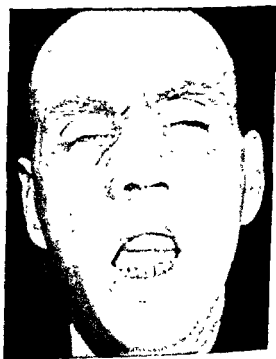


Fig 191—Dermoid cyst of submental region which has been growing slowly for 20 years causing elevation of tongue and interference with eating

the cysts is aspirated, the diagnosis can be generally suspected. The contents of a dermoid cyst varies in consistency from thin to very thick and is composed of desquamated epithelium, sebaceous material, and hair. Dermoid cysts occasionally become infected, forming small abscesses or sinuses from which hair may protrude.

Treatment—Dermoid cysts should be removed surgically. The cyst usually shells out from the surrounding tissue quite readily unless there has been secondary infections or previous surgical interference, in which case there is dense adhesive scar tissue about it. On the scalp or face, the cyst may be attached to the underlying periosteum or even to the dura mater, and therefore removal should be carefully planned.

Acquired Cervical Tumors

This group contains such lesions as carotid body tumor, benign lipoma, sebaceous cyst, benign thyroid adenoma, benign submaxillary salivary gland tumor, schwannoma or neurilemmoma, and ganglioneuroma.

Carotid Body Tumor

Of the benign acquired cervical tumors the most important is the *carotid body tumor*. This is a relatively rare tumor that occurs in the lateral region of the neck just below the angle of the mandible and is situated beneath the sternomastoid muscle at the level of the bifurcation of the common carotid artery. These tumors can occur at any age, although they are more frequently found in middle life and occur equally often in both sexes. The carotid bodies, from which this tumor arises, are paired structures situated within the adventitia of the carotid artery near the bifurcation. The exact function of the carotid bodies is still not clear. They may be chemoreceptors which are sensitive to changes in the hydrogen ion concentration and to the carbon dioxide and oxygen tensions of the circulating blood.

These tumors generally grow slowly and in the early stages produce little or no discomfort. As they grow larger, however, they may press against adjacent organs or structures and give rise to symptoms. The tumor may grow upward toward the base of the

skull, inward toward the tonsillar region and pharynx, or outward, producing a bulge in the neck. The characteristic feature of these tumors is the fact that they can be moved in a horizontal but not in a vertical plane, due to their attachment to the carotid vessels. The tumors are occasionally bilateral. A carotid body tumor may lie either on or behind the bifurcation of the common carotid artery, or it may completely encircle the common, internal, and external carotid arteries. It always gives a transmitted pulsation and occasionally exhibits a bruit. Carotid angiography may confirm the diagnosis by findings suggestive of intrinsic defect in the carotid wall.

Treatment.—Although it was once believed that these tumors were always benign, there have been recent reports in the literature to prove that these tumors are capable of regional lymphatic spread as well as distant dissemination. Because of this fact and because if left untreated they continue to grow and cause trouble locally, removal of carotid body tumors is recommended if at all possible. The chief danger of operating upon these tumors is the fact that the carotid vessels may be completely surrounded by the growth, and in order to rid the patient completely of the tumor, it may be necessary to sacrifice the region of the bifurcation of the common carotid artery. Older patients do not as a rule tolerate this procedure, and in such people it is attended by a high morbidity (hemiplegia) and also a high mortality rate of about 45%. In recent years, however, it has been possible to re-establish the continuity of the internal carotid artery by means of nylon prostheses or venous or preserved arterial homografts. There have also been cases reported in which the distal cut end of the external carotid was successfully anastomosed to the distal end of the internal carotid artery. In such cases the blood flow is said to occur in a retrograde manner from the external carotid artery into the internal carotid artery. This tumor is radioresistant, and no regression can be expected with radiation therapy.

Primary Neurogenic Tumors

Primary nerve tumors of the neck include the following: neurofibromas, schwannomas or neurilemmomas, and ganglioneuromas.



Fig. 192.—Lipoma of the neck

Neurilemmomas are said to be commonest in the head and neck area. They arise most frequently from some portion of the brachial plexus in the neck and are usually seen in the supraclavicular region in the posterior triangle of the neck. They are also rarely encountered in the parapharyngeal region. In this region they arise, most usually, from the vagus or cervical sympathetic nerves. The tumor may occur at any age and the sexes are equally involved. They are usually benign and rarely do they become malignant. The tumors are usually 2-3 cm. in size, but they may reach 5-6 cm. in diameter. They often produce paraesthesia in the arm and hand on the affected side. Those arising in the parapharyngeal region may produce compression of the posterolateral pharyngeal wall with resultant dysphagia and sometimes hoarseness.

Ganglioneuromas arise from the ganglia of the sympathetic nerve trunk. They are more capable of becoming malignant and metastasizing than the previously discussed neurogenic tumors. The majority of ganglioneuromas are thoracic in origin and spread upward into the neck from the mediastinum. They may occur at any age and are found in both sexes.

Treatment.—The treatment of primary neurogenic tumors is surgical excision. They

do not respond to irradiation. Since most of these tumors are well encapsulated, they can be readily shelled out. One must be careful not to injure any of the nerve trunks when dissecting them from the brachial plexus or from the vagus nerve or sympathetic trunk.

Lipomas

The cervical region is the commonest site of lipomas. They are especially seen in the posterior triangle and the back of the neck. Lipomas are soft, painless tumors that grow slowly and cause few symptoms. The more superficial lipomas are usually easy to diagnose, but those that occur in the deeper tissue of the neck may feel quite firm and prove difficult to diagnose except by surgical excision.

Treatment.—Lipomas should be surgically excised both for cosmetic and diagnostic reasons. Furthermore, it is quite possible for deeper lying tumors to become malignant liposarcomas.

Sebaceous Cysts (see Face)

MALIGNANT TUMORS OF THE NECK

Malignant tumors of the neck can be divided into two main groups:

1. Primary malignant tumors of the neck
2. Metastatic or secondary malignant tumors of the neck

By far the most frequent type of malignant tumor in the cervical region is that due to

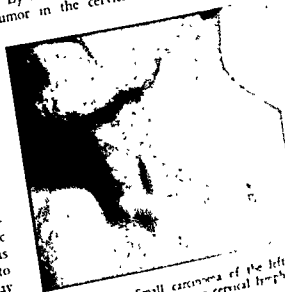


Fig. 193.—A, Small carcinoma of the left lobe of the thyroid gland. B, Large cervical lymph node metastasis.

metastasis in the cervical lymph nodes. The primary sites are usually in the head and neck region but may also be occasionally below the clavicle.

Primary Malignant Tumors

We have excluded from this discussion primary malignant tumors of the thyroid, parathyroid, and salivary glands because these lesions are more conveniently discussed elsewhere. This leaves for discussion the following most frequently encountered types of primary cancer of the neck:

1. Malignant lymphoma which includes all those primary malignant lesions arising in cervical lymph nodes, i. e.
 - (a) lymphosarcoma
 - (b) Hodgkin's disease
 - (c) leukemia
2. Branchiogenic carcinoma
3. Sarcoma of the soft tissues of the neck

Malignant Lymphomas

Lymphosarcoma.—Lymphosarcoma is a progressive disease which generally begins with enlargement of a solitary lymph node in the neck. This solitary tumor may persist for some time, but eventually other nodules appear, gradually enlarge, and coalesce to form a matted chain of lymph nodes without suppuration. We include in our term lymphosarcoma the three generally recognized varieties, namely, lymphocytic, reticulum cell, and the macrofollicular types. Clinically all three types behave alike. It is almost impossible to distinguish, by clinical examination alone, lymphosarcoma of the neck from other varieties of malignant lymphoma or from metastatic cervical carcinoma. In some cases it may be possible to make a diagnosis on aspiration biopsy, but usually the pathologist will request an excisional biopsy in order to be absolutely certain of the diagnosis.

Hodgkin's Disease.—Although at one time Hodgkin's disease was considered to be an infectious process, it is now generally accepted as a form of malignant neoplasm. Again the first symptom of this disease is usually a painless swelling in the lateral part of the neck, which behaves very much like a lympho-

sarcoma. The number of lymph nodes involved in Hodgkin's disease is generally more than in lymphosarcoma, and the lymph nodes are usually firmer with more induration. As with lymphosarcoma, it is difficult to distinguish Hodgkin's disease from tuberculosis, cancer, or other forms of lymphoma. Suppuration does not appear in Hodgkin's disease. A positive diagnosis is made after biopsy of the mass. The disease is more frequent in males than in females, the course runs from months to many years but is usually fatal in 2-4 years. In the final stages there is the typical fever of Pel-Ebstein, loss of weight, and weakness. The spleen and liver may be enlarged, and x-rays of the chest may show a widening of the mediastinum due to involvement of the mediastinal lymph nodes. This type picture is variable, but anemia and leukopenia are frequently present.

Lymphatic Leukemia.—This condition is frequently associated with cervical lymph node enlargements and has the same general clinical characteristics as the lymphomas. Matting of these lymph nodes is less common than in Hodgkin's disease. Histologically, this tumor may resemble a lymphosarcoma, and for that reason a white blood count should always be made in addition to biopsy of a lymph node before a conclusive diagnosis of lymphosarcoma is made.

Treatment of Malignant Lymphoma.—Lymphosarcoma is one of the more radio-sensitive malignant tumors, and in most cases the initial response to radiation therapy will be good. In recent years there has been evidence to show that many of the cases of cervical lymphosarcoma are secondary to a single, discrete primary lesion in some portion of Waldeyer's lymphatic ring in the nasopharynx, palate, tonsil, and base of tongue. It remains localized at first and then later metastasizes to the cervical lymph nodes as does a squamous cell carcinoma. Therefore one should never treat a lymphosarcoma of the cervical lymph nodes without first carefully examining the mouth and pharynx for a possible primary focus. If present, then the primary as well as the secondaries in the neck should be treated with irradiation. In a small number of cases of lymphosarcoma in which the disease appears to have originated either

primarily in the lymph nodes of one side of the neck or to have subsequently appeared in the neck following control of a primary focus in the mouth or throat, it is permissible to attack such disease by radical neck dissection. These cases must be carefully selected, and every effort must be taken to demonstrate that there are no other areas of involvement before a radical neck dissection is performed. If there is evidence of disease anywhere else in the body, then x-ray is the treatment of choice since it indicates that the disease is no longer local but has become generalized. Postoperative x-ray therapy to the neck may be given.

Hodgkin's disease, like lymphosarcoma, is also a radiosensitive lesion. While the neck nodes are the first involved in about 60% of all cases of Hodgkin's disease, generally speaking by the time the patient presents himself for treatment, or a diagnosis is made, the disease is usually widespread. The use of nitrogen mustard and its derivatives, combined with radiation therapy, appears to be the most effective form of therapy in this group of cases. In early cases of Hodgkin's disease where the nodes involved are confined to one side of the neck with no evidence of disease elsewhere in the body, a radical neck dissection is justified, followed by postoperative x-ray therapy.

Branchiogenic Carcinoma

Branchiogenic carcinoma refers to those malignant tumors which are believed to arise in vestigial remnants of branchial pouches. This term has been applied to those cases of malignant epithelial tumors appearing in the lateral aspects of the necks of patients in whom there is no evidence of a primary focus elsewhere in the body. In most hospitals this term is used less today because, with experience, it has been shown that the majority of such malignant tumors in the neck are metastatic from the mouth, throat, accessory sinuses, the thyroid, the major salivary glands, or even from a primary focus below the level of the clavicle. At times the primary focus is so minute that it may be completely missed on initial examination. Indeed it may not reveal itself clinically for several years. Because of this fact, the diagnosis of branchiogenic cancer should always remain tentative until the

patient has survived for five years after the treatment of the growth in the neck, without the discovery of a primary lesion elsewhere.

Treatment.—The diagnosis can be made by an aspiration biopsy or by an incisional or excisional biopsy of the mass. In those cases where the tumor is less than 3 cm in diameter, it is best to treat the lesion by means of radiation therapy. One hesitates to carry out radical surgery in these cases knowing that there is a possible hidden primary focus elsewhere in the mouth or throat which may later become obvious. When the cervical tumor is larger than 3-4 cm. in diameter, the possibility of controlling it by means of radiation therapy is much less successful, and in such cases it is perhaps more advisable to carry out a radical dissection on the affected side. Regardless of the method of treatment, the patient must be followed carefully and at frequent regular intervals in search of a possible primary lesion.

Sarcomas of the Soft Tissues of the Neck (Excluding Lymph Nodes)

Sarcomas may arise from practically any of the soft tissues of the neck, such as nerves, muscles, fascia, and vascular tissue. Such tumors may be situated along the line of the internal jugular vein and consequently may be readily confused with metastatic cervical cancer. The following types of tumors have been reported from this region: fibrosarcoma, liposarcoma, rhabdomyosarcoma, leiomyosarcoma, hemangiopericytoma, malignant schwannoma, and malignant ganglioneuroma. The diagnosis in any given case will ultimately depend upon the histologic report. Aspiration biopsy may be sufficient, but it may be necessary to make an incisional biopsy. The treatment of sarcomas, in general, is best carried out by radical surgical procedures since they are, on the whole, resistant to radiation therapy.

Metastatic or Secondary Carcinoma of the Neck

The most frequent type of cancer in the neck is that due to metastases in the cervical lymph nodes from a primary site outside the neck. It must be remembered that mere pal-

pability of cervical lymph nodes is by itself no proof of abnormality. Lymph nodes up to 2 cm. in diameter in the subdigastric area and up to 1.5 cm. in the submaxillary triangle may exist bilaterally for years in perfectly healthy people. But such bilateral symmetric, soft enlargement of lymph nodes is of little importance. It is the unilateral or asymmetric enlargement which is of more clinical significance. Cancer and tuberculosis are the commonest causes of chronic asymmetric enlargement of cervical lymph nodes, and yet, of the two, metastatic cancer is said to be about 200 times commoner than tuberculosis. Inflammatory hyperplasia of lymph nodes may also give rise to asymmetric cervical lymph node enlargement. The diagnosis in such cases is not too difficult since the nodes are generally tender and show signs of inflammation, and on careful examination one will find a focus of infection in the mouth or throat, most often in the tonsils or about the teeth. Furthermore, such lymph nodes tend to suppurate and result in abscess formation, as previously discussed. In cervical lymph node metastases, however, the lymph nodes are generally firmer in consistency. In the early cases there is a characteristic lack of pain or tenderness or any signs of acute inflammation, while finally there is a definite tendency toward steady and progressive enlargement.

The location of the cervical metastases depends on the site of the primary tumor. The first sign of a cancer of the stomach, rectum, prostate, or uterus may be the presence of a firm, enlarged lymph node above the clavicle. A metastatic lymph node in either supraclavicular area may be an indication of the inoperability of a carcinoma of the bronchus, the esophagus, or breast. An enlarged cervical lymph node may be the first obvious spread from a cancer of the testis. Martin states that the appearance of a cervical metastasis was the only symptom noted by the patient in about 85% of all cases of primary cancer occurring in organs situated above the clavicle, such as in the mouth, throat, and thyroid. When a patient with a lump in the neck presents with an ulcerating cancer of the lip, oral mucosa, tonsil, or larynx, then the diagnosis of the cervical tumor is obvious. In other instances, however, the primary growth

is not so apparent, and one must carry out a very careful examination of the base of the tongue, pharynx, extrinsic larynx, nasopharynx, and the tonsillar region. X-ray examinations of the paranasal sinuses, chest, gastrointestinal tract, and kidneys may all be required before the primary focus is finally discovered. Endoscopic examination of the larynx, the bronchial tree, and the esophagus may prove invaluable. The thyroid gland itself should not be neglected, and the presence of even the smallest nodular enlargement in any portion of the gland should be considered significant.

As the original mass in the neck grows, other lymph nodes gradually enlarge and become palpable and firm. A chain of firm nodules soon appears, and if left untreated the involved nodes eventually become matted together and fixed to the skin and surrounding tissues. Later the cancer may grow into the skin, forming an ulcer with secondary infection and foul discharge. Pain is not an early symptom but occurs later as a result of either secondary infection and ulceration or because of secondary involvement of the sensory nerves or from deep infiltration into adjacent structures. Cancers in the upper cervical areas may cause agonizing pain referred to the ear.

Treatment.—There are two main methods of treating cervical metastatic cancer, namely, radiation therapy and neck dissection. It is now generally accepted that wide removal of the cervical lymphatics offers a better chance of cure than does radiation therapy. However, the method employed in any given case will depend upon several factors, the most important being the following:

1. The location of the primary lesion and whether or not it has been controlled by previous therapy.
2. The histopathology of the malignant lesion.
3. Whether surgical treatment of the metastases will afford a reasonable chance of success.
4. Whether there is clinical or radiographic evidence of distant metastases.

Obviously, before surgery is contemplated, the primary focus, generally somewhere in the head and neck region, must be controlled. If not, it should be possible to remove it

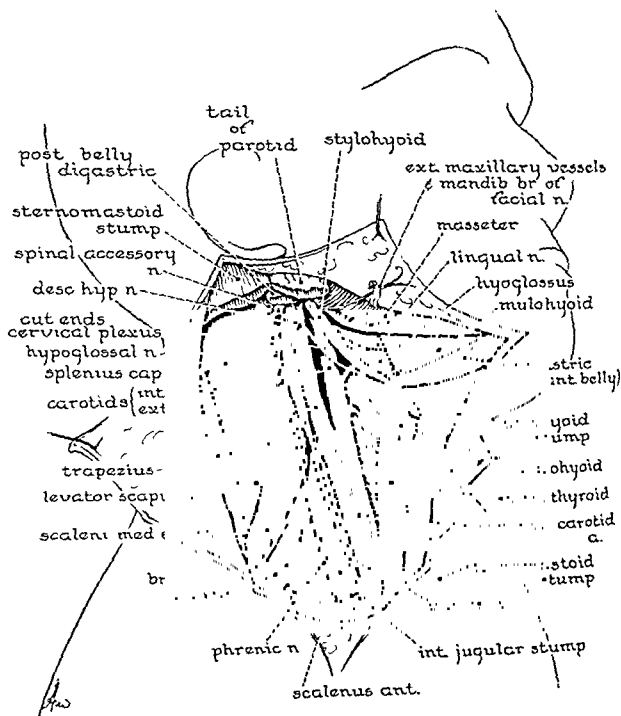


FIG 194.—Neck dissection completed. The operation extends from the midline of the neck to the anterior border of the trapezius muscle and from the clavicle below to the mandible above. Included in the dissection are the sternomastoid muscle, the internal jugular vein, the carotid sheath, the omohyoid muscle, the submaxillary gland and all the deep lymphatics, fatty and areolar tissue in the lateral and anterior aspects of the neck.

surgically at the same time the neck dissection is performed. Otherwise, radiation therapy offers a better chance for cure. The majority of neck metastases are secondary to squamous cell cancer of intraoral origin, and they are relatively radioresistant. However, metastatic lymphosarcoma or lymphoepithelioma, although less frequent, is generally better handled by radiation therapy because of their radiosensitivity.

Before attempting a radical neck dissection, the metastases must be clinically operable, and one must be reasonably sure of success. The main criterion of local inoperability is extension into the carotid vessels, the base of the skull, or the cervical vertebrae. In a small percentage of cases, what was originally felt to be clinically fixed and inoperable may undergo such regression after a course of radiation therapy that surgical excision becomes quite feasible. Finally one is obviously not justified in carrying out a neck dissection in cases having associated distant metastases. Here the employment of judicious amounts of radiation therapy for palliative purposes may be tried.

The term *radical neck dissection* refers to an operation that attempts to remove as completely as possible all the lymphatic tissues (lymph nodes and vessels) that are likely to be involved by metastatic cancer, together with all of the fatty and areolar tissue from the lateral and anterior aspects of the neck. The field of operation extends from the midline of the neck to the anterior border of the trapezius muscle and from the clavicle below to the mandible above. In depth it extends from the undersurface of the platysma muscle to the third layer of the deep cervical fascia. Included in the routine dissection are the sternomastoid muscle, the internal jugular vein, the carotid sheath, omohyoid muscle, the submaxillary gland, and the deep lymphatics of the neck. Any operation short of this is not complete and is generally doomed to failure. In order to afford the patient the best possible chance of cure, the first operation must be thorough and complete. The only valid reason for doing a partial neck dissection and limiting the operation to the submaxillary triangle or to the supra-omohyoid region is the fact that the patient is a poor surgical risk and not

likely to survive a radical procedure. In such cases it may be more prudent to resort to radiation therapy than to surgery.

Cervical Lymphatics

In order to appreciate the principles of carrying out a radical neck dissection one must have a thorough understanding of the anatomy of the cervical lymph channels and nodes which consist of a superficial and deep chain. The *superficial lymphatics* drain the skin and the appendages and empty into the deep cervical lymphatic chain. These lymphatics lie for the most part on the deeper aspect of the platysma muscle. They are rarely involved by metastatic cancer except from the deeper lymphatics in the very late stages of the disease, in which case large areas of skin must be sacrificed with the neck dissection. The *deep cervical lymphatics* are important because they receive lymph drainage from the mucous membrane of the mouth, pharynx, larynx, major salivary glands, and thyroid, and from the skin of the head and neck. The most important nodes in this group are those which run along the internal jugular vein from the level of the posterior belly of the digastric muscle down to its entry into the subclavian vein. These nodes vary from a few mm. to 1½ cm in diameter. The most important node in this group is the subdigastric or jugulodigastric node which is the highest node of the internal jugular chain and lies on the internal jugular vein just below the posterior belly of the digastric. It is more often involved by metastatic cancer than any other cervical lymph node. Because of the close association of the lymphatics throughout the length of the internal jugular vein, it is necessary to sacrifice this vein in order to extirpate the nodes.

The *submental lymph nodes* consist of at least a pair of nodes that lie on the surface of the mylohyoid muscles between the anterior bellies of the digastric muscles. They drain the skin of the lower lip, the chin, and the mucous membrane of the midportion of the lower lip, the anterior midportion of the floor of the mouth, and the tip of the tongue.

The *submaxillary lymph nodes* are divided into three sets: the preglandular, the intra-glandular, and the prevascular and retrovas-

DISEASES OF THE FACE, MOUTH, AND NECK

cular. They drain the mucosa of the lower lip, the cheeks, the alveolar ridge, the floor of the mouth, and the anterior two thirds of the tongue and empty into the deep cervical chain. The intraglandular lymph node which

and runs downward and backward through the posterior triangle along the course of the spinal accessory nerve. It receives the drainage mainly from the nasopharynx, but its upper end is intimately associated with the

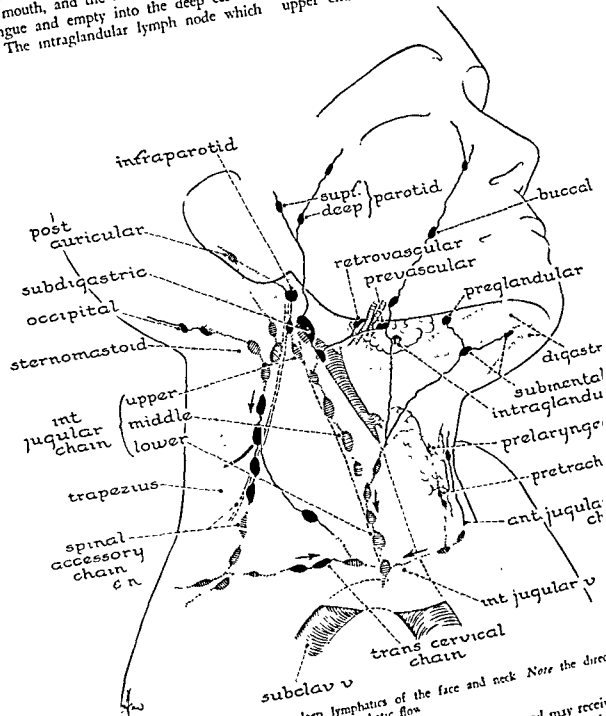


Fig 193--The anatomy of the deep lymphatics of the face and neck. Note the direction of lymphatic flow.

lies within the substance of the submaxillary gland is one of the main reasons for routine removal of the latter during neck dissection. The spinal accessory chain begins beneath the upper end of the sternomastoid muscle

upper jugular chain and may receive lymphatic drainage from it. Sacrifice of the spinal accessory nerve in a radical neck dissection is necessary in order to remove this lymphatic chain completely. The transverse cervical chain

at the root of the neck connects the spinal accessory chain with the lower end of the internal jugular chain.

The *parotid lymph nodes* are arranged in two groups. The first group includes the preauricular nodes which lie superficial to the parotid sheath and drain the temporal and frontal regions of the scalp, the outer portions of the lids, and the outer ear. The second group is deep in the substance of the gland and drains the external auditory meatus, the middle ear, the nasopharynx, the soft palate, and the nose. Both groups transmit the lymph to the lymph nodes in the neck. In addition there is usually a small node lying in the lower pole of the parotid gland which may be involved first in cancer of the parotid gland and at times in cancer of the upper lip.

The *pretracheal and prelaryngeal nodes* are involved early in cancer of the thyroid gland and of the larynx. These nodes are not routinely removed in a radical neck dissection, except when one is dealing with a cancer originating in either the thyroid gland or larynx.

TRACHEOSTOMY

The term *tracheostomy* means an operative opening into the trachea maintained for an indefinite period of time, and this is in contradistinction to the term *tracheotomy* which refers to the surgical procedure of opening the trachea for temporary exploration.

Tracheostomy is a valuable and at times a lifesaving surgical procedure which has, in many instances, been neglected in the treatment of acutely ill patients. Its primary use is to maintain an adequate airway which may be difficult. The indications for tracheostomy are as follows:

1. In acute cerebral injuries and in certain cases of brain tumors and of infections of the brain, the patient may be comatose or stuporous and unable to clear his tracheobronchial tree by coughing. In such cases the aspiration of food, drink, and vomitus may present a very definite hazard, which can be more easily combated with a tracheostomy tube in place.

2. In a severe and massive injury or infection about the face and neck, there may be considerable edema of the tissues of the neck or the pharynx, causing marked respiratory embarrassment. Such patients may be further handicapped by an ineffective

cough, with real danger of aspiration of pharyngeal and other secretions.

3. In patients with severe tracheobronchitis or other similar inflammatory conditions, there may be an inadequate airway due to edema and secretions produced by the inflammatory state.

4. In all penetrating, lacerating, or severe crushing injuries to the trachea.

5. In certain surgical procedures about the head and neck such as a radical neck dissection which is combined with some intraoral procedure, such as a hemiglossectomy or a resection of a portion of the mandible. Patients undergoing a bilateral radical neck dissection in one stage in which both internal jugular veins are sacrificed should also have a prophylactic tracheostomy because of the anticipated postoperative edema.

6. Cases of benign or malignant tumors involving the larynx, pharynx, upper trachea, thyroid, tongue, and mandible which are of such a size or extension that there is compression of the upper airway.

7. In postoperative severely ill patients, in whom the cough is ineffective to clear adequately the tracheobronchial tree. In such patients repeated bronchoscopy constitutes a real risk, whereas a prophylactic tracheostomy could be easily done and prove lifesaving. Similarly in patients who are known to have advanced pulmonary disease and who undergo major surgery, a tracheostomy will usually prevent pulmonary complications.

8. Following thyroid surgery one may have to carry out an emergency tracheostomy because of hemorrhage or injury to one or both recurrent laryngeal nerves.

9. In certain individuals who have received intense irradiation for malignant tumors in the neck and who develop severe tissue reaction and edema of the upper respiratory tract.

10. In the *crushed chest or stove in chest syndrome*, which refers to a crushing injury characterized by multiple rib fractures and causes the movements of a portion of the thoracic wall to be paradoxical to the normal excursions of the thoracic cage. A vicious cycle results in this syndrome. First of all there is incomplete aeration of the lung because of the paradoxical movement of the chest wall. Pain is severe and, combined with the paradoxical movement, renders coughing ineffectual. Secretions accumulate in the terminal bronchioles and alveoli, thus further impairing adequate ventilation of the lung.

Tracheostomy has been shown to be of benefit in this condition in several ways. First of all, it reduces the paradoxical movement of the injured chest segment and in this way decreases pain associated with the movements of the fractured ribs. Second, it provides an easy and ready method of aspirating the tracheobronchial secretions. Third, there is a decrease in the dead space and also a decrease in resistance to the inspired and expired air.

The use of other supportive measures together with the management of any complication, must not be neglected in this condition.

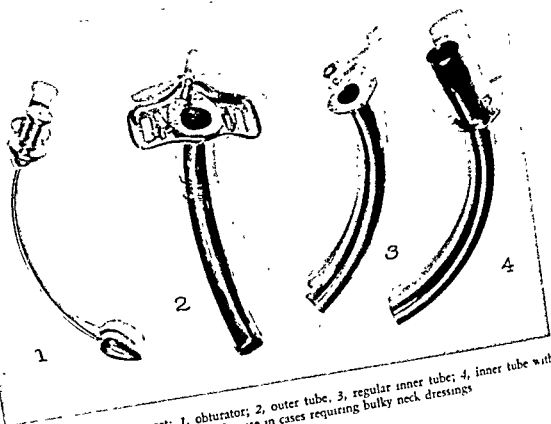


Fig. 196—Tracheostomy set: 1, obturator; 2, outer tube; 3, regular inner tube; 4, inner tube with an outward extension for use in cases requiring bulky neck dressings

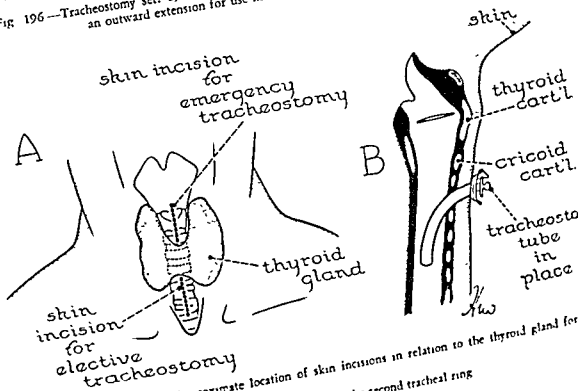


Fig. 197—A, Approximate location of skin incisions in relation to the thyroid gland for emergency or elective tracheostomy
B, Elective tracheostomy tube in place through the second tracheal ring

Technique of Performing a Tracheostomy.—Tracheostomy may be performed either as an emergency or as a prophylactic procedure. The *emergency tracheostomy* is generally performed as a heroic measure to establish an adequate airway in an individual who is choking to death. A *prophylactic tracheostomy* is one which is carried out in a patient in whom some form of respiratory complication is anticipated and in whom the insertion of a tracheostomy tube will prevent or lessen the severity of such a complication.

The procedure is relatively simple and one which can be easily performed under local anesthesia. A vertical incision is preferred since one can extend the incision up or down as required in order to get an adequate exposure of the trachea. The elective tracheostomy is always performed through the tracheal rings, preferably the first and second. The emergency tracheostomy is more easily performed through the crico-thyroid membrane as this area is relatively close to the surface of the skin and usually void of blood vessels.

The vast majority of tracheostomies are of a temporary nature, maintained during the acute phase of illness or diminished air passage. The tube may remain in situ for an indefinite period of time, but 2 to 4 or 5 days is all that is usually necessary. Under no circumstances should the tube be removed until the surgeon is positive that the upper airway is adequate without the assistance of the tube. Ordinarily the wound closes spontaneously within 2-3 days after the tube is removed.

Care of a Tracheostomy.—Bedside suction apparatus is required, and a nurse should be in constant attendance until the patient has learned to suction the trachea himself. The inner tube is removed and thoroughly cleaned inside and out twice daily. The outer tube is changed once every 2-3 days. A moist atmosphere, effected by the use of steam or a cold humidifier, is often helpful.

THE SALIVARY GLANDS

The major salivary glands consist of the paired parotid, submaxillary, and sublingual glands. These organs are liable to injuries, infections, and neoplastic processes.

Injuries

Accidental wounds or surgical operation may cause lacerations of the parotid gland or wounds of the parotid duct. A radical dissection of the cervical lymph nodes usually makes it necessary to remove the lower portion or tail of the parotid gland. In such cases and in those in which there is accidental laceration of the gland, there may be a discharge of saliva for a day or two, but with pressure dressings the wound usually heals in a few days without persistence of a salivary fistula. On the other hand, a complete transection of Stensen's duct may continue as a salivary fistula. In such cases the discharge of saliva is more copious in association with eating, and little or no parotid secretion enters the mouth through its normal channel. Pressure dressings, if instituted early, may aid in healing of the fistula. A fistula that persists after 1-2 months should be treated by operation, either to join the ends of the duct or to transplant the proximal end of the duct into the mucous membrane of the cheek. In fistulas that prove to be difficult to treat, the use of x-ray therapy will aid in reducing the secretion of saliva.

Inflammation

Aside from mumps, infections of the salivary glands are not very frequent. However, when they do occur, they may prove to be extremely painful and disabling. Two types of inflammation are recognized:

- 1 The acute suppurative infection occurring almost always in the parotid glands.

- 2 The chronic inflammation usually associated with symptoms of recurring obstruction of the ducts. In this case the submaxillary is more often affected than the parotid gland.

Acute Suppurative Parotitis.—This occurs most often in debilitated, dehydrated patients, sometimes as a postoperative complication following a serious operation, or in prolonged febrile disease such as typhoid fever, bronchopneumonia, severe tonsillitis, or pharyngitis. Old age, weight loss, chronic infection, and poor nutrition all seem to be important factors in its causation.

The organism most frequently responsible is the staphylococcus and less frequently the streptococcus which is found especially in the fulminating cases. The causative organism

travels from the mouth along the parotid duct into the gland. At times, however, it may be blood-borne

The local symptoms are pain and swelling of one or both parotid glands, associated with fever, chills, headache, and general malaise. The mouth may be foul and dry, and pus may be expressed from the red, swollen orifice of Stensen's duct. There is usually leukocytosis, and abscess formation may occur. Since suppurative parotitis occurs in seriously ill patients, this complication may be a terminal one.

Treatment.—The prophylaxis of this condition is much more important than the treatment. The mouth should be kept clean, and dehydration must be avoided. Seriously ill and debilitated patients should be brought into an optimum condition by means of diet, blood transfusions, and correction of electrolyte imbalance before any operation is undertaken. With the advent of intravenous electrolytic solutions and antibiotics, acute parotitis has become a much less frequent postoperative complication. Chewing of gum promotes salivation and good drainage of the glands. The use of Lugol's or other mild antiseptic solution as a mouthwash is also useful.

The treatment of mild cases of acute parotitis by means of local application of heat or cold, together with intensive antibiotic therapy and oral hygiene, is usually successful. Many severe cases will also respond to this treatment. In the more resistant cases the use of x-ray therapy combined with antibiotic therapy generally succeeds. When there is frank suppuration and abscess formation, incision and drainage become imperative.

Chronic Inflammation.—As previously mentioned, this condition occurs more frequently in the submaxillary than in the parotid gland. The inflammation in this condition is generally a result of a partial or complete obstruction of the duct due to a plug of thick mucus, to swelling of the duct mucosa, or to a salivary calculus. Patients suffering from this disease may have recurrent or persistent swelling of the submaxillary gland or occasionally of the parotid gland. The pain or swelling may be especially pronounced when food is ingested. At times the exacerbations of pain and swelling of the gland are associated with

some minor inflammation such as pharyngitis. A sialogram with Lipiodol may show a narrowing stricture in Stensen's duct, or there may be a partial or complete blockage of the duct by one or more stones, or there may be dilatation of the smaller ducts and acini in the gland with puddling of the Lipiodol, the result of chronic inflammation.

Treatment.—Oral hygiene must be maintained. The actual treatment will vary with the type of obstruction. Simple probing of the duct may succeed in re-establishing the flow of saliva. If the meatus of the duct is constricted, it is incised for a distance of $\frac{1}{2}$ -1 cm. with suture of its epithelial lining to the mucosa of the cheek. If the obstruction is due to a stone in the duct, the calculus should be removed. In those cases where there are multiple stones within the gland substance itself or where there is evidence of chronic inflammation by a sialogram, a partial or complete removal of the salivary gland is indicated.

Mikulicz's Disease.—Until recently this disease has been a rather vague and ill-understood entity. It has been referred to by a variety of terms: chronic inflammation, lymphoepithelioma of Fein, lymphocytic tumor, adenolymphoma; more recently Godwin has suggested the term *benign lymphoepithelial lesion* as describing best its clinical course and histologic characteristics. In the original disease as described by Mikulicz in 1888, both lacrimal glands and all the salivary glands were diffusely involved in a tumefaction. From his description of the clinical and histologic features of the disease, the appearance is very similar to those subsequently described under the headings mentioned above. It is unnecessary for the lacrimal glands to be involved for a diagnosis of Mikulicz's disease. The lesion may present unilaterally in the parotid gland, or bilaterally or successively in both glands, and may or may not involve the other major salivary glands. As a general rule their involvement is extremely rare.

The true nature of this lesion is unknown. Although most likely an inflammatory process, there are some instances, however, which present features suggestive of a neoplasm. Histologically, the lesions consist of a mass of lymphoid tissue containing scattered foci of

epithelial cells that are traceable to ductal origin. The disease may be in scattered foci with involvement of the entire gland, or it may be localized to a single nonencapsulated focus, or it may be circumscribed and encapsulated.

The prognosis of this lesion is excellent, and none of the cases reported have run a malignant course. The patients respond well to surgical or irradiation therapy. If the diagnosis can be established without operation, x-ray therapy is perhaps more satisfactory. With a localized lesion where the diagnosis is uncertain, resection of the tumor may have to be carried out.

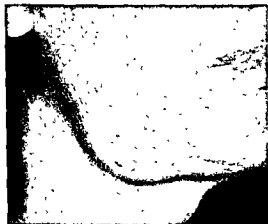


Fig 198—Calculus in Wharton's duct, causing swelling of the submaxillary salivary gland. This lesion must be distinguished from a neoplasm which may present an identical clinical appearance.

Salivary Calculi—Salivary stones occur more frequently in the submaxillary salivary gland or duct than in the parotid gland or duct. As previously mentioned, calculi may cause recurrent inflammation and swelling in a salivary gland, aggravated by eating. Stones vary in size from a few mm up to 2 cm. or more in diameter and are frequently multiple, especially when small. Their exact mechanism of formation is not clearly understood, but they probably result from a precipitation of salts upon desquamated cells in stagnant salivary secretion. Ninety per cent of the stone is composed of calcium carbonate and calcium phosphate, while the remainder is made up of cellular debris, cholesterol, and other organic material.

Clinically the patient complains of an intermittent, swollen, and perhaps painful mass in the vicinity of the submaxillary gland or less frequently of the parotid gland. The pain is usually aggravated when food is taken. On examination the calculus may be felt somewhere along the course of the duct by combined intraoral and extraoral palpation. Inspection will disclose some redness or swelling about the orifice of the duct from which pus may be expressed. X-ray of the gland or duct will demonstrate the calculus.

Treatment—A stone in Wharton's duct may be removed by an incision in the floor of the mouth. A stone in Stensen's duct is removed by exposing the duct through an incision in

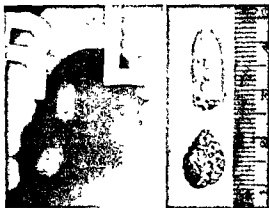


Fig 199—X-ray and photograph of salivary calculi.

the buccal mucosa, feeling for the stone, and removing it by an incision into the lumen of the duct directly over the stone. If the calculus is in the submaxillary gland, it is better to excise the gland through an incision in the submaxillary triangle. Similarly, troublesome stone or stones in the parotid gland are best treated by a partial parotidectomy.

Tumors

The parotid salivary glands account for approximately 87% of all such tumors, the submaxillary glands for about 12%, and the sublingual glands for less than 1%. In addition to arising from the major salivary glands, salivary gland tumors may arise from the minor salivary glands situated within the oral mucosa or in the upper respiratory tree, so that these tumors may appear in the tongue, hard or soft

DISEASES OF THE FACE, MOUTH, AND NECK

TABLE 13
CLASSIFICATION AND APPROXIMATE RELATIVE INCIDENCE OF SALIVARY GLAND TUMORS

Benign Tumors 70%		Malignant Tumors 30%	
1 Benign mixed tumors	60%	1 Malignant mixed tumors	6%
2 Adenomas	1%	2 Carcinoma	22%
a Ordinary adenoma		a Adenocarcinoma	
b Oncocytic adenoma		b Squamous cell carcinoma	
c Sebaceous cell adenoma		c Mucoepidermoid cancer	
3 Benign lymphoepithelial lesion	1%	d Cylindroma	
i Warthin's tumor	6%	e Undifferentiated cancer	2%
5 Miscellaneous	2%	3 Miscellaneous	
a Lipomas		a Fibrosarcoma	
b Simple cysts		b Malignant melanoma	
c Neurofibroma and schwannoma		c Hemangioperithelioma	
		d Lymphosarcoma	
		e Hodgekin's disease	

palate, buccal mucosa, floor of mouth, trachea, or bronchus. The present discussion will be confined to tumors of the major salivary glands.

By far the most frequent type of tumor is the so-called benign mixed tumor. However, it is estimated that approximately 30% of parotid tumors are malignant, and one must keep this possibility in mind when dealing with any parotid tumor.

Our knowledge of the classification and behavior of salivary gland tumors is still incomplete. The pathologist continues to describe new histologic entities which as yet have not been completely correlated with specific clinical findings. In Table 13 is compiled a classification of benign and mixed salivary gland tumors with their relative incidence.

Clinical Features.—Tumors arising in the major salivary glands are found in patients of all ages. Benign salivary gland tumors are commoner in the 5th decade, while malignant tumors are more frequent in the 6th and 7th decades. The sex ratio between patients is almost equal, although malignant tumors are more frequently found in males.

In the case of the parotid gland approximately 50% of the tumors are situated in the tail or inferior portion of the gland, that is, that part below and slightly anterior to the lobule of the ear. The next most frequent site is in the anterior or mid-portion of the gland, accounting for approximately 25%. Thus the majority of parotid tumors are found in the so-called superficial portion of the gland, which is that part of the gland super-

ficial to the facial nerve and its main branches. At times the growth may arise in the deep or retromandibular portion of the gland. Since extension laterally is blocked by the ascending ramus of the mandible, the tumor tends to grow medially and may present as a bulge in the pharynx or tonsillar region.

In over 80% of the patients the only initial symptom of a benign salivary gland tumor is the presence of a slow-growing lump in the parotid or submandibular region. This mass is typically freely movable and painless and may have been present for months or even years before medical advice is sought. There is no fixation to the skin or to the surrounding gland substance. In a smaller percentage of patients, there is some degree of local discomfort associated with the tumor. In the case of malignant salivary tumors pain may be an early and quite prominent symptom. The rate of growth is slow, but it may suddenly become more rapid, at which time one must suspect a malignant lesion.

Benign mixed tumors are so called because of the varied microscopic picture which they present. At one time it was believed they were composed of a number of elements, fibrous and mucous tissue, cartilage, epithelial and endothelial cells. It is fairly well accepted today that these tumors arise from the epithelium of the salivary gland. There is definite evidence to show that a benign mixed tumor is capable of becoming malignant. At times this change takes place suddenly without previous operative interference, while at other times the change occurs only after multiple operations over a period of many years.

In the diagnosis of tumors of the salivary gland one must differentiate chronic inflammation, hypertrophy of the preauricular lymph nodes, sebaceous cyst, lipoma, branchiogenic cyst, metastatic cervical cancer, and a calculus in the submaxillary or parotid salivary ducts or glands.

Malignant tumors in their early stages may differ in no way from simple mixed tumors. Their rate of growth at first, like that of a mixed tumor, may be quite slow. However, they usually undergo a period of sudden or

effective method of treatment is surgical. However, radiation therapy does play a very useful role, particularly in inoperable or recurrent lesions. Even the surgical management of salivary gland tumors (especially parotid) is not as good as it might be as judged by the average recurrence rate of 30-35% of benign mixed tumors reported in medical literature.

In this group of tumors there are a number of reasons for failure of initial therapy:

1. Lack of adequate knowledge of the surgical anatomy of this region. The constant fear of injury to the facial nerve prevents the surgeon from carrying out an adequate and complete operation.

2. The tendency of many surgeons to simply enucleate the tumors. This principle of treating salivary gland tumors is wrong. It has been

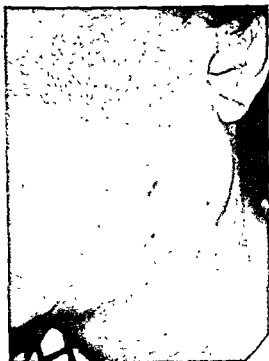


Fig 200—Benign mixed tumor of the parotid salivary gland. Note that the most prominent portion of the tumor lies below the lobule of the ear.



Fig 201—Benign mixed tumor arising from the minor salivary glands in the buccal mucosa.

rapid increase in size. The tumors are generally quite firm and in the more advanced stages may become fixed. Facial nerve paralysis, when associated with a persistent swelling in the parotid region, is almost always diagnostic of a malignant salivary gland tumor. Metastases to the regional lymph nodes occur in approximately 30-50% of the patients, depending upon the histologic type of cancer. Distant metastases to lungs, bones, liver, and brain are not infrequent.

Treatment.—In general, salivary gland tumors are not radiosensitive, and the most

shown on repeated occasions that the capsule itself is often incomplete and that microscopic tumor cells extend into and even beyond the capsule into the adjacent parotid tissue.

3. Attempts are made to remove a tumor without adequate preparation or assistance. This can happen when a doctor attempts to remove in his office or in the clinic a pea-sized nodule in the parotid region with the mistaken impression that it is a sebaceous cyst or lymph node. He generally finds himself dealing with a larger or more deeply seated parotid tumor with ultimate spillage of tumor cells through-

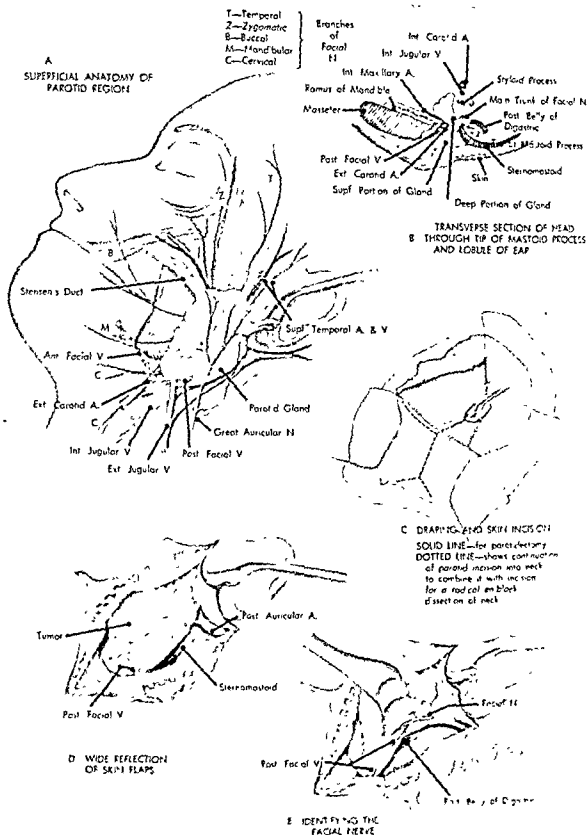


FIG. 202—A and B, The surgical anatomy of the parotid region
C, Outline of skin incision for parotidectomy
D to F, Steps in the procedure of exposing and preserving the facial nerve during the course
of a partial or a total parotidectomy.

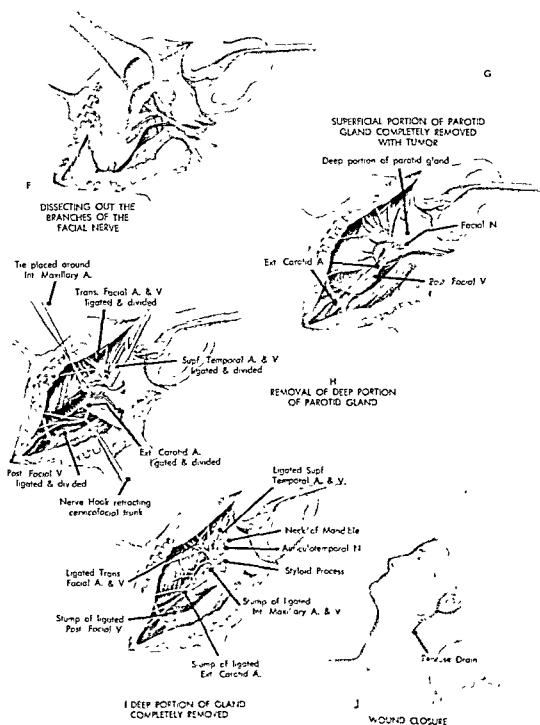


Fig. 202 (cont d) - (See opposite page for legend)

out the operative field or injury to the facial nerve or both

In our opinion, the best exposure for the excision of a parotid tumor is a Y incision, two arms of which run up in front of and in back of the lobule of the ear, third arm extending downward and forward into the upper neck. The first principle of the operative removal of any parotid tumor is to secure complete exposure of the parotid gland and tumor by wide reflexion of the skin flaps. It is imperative that one avoid injury to the 7th cranial nerve. The best way to prevent such an accident is to first expose the main trunk of the nerve as it emerges from the stylomastoid foramen and then to proceed to dissect out its various branches within the substance of the parotid gland. Since most of the tumors are situated in the superficial portion of the parotid gland, one should carry out, in the majority of cases, a superficial parotidectomy. In smaller tumors it may be justifiable to remove the tumor alone, together with a rim of normal-appearing parotid tissue around it. There have been many other methods advocated in the literature for identifying the facial nerve. In most of these, one or more of the peripheral branches of the nerve on the face or in the upper neck are isolated and then traced back through the substance of the gland to the main trunk of the nerve. This method is much more difficult and is fraught with real danger of injuring one or more of these small peripheral branches.

In cases of cancer and in cases where one is dealing with a benign mixed tumor arising in the deep portion of the gland, it is necessary to carry out a total parotidectomy. In a benign tumor, every effort should be made to spare the facial nerve and its branches. However, when dealing with cancer, especially when the growth lies adjacent to or infiltrates the nerve or its branches, then one should not hesitate to sacrifice those branches or even the entire nerve in order to remain wide of cancer.

In cancer of the parotid gland where there is clinical evidence of metastatic cervical lymph nodes, a radical neck dissection should be carried out as part of the primary surgical treatment of the cancer. In those cases in which there is no clinical evidence of cervical

lymph node metastases, one must be guided by the type of tumor in question and its relative malignancy.

In benign submaxillary salivary gland tumors, one generally carries out a complete dissection of the contents of the submaxillary triangle through a curved incision situated approximately two fingerbreadths below and parallel to the lower margin of the mandible. The lymphatics as well as the submaxillary salivary gland are completely removed *en bloc*. In a malignant submaxillary gland tumor, one should also carry out a radical neck dissection, whether or not the other deep cervical lymph nodes are involved. Since the neck must be entered surgically, it is easier to complete the entire operation at this time than to wait for clinical evidence of metastases to appear later, at a time when previous scarring will have made the neck dissection much more difficult.

Complications of Treatment.—

Recurrence of Tumor.—The postoperative recurrence rate of benign mixed tumors of the parotid gland reported in the literature averages around 30%. It is our opinion that the majority of recurrences is due to faulty technique, such as the enucleation of a tumor, the spillage of tumor cells in the wound inadvertent or otherwise, and failure to resect in a sufficiently wide or radical manner. Reports from the Lahey Clinic by Marshall and Force and from the Memorial Hospital by Farrell show a recurrence rate for the primary benign mixed tumor of 6.4% and 4.8%, respectively, when the tumor has been treated in the manner described above.

The recurrence of malignant tumors following surgery is a serious complication, since it is generally associated with further spread of the disease and the ultimate death of the patient.

Facial Nerve Palsy.—Disregarding those cases in which one deliberately sacrifices one or more branches of the facial nerve because of involvement by tumor, one still sees an incidence of approximately 12-15% of facial nerve paralysis which varies from a minor degree of weakness about the mouth to total paralysis of the side of the face. The cause of the paresis is generally due to the trauma caused by excess handling or stimulation of the nerve. In the majority of cases one can

assure the patient of eventual and complete recovery.

Frey Syndrome.—This is also referred to as the auriculotemporal syndrome and is characterized by sweating and flushing of the cheek over the cutaneous distribution of the auriculotemporal nerve. The exact etiology of this phenomenon is not too clear.

Salivary Fistula.—This is a rare complication following subtotal parotidectomy. One may occasionally encounter a small accumulation of saliva under the skin where pressure dressings were not adequately maintained to permit adherence of the skin flaps to the underlying tissue. This soon subsides.

Loss of Sensation About the Ear.—This condition is due to damage or section of the great auricular nerve and is rarely permanent.

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<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procureable From</i>
Combined Surgery in the Treatment of Carcinoma of the Tongue (1954) (By Oliver M. Beahrs, M.D., and Kenneth D. Devine, M.D., Rochester, Minn.)		Color Silent	American Cyanamid Co. Surgical Products Division Danbury, Conn.
Resection of Complete Branchial Cleft Fistula (1954) (By B. Marden Black, M.D., Rochester, Minn.)	10 min		American Cyanamid Co. Surgical Products Division Danbury, Conn.
Radical Excision of Thyroglossal Sinus (Sistrunk Method) (1954) (By Edward S. Judd, Jr., M.D., Rochester, Minn.)			American Cyanamid Co. Surgical Products Division Danbury, Conn.
Oral Cancer The Problem of Early Diagnosis (1953) (By American Cancer Society, New York, and National Cancer Institute, Bethesda, Md.)	31 min	Color Sound	American Cancer Society 521 W. 57th St. New York 19, N. Y.

Chapter 13

Surgery of the Endocrine Glands

Stewart Baxter, MD, Chauncey J Partee, MD, James F Hopkirk, MD,
and John T. MacLean, MD

The title of this chapter is somewhat misleading since surgery of the endocrine glands is not a completely separate surgical specialty. The endocrine glands are so widely scattered anatomically that they normally fall into the province of the regional specialist. Thus pituitary conditions are dealt with by the neurosurgeon, the adrenal lesions by the urologist or abdominal surgeon, and so on.

However, for the convenience of the reader, the surgery of the endocrine glands has been incorporated into one chapter. The physiology of the organs discussed has been presented in some detail, since this knowledge is necessary for an understanding of the surgical problems involved. The careful preoperative, operative, and postoperative handling of these patients depends on this knowledge and is essential for the successful outcome of operative procedures.

THE PITUITARY GLAND

Anatomy

The human pituitary gland consists of a *pars anterior*, separated from a *pars posterior*, or neural lobe, by cystic spaces lined by epithelial cells which make up the *pars intermedia*. The *pars posterior* is connected by the infundibulum with the median eminence of the tuber cinereum in the floor of the third ventricle. The *infundibulum* is a stalk which is invested by a vascular epithelial cuff, the *pars tuberalis*.

The gland lies in a bony fossa, the sella turcica, within the body of the sphenoid bone.

The floor is bone, immediately below which lies the sphenoid sinus. The roof is formed by a dural membrane which is continuous with the dura covering the floor of the skull. It is perforated by a foramen through which passes the infundibulum. The anterior wall of the sella turcica terminates in the tuberculum sellae, on either side are seen the anterior clinoid processes. Posteriorly it is bounded by a plate of bone, the dorsum sellae, from which project the posterior clinoid processes.

Cytology.—The pituitary gland arises from both epithelial and neural tissue. The cells in the anterior lobe are arranged in closely packed columns or irregular masses, separated by vascular sinuses lying in a connective tissue stroma. There are three main types readily divided by their staining reaction—*chromophobe*, the cytoplasm of which does not stain, and the *chromophil* which is subdivided into acidophil and basophil according to the staining reaction of the granules. The posterior lobe and infundibulum are composed of a network of nonmyelinated nerve fibers and supporting neuroglial cells called *pituitocytes*; these latter may contain melanin-like pigment granules. Basophil cells similar to those found in the anterior lobe are commonly found in the posterior lobe.

Blood Supply.—Blood is supplied to the anterior lobe by a number of hypophyseal arteries which arise from the internal carotid and posterior communicating arteries. Most of these enter the *pars tuberalis* and thence course

to the anterior lobe; others enter the body of the gland directly. The inferior hypophyseal arteries arise from the internal carotid arteries in the cavernous sinuses and enter the posterior lobe. The venous blood return is not well established. The afferent or portal vessels run down the pars tuberalis, and an efferent or systemic vessel drains the gland from its lateral walls. The hypophyseal portal veins arise from a plexus of wide capillaries which lie on the surface of the infundibulum between it and the pars tuberalis. The systemic veins arise from the sinusoids of the anterior lobe.

Nerve Supply.—A considerable number of nonmyelinated nerve fibers arise from the cavernous sympathetic plexus and run in the connective tissue capsule of the gland. Many of these pass into the gland in the anterior and lateral walls. A few of these ramify among the epithelial cells. Two other large groups of fibers enter the gland with the hypophyseal arteries.

The posterior lobe is connected with the hypothalamus by thousands of nonmyelinated nerve fibers—many are connected to both the paraventricular and the supraoptic nuclei.

Physiology

The number of anterior pituitary hormones is uncertain. Ultimately the exact number will be determined when they are all obtained in pure form. The hormones are proteins or proteinlike substances and the establishment of biologic purity is difficult and sometimes apparently an impossible task. The measures used to obtain biologic purity include electrophoresis, ultracentrifugation, and solubility analysis.

Six anterior pituitary hormones have been obtained as relatively homogeneous proteins. These have been localized as follows: luteinizing hormone, ICSH, growth hormone, and prolactin in the acidophilic granules and follicle-stimulating hormone, corticotropin, and thyrotropin in the basophilic granules.

Adrenocorticotrophic Hormone.—Potent preparations of corticotropin isolated from bovine, sheep, and whale pituitaries manifest similar physiologic effects in animals and in man.

No significant increase in adrenal weight is observed in the first few hours after initiation

of stress or injection of corticotropin. Continuous exposure to stress or continuous administration of corticotropin induces adrenal hypertrophy. The increase in weight is proportional to the dose. The adrenal cortex is depleted of ascorbic acid, cholesterol, and the sudanophilic material. The increase in urinary steroids and eosinopenia are only rough guides of corticotropin activity. A mixture of steroids is elaborated from the adrenal cortex on being stimulated by corticotropin. In the dog, at least three biologically active steroids have been isolated from adrenal vein blood. 17-hydroxycorticosterone (hydrocortisone compound), corticosterone (compound B), and a potent sodium-retaining factor (aldosterone).

Corticotropin is rapidly inactivated in the organism. It is not excreted in the urine in a biologically active form. It is absorbed readily from intramuscular and subcutaneous sites and is effective by intravenous injection. It is inactive by the oral route.

This hormone is used therapeutically either intramuscularly every 4 hours, one injection of the long-acting substance every 24 hours, or by intravenous infusion.

Growth Hormone.—A purified hormone has been obtained from bovine and porcine pituitaries. This hormone normally accelerates growth in the young organism. The increase in body weight is characterized by the deposition of a tissue high in protein and low in fat. Optimal growth can only occur when the tissues of the organism have adequate quantities of thyroid and adrenal cortical hormones. Animals treated with the growth hormone utilize elements of the diet more efficiently. There is a marked stimulation of the epiphyseal cartilages.

Nitrogen is retained in association with a gain in body weight, but it is felt that the full potentialities of protein synthesis are evident only when insulin is present in appropriate quantities. Purified hormone is said to produce both a hypoglycemia and a ketonemia in animals as well as to reduce greatly the glomerular filtration rate and tubular secretory activity of the kidneys.

Evidence has been presented that a substance is present in the blood of patients suffering from gigantism, which induces an increase of epiphyseal growth in hypophy-

sectomized rats. However, the present purified preparations of growth hormone only occasionally have a protein anabolic effect in man. This presents a frustrating problem and perhaps will only be answered by further purification of this substance.

Thyrotropin (TSH)—Highly potent preparations have been obtained from beef and sheep pituitaries. Administration to the normal animal results in an increase in the weight of the thyroid. Hyperplasia is induced while there is loss of colloid and an increase in acinar cell height. The rate of accumulation of I^{131} in the thyroid is increased. The conversion of iodides to thyroxine is increased as indicated by the rise in plasma protein-bound iodine, following administration of TSH. The identity of TSH with the exophthalmic factor has not been established. This hormone is effective by the parenteral but not by the oral route.

Gonadotropin (FSH and LH)—Isolation of pure FSH from sheep pituitary has been claimed recently. LH as a chemically homogeneous protein has been isolated as a preparation from hogs and sheep. The ovary responds to the gonadotropins with growth of the primordial follicle, ovulation and corpus luteum formation. Steroid hormones which affect growth and development of the endometrium and other sex organs are liberated from the ovary under stimulation of the gonadotropins. The corpus luteum is maintained by prolactin or luteotropin (ICSH). In the male rat, FSH initiates and stimulates spermatogenesis, while LH stimulates Leydig cells which secrete the androgenic steroid. FSH and LH are inactivated rapidly by the proteolytic enzymes of the gastrointestinal tract and have no effect if administered orally.

Prolactin—This protein has been extracted from hog, beef, and sheep pituitaries. Riddle named the substance prolactin which at that time stimulated the crop sac of pigeons and induced lactation in mammals. Although it has been established that extracts of the anterior pituitary maintain lactation in various animals, the exact nature of the galactopoietic agent is uncertain. It is felt that prolactin is confined to lactogenesis. This hormone has been claimed to be luteotropic, final evidence has not been presented in man that this is so. This hormone is ineffective when administered orally.

Anterior Pituitary Endocrinopathies

A variety of endocrinopathies are produced by either hyperfunction or hypofunction of the anterior pituitary. The commonest cause of such endocrinopathies is intrasellar or extrasellar tumors.

Gigantism and Acromegaly.—These diseases can be ascribed to an excessive secretion of growth hormone. The time of onset determines whether gigantism or acromegaly will develop. When the onset follows epiphyseal closure, acromegaly results.

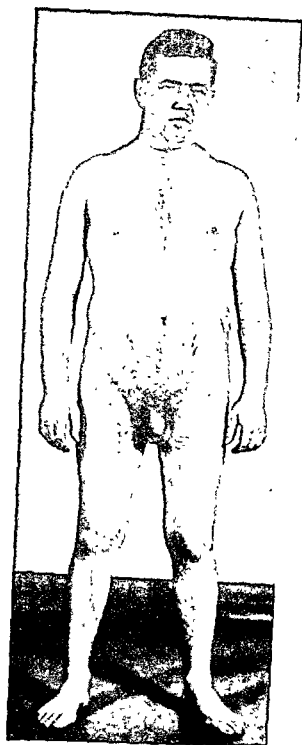
Briefly the outstanding features are as follows:

In *gigantism*, there is excessive height with fairly normal body proportions and increased length and thickening of the bones.

In *acromegaly*, the skin is thickened and thrown into folds. The tongue, lips, nose, and ears enlarge; and there are prognathism and enlargement of malar and supraorbital ridges, kyphosis and enlargement of the hands and feet, and visceromegaly and thickening of bones.

Common to both are adrenocortical hypertrophy, thyroid hypertrophy, and gonadal atrophy or hypertrophy. Serum inorganic phosphorus is increased, glucose tolerance is often impaired, and there is insulin resistance. The BMR is increased in some patients. Hypertension and diabetes mellitus are commoner in the acromegalic. Such disturbances are accounted for by an increase in growth hormone and a possible increase in TSH, ACTH, and gonadotropins.

Cushing's Disease.—The underlying pathology in this disease is that of a basophilic adenoma of the anterior pituitary gland. This tumor results in an excessive secretion of corticotropin (ACTH). Almost all features of the disease have been reproduced by the administration of ACTH or cortisone. Briefly the outstanding features are as follows: Acne, hirsutism, striae, easy bruisability, thinning of skin, moonface, buffalo hump, thinning of arms and legs, muscular weakness, depression of libido, and amenorrhea. Hypertension is present which may or may not be accompanied by renal disease. Polycythemia, lymphocytopenia, and eosinopenia are found. The glucose tolerance curve is impaired and there is insulin resistance. The plasma sodium and



A.



B.

Fig 203 -A, Patient with Cushing's disease. The pituitary tumor was excised, showing mixed carcinoma.

B Typical abdominal striae, same case.

chloride are normal or slightly elevated, while the potassium is low. The patient is in a negative nitrogen balance, due undoubtedly to an increase in the output of compound F or "S" hormone and a relative decrease in androgenic substances or "N" hormone. This also accounts for the osteoporosis so commonly found.

Adrenocortical hypertrophy and gonadal atrophy are present

Hypopituitarism.—The disturbances of this disease are due to deficiencies of all tropic hormones of the anterior pituitary. Such deficiencies may be caused by post-partum necrosis (Sheehan's syndrome) and by pressure from an adjoining tumor (chromophobic, eosinophilic, basophilic, craniopharyngioma, or extrasellar). Irradiation may have the same effect. Simmonds originally described the condition of pituitary cachexia. Such patients are rarely seen now, chiefly because the condition is diagnosed at an earlier stage, prior to the development of extreme cachexia.

Essentially the disturbances seen in pan-hypopituitarism are due to the inability of the target organs, the thyroid, adrenal, and gonads, to function due to the fact that they are not being stimulated. The clinical picture presented represents a combination of the signs and symptoms of underfunction of all three endocrine glands. Hypofunction of these glands need not be complete. The most common form of hypopituitarism seen is that of partial hypopituitarism. The cause is most commonly that of partial destruction due to an expanding chromophobe adenoma. When such a tumor is present, the gonadotropic hormone is the first one of the pituitary hormones to be deficient. This results in amenorrhea or impotence, a point well to remember when amenorrhea is the presenting symptom.

Craniopharyngiomas have been mentioned previously as a cause of hypopituitarism. This is referred to as Frohlich's disease. In this condition, as originally described by Frohlich, the hypophyseal duct cyst or craniopharyngioma produced hypothalamic as well as pituitary destruction, with obesity, retarded growth, hypogonadism, and diabetes insipidus being present. It is assumed that the obesity and diabetes insipidus were present because of hypothalamic damage as neither the TSH nor

ACTH is shown to be deficient. This condition represents a form of partial hypopituitarism.

Diagnostic Techniques.—

1. Fundi examination: Papilledema is not often present, but careful examination of the fundi is necessary.

2. Visual fields: These should be determined. Various degrees and combinations of field defect can occur, depending upon the pattern of infringement of the optic nerves or chiasm. A bitemporal hemianopsia is the most common finding.

3. X-ray sella turcica—stereolateral views are recommended. Erosion of the clinoid processes or the floor may be present.

4. Ventriculogram: This procedure may be helpful or necessary when it is suspected that the tumor responsible for the hypopituitarism is extrasellar.

Treatment.—Treatment of pituitary syndromes represented by acromegaly, gigantism, or Cushing's disease will not be discussed in detail. Once the diagnosis is established there are three types of treatment. (1) medical, (2) x-ray, or (3) surgical.

Medical.—The administration of stilbestrol or a natural estrogen has been shown by Albright to depress pituitary function. Stilbestrol 3-5 mg. per day is usually adequate. This is most useful in women.

Irradiation.—Eosinophilic adenomas are radiosensitive in approximately 50% of instances. The effect, however, is likely to be transient.

Basophilic adenomas are radiosensitive in only a small percentage of cases.

Chromophobe adenomas are not amenable to medical treatment, as the chromophobe cell is nonfunctioning. They are radiosensitive in approximately 65% of instances.

The effect of x-ray treatment to the pituitary is not manifest for 3-6 months after cessation of treatment. If the patient has symptoms or signs of hypopituitarism prior to treatment, further hypofunction may ensue. Adrenal crisis must be kept in mind. Supportive therapy with cortisone during this period is sometimes helpful. The course of x-ray usually takes 5-6 weeks—4,000-4,500 roentgen units are delivered over bitemporal portals.

SURGERY OF THE ENDOCRINE GLANDS

Craniopharyngioma is a cyst and is not radiosensitive; therefore surgery is indicated if the cyst is expanding.

Surgical—Removal of a pituitary tumor is indicated whenever the lesion has expanded sufficiently or persistently to produce progressive loss of vision or severe headaches. There is no difficulty with the former indication; the latter creates considerable controversy. To state that headaches are definitely secondary to an expanding lesion, in the absence of eye signs, is not always easy.

Prior to surgery it is essential to know what, if any, supportive therapy is necessary. The main concern is to determine the degree of function of the adrenal gland. It is well for the surgeon to remember that hypopituitarism always develops after surgery. Should likely that the patient will survive the stress of the operation. Some centers administer cortisone routinely prior to the removal of a pituitary adenoma. If time does not allow for adequate preoperative evaluation, 25 mg. of cortisone is administered orally q6h., beginning two days preoperatively. On the morning of operation 100 mg. of cortisone is given intramuscularly. This is repeated in eight hours' time. When the patient is able to swallow, cortisone may be given orally in 25 mg doses q6h., or 100 mg of Hydrocortone may be given intravenously at the time of operation, if there is a drop in blood pressure. Cortisone is slowly discontinued over the next week, eliminating 12.5 mg each day.

THE THYROID GLAND

Embryology

The major portion of the thyroid gland originates from the pharyngeal endoderm at

the level of the first pouch. It soon develops into a solid mass attached to its point of origin by a narrow stalk—the thyroglossal duct. About the fifth week this stalk disappears, leaving a small depression at the base of the tongue, known as the foramen cecum. The remainder of the stalk is often found as a fibrous cord, but in some individuals remnants of epithelium lead to the formation of thyroglossal cysts and sinuses. The mass now assumes its position with a lobe on each side of the trachea and in the seventh week fuses with the rudimentary fifth pouches, which are ultimately transformed into thyroid tissue. The primitive mass now undergoes transformation into acini and follicles containing colloid material.

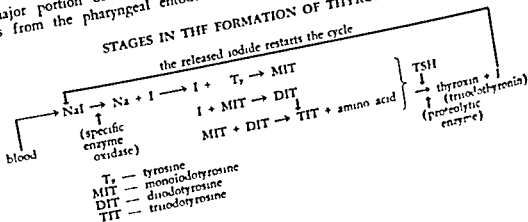
Anatomy

In the adult, the thyroid gland, consisting of a right and a left lobe, joined by the isthmus, lies on the anterior surface of the larynx and trachea at the level of the 5th, 6th, and 7th cervical vertebrae. A third, or pyramidal lobe, often extends from the isthmus or medial aspect of either lateral lobe to the level of the hyoid bone. The average weight of the gland is 30-50 Gm.

The arterial supply comes from:

- 1 The superior thyroid artery, a branch of the external carotid
- 2 The inferior thyroid artery, a branch of the thyrocervical trunk of the subclavian
- 3 The thyroidea ima artery which arises from the innominate artery or arch of the aorta. The veins form a plexus from which emerge the superior and middle thyroid veins, which empty into the internal jugular vein. The inferior thyroid vein joins the innominate vein.

STAGES IN THE FORMATION OF THYROXIN



The gland is covered by a loose external capsule, a part of the deep cervical fascia, and an internal one bound to the gland forming the interlobular septa. The parenchyma is composed of groups of acini or follicles lined by low cuboidal epithelium. The acini are normally filled with iodine containing colloid, which after fixation stains deeply with eosin. The important relations of the thyroid gland are the larynx and trachea which it partly surrounds, the carotid sheath, laterally; the esophagus posteriorly to the trachea, the recurrent laryngeal nerve, and the parathyroid bodies.

which is released into the circulation under control of the thyroid-stimulating hormone of the anterior part of the pituitary gland.

Recently the presence of free iodinated amino acids such as triiodothyronine has been identified in thyroid tissue, and it is suggested that these free amino acids play an important part in thyroid function.

The control of hyperthyroidism by iodine is thought to be due to the following:

- 1 Inactivation of thyrotropin
- 2 Interference with the action of thyrotropin on the thyroid

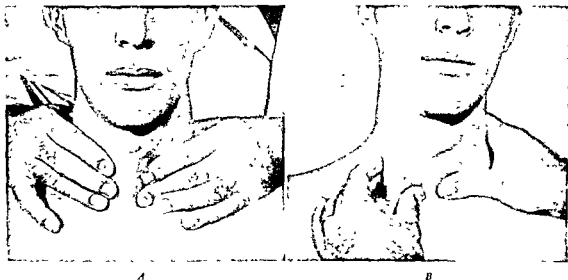


Fig. 204—Palpation of the thyroid gland. This examination can be made with the examiner standing behind or in front of the patient.

A, Palpation of the thyroid gland from behind the patient. The patient is asked to swallow, and as the gland moves between the fingers, each lobe can be carefully assessed and compared for size and consistency.

B, With the examiner standing in front of the patient, the larynx is displaced by the examiner's thumb, first to one side and then to the other. The dislocated lobe can now be accurately palpated between the fingers and the thumb of the opposite hand.

Physiology

The thyroid is an important ductless gland, regulated by the thyrotropic hormone of the anterior lobe of the pituitary gland. The formation of its internal secretion, *thyroxin*, is complex and as yet not completely understood. Ingested iodine must first be converted into sodium iodide and then, by an oxidase reaction, united with tyrosine to form diiodotyrosine. A second oxidation reaction links two molecules of diiodotyrosine to form thyroxin,

- 3 Prevention of the release of thyroxin, causing its retention in the gland.

Antithyroid drugs are believed to block an essential oxidation step in the formation of thyroxin, preventing the union of tyrosine with iodine.

There are two main functions of thyroxin.

1. To regulate body metabolism
2. To stimulate physical and mental growth

An overactive gland in adults produces a clinical state of hyperthyroidism or Graves'

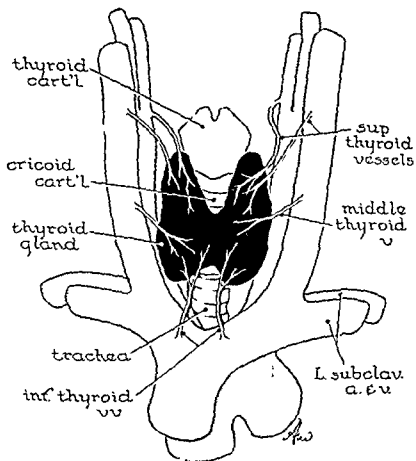


Fig. 205—Anatomy of thyroid and parathyroid glands (anterior view)

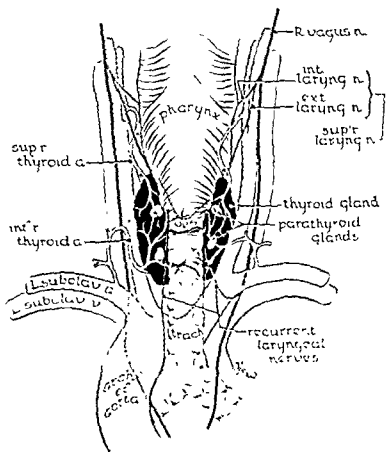


Fig. 206—Anatomy of thyroid and parathyroid glands (posterior view)

disease, whereas an underactive gland results in myxedema. In children, an underactive gland is associated with a state of cretinism—dwarfed stature, underdeveloped secondary sex characteristics, and marked mental retardation. The hypoaactive state can be controlled by the administration of thyroxin, the active hormone of the gland, or thyroid extract. Cholesterol metabolism is also influenced by the thyroid gland. Thus in hyperthyroidism, the blood cholesterol is reduced and in myxedema, elevated.

CONGENITAL ANOMALIES

Lingual Thyroid

This anomaly represents the persistence of a part or all of the thyroid gland at its point of origin at the base of the tongue or the foramen cecum. It is present from birth but tends to enlarge at puberty, during pregnancy, and at the menopause. Symptoms are due to the presence of a swelling at the base of the tongue, difficulty in speaking, swallowing, or breathing, and a desire to swallow continuously. Bleeding may result from trauma and ulceration. The tumor is single, in the midline, and occurs predominantly in females. The treatment consists of surgical removal, provided that there is thyroid tissue in the normal site; otherwise total myxedema will result.

Thyroglossal Cysts and Sinuses

Signs and Symptoms.—These anomalies may occur at any site along the course of the thyroglossal duct. They are most common below the hyoid bone but may be found at any level from the foramen cecum to the sternal notch. They present in the midline as a firm, round swelling averaging 1-2 cm in diameter. They are attached to the deep structures, particularly the hyoid bone, but not to the skin unless infection has occurred. If a sinus is present, it is also in the midline and frequently results from incision and drainage of an infected cyst. It exudes clear or purulent mucoid material. If the sinus connects with the foramen cecum, clear saliva exudes.

The differential diagnosis includes submental lymphadenitis, dermoid or sebaceous cyst, ec-

topic thyroid or pyramidal lobe enlargement, and tuberculous fistula.

The treatment is surgical removal of the cyst and tract following it to the foramen cecum, by division of the hyoid bone, if necessary. Injection of the cyst or tract with methylene blue prior to the dissection aids in tracing the sinus tract to its highest level. Unless completely removed, recurrence is inevitable. As in lingual thyroid, proof of the existence of a normal thyroid prior to excision of a cyst is essential (See also Chapter 12.)

INFLAMMATORY DISORDERS

Acute and Subacute Thyroiditis

Thyroiditis is a disease of unknown etiology. It may occur in a subacute form or as an acute suppurative process which goes on to a localized abscess requiring incision and drainage. The subacute form is the more frequent. The disease affects females twice as frequently as males and occurs chiefly in the 20-50-year age group. Upper respiratory infections are frequently a predisposing cause.

Signs and Symptoms.—There is usually a sudden onset of swelling and tenderness of one lobe of the thyroid gland, although the entire gland may be affected. Migration from one lobe to the other is common. There may or may not be fever, chills, cough, and dysphagia. The affected part of the gland is hard, tender, and the superficial tissues are frequently reddened and edematous if the process is situated anteriorly. The basal metabolic rate is not elevated, and there are no eye signs of hyperthyroidism. Most cases run a self-limiting course of several weeks or months if untreated or if localization and suppuration do not supervene.

The differential diagnosis includes hemorrhage into a degenerating colloid nodule, carcinoma, and phlegmon of the neck. In protracted and doubtful cases biopsy is indicated.

Various specific treatments have been advocated. Antibiotics do not affect the subacute type. X-ray therapy has brought about resolution of the process in many cases. The use of antithyroid drugs such as propylthiou-

racil over weeks has also produced the same result. Recently cortisone has been found to influence the fibrotic pathologic process and return the gland to normal. If a localized abscess occurs, it should be incised and drained.

Pathologically, there is evidence of a diffuse subacute inflammatory process. Leukocytic infiltration and giant cells are noted.

Chronic Thyroiditis

Two distinct forms of a chronic thyroiditis, struma lymphomatosa (Hashimoto's disease) and Riedel's struma (woody thyroiditis), are recognized

Struma Lymphomatosa.—This is a chronic degenerative disease of the thyroid gland in which there are varying degrees of lymphoid and fibrous tissue replacement. It is not regarded as the end result of subacute thyroiditis, nor is it associated with Riedel's struma. The etiology is unknown. It occurs chiefly in women in the 30-40-year age group

Signs and Symptoms.—Firm symmetric or finely nodular enlargement of the gland is the chief symptom. It may be present for years. There are no associated toxic or febrile symptoms. Many cases show a moderate degree of hypothyroidism and occasionally myxedema. Large tumors encircling the trachea cause symptoms of compression. There is no tendency toward spontaneous remission.

The *diagnosis* is usually only made at operation, and cancer of the thyroid may be suspected. Recently Crile has advocated biopsy with the Silverman needle and mild doses of x-ray therapy.

Treatment.—Thyroidectomy is usually performed on the larger types. Conservative resection is advised, since subtotal or total resection leads to severe myxedema. Enough gland is removed from the isthmus and lobes to relieve pressure on the trachea.

The *pathologic picture* is characterized by extensive degeneration of the thyroid acini and replacement by lymphoid and fibrous tissue. Well-developed germinal centers are present. There is no extension to the capsule or surrounding tissues.

Riedel's Struma.—Riedel's thyroiditis is a chronic inflammatory fibrosing process involv-

ing one or both lobes of the thyroid as well as the surrounding capsule, muscles, trachea, and blood vessels. It produces a hard bulky tumor which may be indistinguishable from advanced cancer. It is a distinct clinical and pathologic entity of which the etiology is unknown.

Signs and Symptoms.—The onset is slow, insidious, and not accompanied by pain or tenderness. Pressure symptoms are common, due to distortion or compression of the trachea. There is little systemic reaction, and the metabolic rate is normal except in advanced bilateral cases, when it is subnormal. The tumor is stony hard and fixed, leading to the presumptive diagnosis of cancer.

Treatment.—X-ray has no effect on this type of thyroiditis. Surgical removal is difficult due to fibrosis extending outside the gland. Enough gland should be removed to relieve pressure symptoms. Recurrence in the remaining lobe in unilateral cases has been reported. Radical removal is usually impossible, and the danger of injury to recurrent nerves, vessels, and parathyroid glands is increased.

Specific Thyroiditis.—Tuberculosis and syphilis of the thyroid gland are practically unknown.

TUMORS OF THE THYROID GLAND

This classification for descriptive purposes is divided into (1) nonmalignant and (2) malignant tumors.

Nonmalignant Tumors

By far the most common tumor of the thyroid gland is described loosely by the term *goiter*; other benign histoid tumors of the thyroid gland, such as fibroma, chondroma, myxoma, are so rare that no further elaboration is necessary.

Goiter, as a term applied to various types of enlargement of the thyroid gland, has been classified in various ways, none of which is entirely satisfactory. That adopted by the American Society for the Study of Goiter follows:

(A) Diffuse goiter

- (1) Without hyperthyroidism
- (2) With hyperthyroidism

(B) Nodular goiter

- (1) Without hyperthyroidism
- (2) With hyperthyroidism

In this classification diffuse goiter with hyperthyroidism corresponds to Graves' disease and nodular goiter with hyperthyroidism to "toxic adenoma."

Diffuse and nodular goiters without hyperthyroidism represent the various manifestations of endemic or colloid goiter. In addition, Crile uses the term "discrete adenoma" to describe a clinical entity when there is an adenoma which from the clinical standpoint has the qualities of neoplasia rather than of an involuntary colloid nodule. This concept has some bearing on the higher incidence of carcinoma in single as opposed to multinodular goiters.

Diffuse Goiter Without Hyperthyroidism

This corresponds to the simple adolescent colloid goiter—endemic goiter of iodine-deficient areas. It appears usually during puberty or pregnancy when the physiologic demands on the gland are greatest. With recurring episodes of iodine deficiency, the gland begins to react irregularly, and the multinodular involutionary type of goiter develops. This is the end result of many adolescent colloid goiters and accounts for the large nodular glands seen in older women in goitrous areas.

The treatment of adolescent goiter lies chiefly in its prevention, since once hypertrophy is established it never completely regresses, and this type of goiter progresses over a period of years to multinodular goiter and its complications. The universal use of iodized salt, the addition of small amounts of iodine (e. g., 1 m. Lugol's solution once a week, in early cases, will help to prevent subsequent enlargement.

Nodular Goiter Without Hyperthyroidism

This type of goiter results from previous adolescent enlargement and is the end result of endemic goiter. The complications of this type are (1) pressure symptoms from excessive growth—dyspnea, dysphagia, and hoarseness; (2) hyperthyroidism, (3) intrathoracic growth, (4) development of malignancy (5-10%).

The treatment of multinodular goiter is somewhat controversial. In the early stages, surgery may not be indicated. However, when any of the above complications are present, as well as for cosmetic reasons, thyroidectomy should be performed.

The Single Discrete Adenoma

Recently the existence of a discrete firm nodule in the thyroid gland has come to be regarded as a different and more serious problem, quite separate from that of multinodular goiter. The discrete adenoma has clinical and pathologic possibilities that make its removal advisable regardless of size or other constitutional symptoms, for it is this type of nodule that shows the highest incidence of malignancy (20%).

Diffuse Goiter With Hyperthyroidism (Graves' Disease)

The exact etiology of toxic goiter is not definitely known. Certain theories have been formed. It is thought by many that overactivity of the thyroid gland is brought about by excessive stimulation by the thyrotropic hormone of the anterior pituitary. The varying degrees of exophthalmus, when present, are regarded as due to this same source.

The use of tracer doses of radioactive iodine and the radioautograph of the gland have demonstrated important facts. By this method it has been shown that in many cases of solitary toxic adenoma, the adenoma is solely responsible for the toxicity, the remaining thyroid tissue being inactive.

Severe mental strain, anxiety, shock, and acute illnesses have been regarded as predisposing factors. It has also been observed that people with inherent psychotic personalities are likely to develop hyperthyroidism. Females are affected 3-4 times as often as males. The 20-50-year age group includes the majority of cases.

The signs and symptoms of hyperthyroidism are produced chiefly by the increased metabolic rate and increased oxygen consumption of the tissues. The objective signs are characteristic at first, but the symptoms have considerable variations and in the absence of objective signs are not reliable in diagnosis.

Nervousness, tremor, sweating, and excessive appetite are common symptoms. The objective signs include tachycardia, capillary pulse, high pulse pressure, loss of weight, diffuse enlargement of the thyroid gland, and various eye signs. Palpable enlargement of the gland and exophthalmus are not always present. Diarrhea and a negative calcium balance, resulting in skeletal decalcification, may be found in severe cases. Amenorrhea also occurs. Weakness of various groups of skeletal muscles may be a feature in some cases, especially the quadriceps group. Atrophy of the shoulder



Fig. 207.—Photograph of patient with exophthalmic goiter, showing exophthalmus and typical facies

girdle muscles is not uncommon. Carbohydrate metabolism is disturbed due to impaired glycogen storage in the liver, leading to glycosuria and elevated glucose tolerance curves, during the digestive phase.

Diagnosis.—Symptomatically, hyperthyroidism may be confused with anxiety states or neuroses, and in older individuals cardiac symptoms such as fibrillation or early decompensation may obscure the picture. The following tests are available as diagnostic aids.

1. *The Basal Metabolic Rate.* This is the oldest and most widely used test but is open to certain mechanical and personal inaccuracies. Bed rest, sedation, and repeated tests may be necessary to obtain the true level.
2. *Serum Cholesterol.* The serum cholesterol is lowered in most cases of hyperthyroidism, but this test is of limited value since the normal range is so wide (150-250 mg. %). This test is particularly useful when a change in original cholesterol value is noted with treatment.
3. *Estimation of Protein-Bound Iodine.* This test measures the amount of circulating thyroid hormone. It is considered by some to be the most valuable test, but due to technical difficulties it is not readily available.
4. *Therapeutic Tests.* The response to therapeutic treatment with iodine or an antithyroid drug can be used to determine the presence of mild hyperthyroid states. The objective signs such as slowing of the pulse rate, lowering of the blood pressure, weight gain, and reduction of basal metabolic rate, etc., will be noted following administration of the drug if thyrotoxicosis is present. The therapeutic test should not be continued longer than 2-4 weeks, after which time definitive treatment must be planned.
5. *Radioactive Iodine (I^{131}).* The estimation of the percentage uptake of a tracer dose (50 microcuries) of I^{131} by the thyroid tissue is a valuable method of diagnosis. The patient must not have taken any thyroid medication or preparation used for cholecystograms, pyelograms, bronchograms or myelograms for several weeks or months prior to the test. Correlation with the clinical picture and other thyroid tests is essential, since there is a considerable variation in the uptake of normal individuals (15-40%). This test is also required prior to the administration of a therapeutic dose of I^{131} .

Treatment.—The treatment of hyperthyroidism has in recent years become more complicated. For many years the standard procedure was preoperative preparation with iodine and thyroidectomy. While this is a satisfactory procedure in many cases, the degree of improvement prior to operation was minimal in severely toxic debilitated patients or in those with cardiac complications. The discovery of the antithyroid drugs propylthiouracil and Tapazole brought about a new era in the treatment of toxic goiter. With these drugs it is now possible to reduce a high metabolic rate to normal and keep it there for indefinite periods of time so that the patient comes to operation in the best possible physical condition.

In certain cases, the prolonged use of these drugs may bring about a permanent remission (40-60%).

Recently the use of radioactive iodine has opened a new method of controlling and curing hyperthyroidism.

CHOICE OF TREATMENT—The decision as to the best form of treatment in cases of diffuse or nodular hyperthyroidism is based on a careful clinical, metabolic, and social investigation of the individual case. While thyroidectomy after adequate preparation is still the treatment of choice in patients who are young and good surgical risks, there are definite indications for the other forms of treatment now available.

INDICATIONS FOR DEFINITIVE MEDICAL TREATMENT WITH ANTITHYROID DRUGS—

1. Recurrent hyperthyroidism following thyroidectomy

2. Older age groups or those with some physical debility contraindicating surgery

3. Certain patients who elect medical therapy. Propylthiouracil 100-200 mg. every 8 hours is given until the metabolic rate falls to normal or slightly subnormal. The dosage can then be reduced until the maintenance dose is found. Treatment usually must be continued for 12-14 months, at which time the drug is gradually withdrawn. Permanent remissions vary between 40-60%. In moderately and severely toxic patients, with enlarged glands, the recurrence rate after discontinuing antithyroid drugs is high and this type of case does better with thyroidectomy.

INDICATIONS FOR USE OF RADIOACTIVE IODINE—The use of radioactive iodine has increased in recent years with the greater availability of radioactive isotopes. Encouraging results are reported from various centers. The drug is given in water, either one estimated total dose or multiple small doses of 5-10 millicuries at intervals. The incidence of myxedema seems to be higher with the single-dose technique. The multiple-dose method requires about 6 months to secure control of hyperthyroidism. The possibility of late effects of gamma radiation on the thyroid gland is not yet fully known. Whether radioactive isotopes have carcinogenic and genetic effects is still

controversial. Some centers restrict their use to the following groups:

1. Those patients who are 45 years of age and over

2. Recurrent cases that are poor surgical risks

3. Patients who refuse surgery and do not respond to antithyroid drug treatment

Thyroidectomy.—

PREOPERATIVE TREATMENT.—In preparation for operation, the patient is given propylthiouracil, 100-200 mg. every 8 hours until an euthyroid state is reached. Lugol's solution 5-10 m three times a day is next added for 7-10 days to reduce the vascularity and friability of the gland. Subtotal thyroidectomy is then performed. During the administration of iodine or antithyroid drugs, careful attention must be given to other aspects of hyperthyroidism. Adequate rest or bed rest with bathroom privileges is required. A high, caloric, high vitamin diet is essential to restore lost weight. Extra carbohydrates are necessary to replace glycogen depletion of the liver. Sedation, bromides, and barbiturates are useful in controlling nervous manifestations. Patients with incipient decompensation or auricular fibrillation should be digitalized prior to operation.

ANESTHESIA—The early technique of local infiltration and cervical block has largely disappeared in favor of newer anesthetic agents. The administration of a basal anesthetic such as Pentothal or Avertin in the patient's room, followed by intubation and endotracheal nitrous oxide or cyclopropane anesthesia, is the ideal combination. It assures an unobstructed airway at all times and reduces the total amount of anesthetic agent required.

TECHNIQUE—The technique of thyroidectomy is now well standardized and emphasizes the following points: (1) a well-controlled properly prepared patient; (2) anatomic dissection of the gland; (3) exposure and protection of the recurrent laryngeal nerves; (4) preservation of the parathyroid glands; (5) resection of the optimum amount of gland to effect a cure but not to produce myxedema; (6) careful hemostasis and good cosmetic closure of the incision.

A low collar incision is placed 1-1½" above the sternal notch. The superior thyroid artery

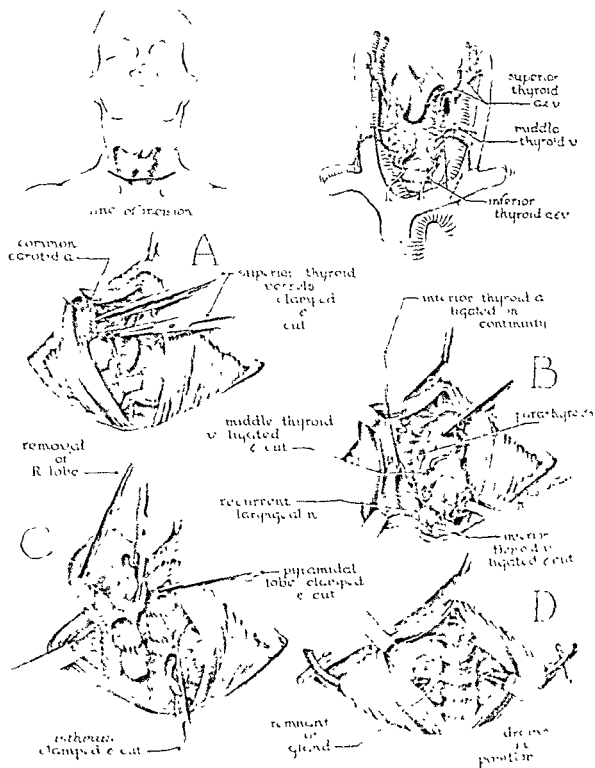


Fig 208—Technique of the stages of thyroidectomy

is then isolated, clamped, and doubly ligated with silk or heavy catgut, care being exercised not to injure the superior laryngeal nerve

The middle thyroid and inferior thyroid veins are now divided and ligated, and the gland is rotated medially to expose the posterior capsule. The inferior thyroid artery is identified and ligated well away from the capsule. The parathyroid glands may occasionally be identified close to the entrance of the branches of the inferior thyroid artery into the gland. Careful dissection in the region of the branches of the inferior thyroid artery exposes the recurrent laryngeal nerve as it approaches the gland and reduces the incidence of nerve damage. The gland is now divided across the isthmus, and the lobe is resected from the midline outward, leaving the posterior capsule intact over the recurrent nerve and the parathyroid bodies. The amount of tissue left is largely an individual factor. Usually it represents only a thin layer of tissue in the tracheo-esophageal angle and on the posterior capsule. The isthmus and pyramidal lobe, if present, should be removed. The opposite lobe is similarly resected, careful hemostasis is secured; drains are placed to either thyroid bed emerging behind the sternomastoid muscles in the line of incision. The cervical fascia and thyroid muscles are sutured, and the skin is approximated carefully with clips.

COMPLICATIONS —

A. Early —

1 *Postoperative shock* is prevented by the use of adequate amounts of glucose solution and blood or plasma if indicated.

2 *Postoperative hemorrhage* Postoperative bleeding will occur occasionally even after careful attention to hemostasis. If a large enough clot develops, it will eventually cause sufficient pressure on the trachea and larynx to produce dyspnea and stridor. If these symptoms are not relieved, the patient will die from asphyxia. Inspection of the wound and, if bulging and tense, removal of clips and opening of deep cervical fascia may be a lifesaving procedure. Later evacuation of the clot and ligation of the bleeding point can be done in the operating room. If the laryngeal compression has been present for some hours before release, a temporary tracheostomy may be required to pro-

vide an adequate airway since edema and swelling of the true and false cords may be marked enough to obstruct respiration.

3. *Recurrent nerve injury.* Partial or complete injury to one recurrent laryngeal nerve does not, as a rule, give any serious respiratory embarrassment. There will be difficulty in expectoration of mucus. The voice will be hoarse but ultimately will return more or less to normal. Section of both nerves produces complete obstruction of the airway since both cords fall into the cadaveric position. Serious dyspnea, cyanosis, and stridor develop soon after operation and require lifesaving tracheostomy.

4. *Tetany* results from removal of parathyroid bodies and may be temporary or permanent. It usually manifests itself about the third or fourth postoperative day with numbness and tingling, carpopedal spasm, and positive Chvostek's sign. The administration of calcium gluconate intravenously will relieve any alarming symptoms. The blood calcium is usually below 8 mg %. The majority of cases are transitory and yield to symptomatic treatment. Permanent tetany requires the use of A. T. 10 (dihydrotachysterol) and increased calcium ingestion.

5 *Thyroid crisis* This is an increase in all the preoperative symptoms, tachycardia, hyperthermia, extreme restlessness, delirium, and coma, which may appear during the first 24-48 hours postoperatively. It occurs rarely and less frequently since the use of the anti-thyroid drugs. The treatment consists in heavy sedation, morphine 15-30 mg or even sodium Pentothal intravenously, and copious intravenous fluids, 3,000-5,000 ml of 5% glucose solution daily. Lugol's solution 20-60 ml may be added to the intravenous fluid once or twice a day. The hyperthermia is controlled by ice water or alcohol sponges, or the patient may be literally packed in ice. Death in delirium and coma rarely results.

B. Late —

1 *Recurrent hyperthyroidism* as distinguished from continuing hyperthyroidism occurs in about 2% of thyroidectomies for toxic goiter. Years may elapse before the recurrence.

2 *Myxedema* or *hypothyroidism* is seen in temporary or permanent form in 3-5% of

thyroidectomies. It yields readily to the administration of thyroid extract and does not constitute a disability.

3. *Malignant exophthalmos*, a progression of the exophthalmos found in diffuse toxic goiter, occurs in a small number of cases even when the hyperthyroidism has been controlled by medication or operation. It is thought to be due to a continuation of stimulation by the thyrotropic factor of the anterior pituitary. In extreme cases marked proptosis, edema of the cornea, sclera, and eyelids, diplopia, corneal ulceration, and ophthalmitis may occur. Gradually increasing doses of thyroid extract 0.12-0.48 Gm. a day over long periods of time have produced clinical improvement. Large doses of estrogens and pituitary irradiation have also been used with varying success.

Orbital decompression by removal of the roof of the orbit and periorbital fat is indicated in severe cases. Osteoplastic recession of the eyeball (Naffziger) has been necessary to avoid the possibility of an enucleation of the eye.

Nodular Goiter With Hyperthyroidism

Hyperthyroidism associated with nodular goiter occurs most often in older age groups from 40-60 years. There is little difference in the signs and symptoms from those of Graves' disease. There is a higher incidence of cardiac complications, auricular fibrillation, and early decompensation, due partly to the advanced age group and partly to the chronicity of many of these cases. The toxicity may be undiagnosed until cardiac symptoms supervene. The same principles apply to the management of this type of goiter as those laid down for diffuse toxic goiter. It is usually unwise to attempt definitive medical treatment with antithyroid drugs, and thyroidectomy after suitable preparation is indicated.

Malignant Tumors of the Thyroid Gland

Carcinoma of the thyroid gland is the most common form of malignant tumor encountered, but rare cases of sarcoma have been reported. Metastatic involvement of the thyroid gland is also very rare.

Carcinoma of the Thyroid Gland

The etiology of cancer of the thyroid is unknown. The presence of a pre-existing nodule has been reported in as high as 50% of cases. Diffuse hyperplasia rarely results in carcinoma.

The antithyroid drugs are carcinogenic in animals in large doses, and reports of carcinoma developing in patients with toxic goiter who are receiving prolonged thiouracil therapy have been recorded.

Cancer of the thyroid is more common in goitrous districts, affects females 3-4 times as frequently as males, and occurs most often in the 40-70-year age group.

Classification.—The various types of cancer of the thyroid differ markedly in their degree of malignancy and hence their prognosis.

A classification on this basis is the most satisfactory.

- I. Tumors of low-grade malignancy
 - (a) Papillary cystadenoma
 - (b) Hurthle's cell tumor
- II. Tumors of moderate malignancy
 - (a) Papillary adenocarcinoma
- III. Tumors of high-grade malignancy
 - (a) Adenocarcinoma
 - (b) Giant cell carcinoma
 - (c) Spindle cell carcinoma

The earliest symptom is the presence of a nodule in the thyroid gland which suddenly and progressively enlarges and becomes firmer than the surrounding gland. Nodularity and calcification may be noted. The appearance of dyspnea, dysphagia, pain, voice changes, loss of weight, and fixation of the mass to surrounding structures are all late manifestations and represent usually an inoperable or incurable stage of the disease. About 15-20% of cases may show varying degrees of toxicity.

The clinical diagnosis of carcinoma of the thyroid is a dubious one and, if obvious, usually represents an inoperable stage. The diagnosis is unsuspected in about 40-50% of cases of surgically removed nodules and is only made on careful pathologic study; 10% of surgically removed nodular goiters and 24% of solitary nodular goiters have been reported carcinomatous. Careful scrutiny of all nodular goiters and exploration of all suspicious cases, especially the solitary nodules, are clearly indicated. The scanning of nodules following the

use of a tracer dose of I^{131} is a useful procedure. The finding of a so-called "cold" nodule may indicate a carcinoma.

The treatment is radical thyroidectomy, but the technique is dictated by the individual case and the type of lesion suspected. In those cases where a subtotal thyroidectomy has been performed and the diagnosis made only on histologic examination, if the focus is small and completely removed, the prognosis is good. Postoperative x-ray therapy gives added protection. In the papillary type of carcinoma, if glandular metastases are present, a careful dissection of the region involved is performed. X-ray therapy postoperatively is usually indicated.

In treating the more malignant types of carcinoma, ligation and resection of the internal jugular vein well above the limits of the gland are performed before removal of the thyroid lobe on the affected side is undertaken. Similarly, the inferior thyroid veins are ligated early. This is done to prevent blood stream contamination during operation. The strap muscles and recurrent laryngeal nerve are sacrificed if involved by the tumor. Postoperative radiation is essential. Bilateral involvement by carcinoma is rarely operable. Frankly inoperable cases may be temporarily improved by heavy roentgentherapy usually combined with tracheostomy.

Radioactive iodine has been used in the treatment of carcinoma of the thyroid. Unfortunately most carcinomas of the thyroid are nontoxic and do not take up radioactive iodine in sufficient quantity to be of any therapeutic value.

The rare cases with toxicity have been successfully controlled with large doses of radioactive iodine over a long period of time (250 millicuries over a 3-year period). The use of tracer doses and estimation of the uptake by the involved gland or metastatic lesions indicate the possibility of treatment by this method.

The use of antithyroid drugs has been found to increase the absorption of radioactive iodine in certain cases.

Total thyroidectomy has been performed and causes an increased uptake of radioactive iodine by the metastatic lesions.

Radioactive iodine has not proved to be of significant value for the treatment of carcinoma of the thyroid gland.

THE PARATHYROID GLANDS

Surgery of the parathyroid glands involves chiefly the treatment of hyperparathyroidism, which is caused by an adenomatous enlargement of one of the glands.

Embryology

The parathyroid glands first appear as thickenings of the third and fourth pharyngeal pouches in the 10 mm embryo. Those arising from the third pouch remain attached to the thymic body, descend with it, and eventually lie on the posterior surfaces of the inferior poles of the thyroid. The other two glands remain in their original positions and are related to the posterior surfaces of the superior poles of the thyroid gland. This mode of origin accounts for the variability in the number and position of the parathyroid glands.

Physiology

The function of the parathyroid glands is to regulate calcium metabolism, through their internal secretion, *parathormone*, which was isolated by Collip in 1925. Surgical removal of the parathyroids leads to a state of tetany. Overactivity of the parathyroids due to hyperplasia or adenoma results in the clinical syndrome of hyperparathyroidism. The parathyroid hormone produces first an excess secretion of phosphorus in the urine, leading to hypophosphatemia. Then due to the solubility relationship with calcium, hypercalcemia develops, accompanied by hypercalcinuria.

When the serum phosphorus is lowered the serum calcium is raised and vice versa. Thus in hyperparathyroidism the alteration of phosphorus metabolism creates a demand for calcium which is derived first from the gastrointestinal tract or, failing this, from the bones.

There is some evidence that the parathyroid hormone may have a slight direct action on bone itself.

Primary Hyperparathyroidism

This disease was known for many years as von Recklinghausen's disease of bone or

osteitis fibrosa cystica until 1925 when Mandl demonstrated the exact relationship between the clinical syndrome and parathyroid adenoma.

Etiology.—The cause of this condition in the great majority of cases is due to adenoma of a parathyroid gland, but in occasional instances there is hyperplasia of all parathyroid glands. The symptomatology is the same in either case. It occurs most commonly in the 20- to 40-year age group.

Signs and symptoms may be divided into three separate groups

1. Those due to increased blood calcium. The blood calcium is always elevated, 12-18 mg % and the blood phosphorus reduced below normal (1-2 mg %). This results in lassitude, hypotonia, weakness, constipation, anorexia, and loss of energy.
2. **Skeletal involvement.** The excessive mobilization of calcium from the bones results in marked skeletal decalcification as evidenced by x-ray. Spontaneous fractures are common. Bone cysts and tumors which may be single, but are usually multiple, are found on x-ray survey of the skeletal system.
3. **Symptoms of renal involvement.** Due to excessive urinary excretion of calcium and phosphorus, calculi, or calcinosis of the renal parenchyma, may occur. Signs of renal colic and renal infection are common.

The diagnosis may be difficult due to the variability of the signs and symptoms and simulation of other diseases. However, since the clinical entity has become better known, early diagnosis is less difficult. The x-ray and blood pictures are characteristic and usually diagnostic. The performance of a calcium balance study indicates that the patient is markedly negative balance. The patient is placed on a measured calcium intake (120 mg daily) and the excretion in the urine estimated. If the urinary excretion is above 200 mg/24 hours, hyperparathyroidism is suspected.

Rarely the tumor may be palpable in the neck, or there may be forward displacement of the lateral thyroid lobe.

The treatment of hyperparathyroidism is exploration of the posterior surface of the thy-

roid gland and removal of the adenoma or resection of two or three glands if hyperplasia is found. Considerable difficulty may be encountered if the adenoma is situated anomalously; cases requiring three, four, and five operations are reported.

The most common site for adenomas is at either lower pole (35% each), but they may be found in the carotid sheath, behind the esophagus, and in the superior mediastinum.

Careful anatomic dissection must be maintained until the tumor is found. Frozen section of suspicious nodules removed at operation is a great aid in finding the adenoma, since the histologic picture is quite characteristic.

Complications.—Injury to recurrent laryngeal nerves is best avoided by careful dissection and identification of these structures early in the operation.

Postoperative tetany due to a sudden drop in blood calcium levels must be anticipated. Frequent determination of the blood calcium during the first postoperative days and the use of calcium gluconate intravenously and by mouth will usually control the hypocalcemia until the remaining glands stabilize the blood level.

A. T. 10 (dihydroxycholesterol) also aids in maintaining normal blood calcium levels over long periods of time.

Results.—Complete recalcification of the skeletal system occurs in about a year following removal of a parathyroid adenoma.

Secondary Hyperparathyroidism

This results from an overcompensating mechanism in conditions where there is a disturbance of the calcium-phosphorus metabolism. This disturbance is a tendency to low serum calcium found in pregnancy, lactation, rickets, osteomalacia, and renal osteitis fibrosa generalisata. This type of hyperparathyroidism usually responds to medical measures, and operation is rarely necessary.

THE ADRENAL GLAND

The cortex and the medulla are discussed separately because of the distinct difference in their physiologic activity and pathologic states.

THE ADRENAL CORTEX

Anatomy

The adrenal glands are situated extraperitoneally, above and in front of the upper poles of the kidneys and on either side of the spine. The left gland is usually larger than the right one. The average weight of a gland is from 3.4 Gm. They are surrounded by a considerable amount of fat and connective tissue. Each gland is divided into the cortex and the medulla.

The cortex is deep yellow in color and contains three distinct layers.

The *external zone* (zona glomerulosa) is relatively small in the adult and consists of

cortex the columns of cells are surrounded by a capillary network, while in the zona reticularis there are sinusoids formed between the cell groups. These sinuslike spaces then drain into sinusoids present in the medulla which open into veins that merge and leave the gland as the adrenal vein. On the right side this drains into the inferior vena cava, on the left into the renal vein.

The lymphatics run in the capsule and drain into the lumbar glands.

The nerve supply is derived from the celiac and renal plexuses, which in turn receive cholinergic fibers from T₅-L₁ via the splanchnic nerves.

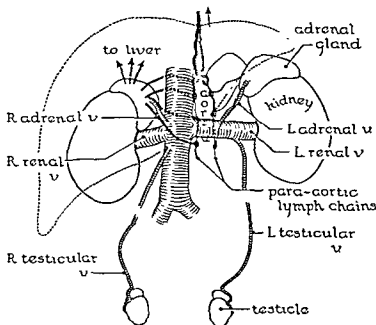


Fig. 209—Venous drainage of the three paired glands, and lymphatic drainage of the adrenals

small cells forming groups indicating an alveolar structure.

The *middle zone* (zona fasciculata) makes up the bulk of the cortex. Large polygonal cells run in regular rows between which are sinusoids.

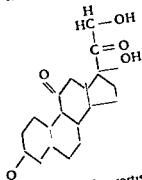
The *inner zone* (zona reticularis) consists of small cells with dark nuclei, probably the remnant of the fetal zone which did not undergo involution.

The blood supply is rich and derived from branches of the aorta and the inferior phrenic and renal arteries. In the outer part of the

Physiology

Twenty-eight crystalline steroids have been isolated from the adrenal cortex and their structures identified chemically. Of these 28 steroids, seven have been shown to have physiologic activity. They are corticosterone (cpd B), 17-hydroxycorticosterone (cpd F), 11-dehydrocorticosterone (cpd A), 17-hydroxy-11-dehydrocorticosterone (cortisone) (cpd E), 11-desoxycorticosterone (DCA), and 11-desoxy-17-hydroxycorticosterone (cpd S). Aldosterone has recently been isolated. Following extraction of the known active principles,

the amorphous fraction is left. Many investigators feel that a wealth of material remains to be discovered in the amorphous substance



Chemical formula for cortisone

It is convenient to think of panels of activity in discussing adrenal cortical function

Carbohydrate panel 17-hydroxycorticosteroids are probably the most active of the 11 oxygenated adrenal steroids in causing liver glycogen deposition. This glycogen is derived from an increased catabolism of protein. The action of the hormone or hormones is at the level of the whole protein or the inhibition of synthesis of protein from amino acids. Gluconeogenesis is the term used for this breakdown of protein to carbohydrate.

This activity is measured in the blood by the Porter-Silber technique or by the Nelson-Samuels modification. Free 17-hydroxycorticoids or possibly conjugates are extracted. Normal values range from 7-15 gamma %.

The total urinary 17-hydroxycorticoids measured by the Porter-Silber method includes both biologically active, essentially free material and mostly inactive conjugates. Normal values range from 3-9 mg/24 hours.

Electrolyte panel. The influence on electrolyte metabolism is on the renal tubular function as well as the distribution of electrolytes in the intracellular and extracellular compartments. In the absence of cortical hormones, the tubules are incapable of reabsorbing sufficient sodium and chloride from the glomerular filtrate to maintain normal electrolyte and water balance. The ability of the kidney to excrete potassium is decreased, and a rise in serum potassium follows.

All the biologically active corticosteroids increase the reabsorption of sodium and chloride by the renal tubules. There is a considerable difference in magnitude of effect. Hydrocorti-

sone has the least effect, while deoxycorticosterone and aldosterone have the greatest effect on sodium retention. The latter compound is at least 1,000 times more effective in this regard than is hydrocortisone. With the retention of sodium there is a reciprocal loss of potassium. Cortisone and hydrocortisone maintain cellular hydration at an optimum, while deoxycorticosterone leads to intracellular overhydration and water intoxication when used in excess. Aldosterone occupies an intermediate position.

Androgenic panel: This function is measured as 17-ketosteroids in the urine. The values vary with the method used. The excretion remains at a low level from birth to the age of 7 years (0.1-1.5 mg./24 hours). With the onset of puberty the values rise sharply until adult levels are reached (normal male average, 14 mg./24 hours; female average, 9 mg./24 hours). After the age of 50 years the excretion gradually decreases in both sexes.

In women the adrenal cortex is the only source of 17-ketosteroids from the testes and production of 17-ketosteroids from the testes and adrenal cortex is under the influence of the pituitary, disorders in anterior pituitary function affect the output. In primary hypothyroidism, liver damage, and malnutrition, the output of 17-ketosteroids is very low. Adrenal hyperfunction due to hypertrophy, adenoma, or carcinoma of the cortex results in high urinary values of 17-ketosteroids. The assay of the β -hydroxy fraction has been used as a means of differentiating between cortical hyperplasia and malignant tumors. This fraction is high when such tumors are present and is not raised when a benign tumor is present.

Diseases of the Adrenal Cortex

In this instance our concern is only with pathologic conditions of the cortex which result in hyperfunction. Hypofunctioning syndromes are not amenable to surgery.

Hyperfunction of the Adrenal Cortex. Hyperplasia, adenoma, or carcinoma of the cortex may be responsible for the clinical syndromes. Such diseases may be conveniently classified as follows:

1. Complete hyperfunction
- a. Cushing's syndrome

- b. Mixed syndrome
- c. Anterior pituitary basophilic adenoma
- 2. Partial hyperfunction
 - a. Adrenogenital syndrome
 - b. Pseudohermaphroditism
 - c. Feminization in the male
 - d. Adrenal hyperplasia associated with symptoms of Addison's disease

Cushing's Syndrome—This syndrome has been discussed under the pituitary gland (see page 385). Specific diagnostic aids with reference to adrenal pathology include the following.

1. Flat plate of the abdomen to note any abnormal shadows and position of the kidney.

Tomograms may be helpful; however, false positive interpretations may result as has been found for insufflation studies.

ACTH stimulation and cortisone suppression tests have been widely used to differentiate between adrenocortical hyperplasia and adenoma. Hyperplasia of the cortex will lead to a brisk response to ACTH stimulations (20 IU. q 6h for 48 hours), whereas administration of cortisone (25 mg. q 8h) or 9-alpha-fluorohydrocortisone (1 mg. b i d) will result in suppression of blood 17-hydroxycorticosterone and urinary corticoids. Adenoma or carcinoma of the cortex will show little change when such procedures are performed. Further work must

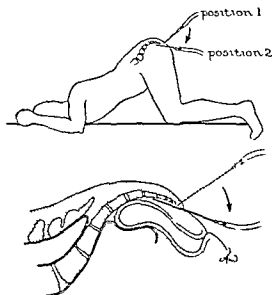


Fig. 210—Technique of presacral peritoneal insufflation of oxygen

The patient is placed in the knee-chest position, and the sacrococcygeal and surrounding area is cleaned and painted with Merthiolate solution. The patient is draped with sterile towels. The skin and subcutaneous tissue at the tip of the coccyx are infiltrated with 1% procaine. A No. 20 lumbar puncture needle is inserted just beneath the tip of the coccyx, then the direction altered so that the tip points dorsally into the hollow in front of the sacrum. When the end of the needle is felt to be in the sacral hollow, 1,500 ml of oxygen are slowly injected. This diffuses upward and surrounds the kidneys and adrenals. The positioning of the patient controls the diffusion of gas.

2. Intravenous and retrograde pyelography which may demonstrate downward displacement of one kidney

3. Presacral peritoneal insufflation of oxygen. This procedure is sometimes helpful but in many instances results in either shadows being read as tumor when none was present or failing to demonstrate a tumor when such was present. The technique of this procedure is shown in Fig. 210.

be done before these tests can be interpreted with accuracy.

Mixed Tumors—This term is used to denote both androgenic and metabolic disturbances. In such instances the patient will usually present signs of virilization but of a milder degree than that seen in the adrenogenital syndrome (following), as well as some disturbance of carbohydrate metabolism, although less than is seen in Cushing's syndrome.

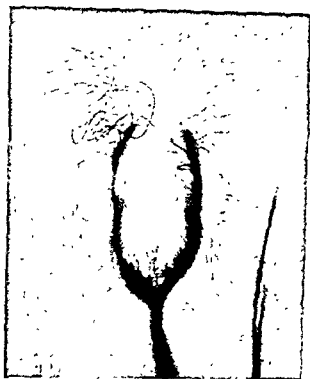


Fig. 211 —Congenital bilateral adrenal hyperplasia. The skin biopsy was examined by Dr. Barr and the patient found to be a female. She was 5 years of age.



Fig. 212 —A, Presacral air insufflation showing normal adrenals.
B, Presacral air insufflation showing large, round shadow in region of left adrenal.

The pathology present may be that of hyperplasia or adenoma. The hyperplasia may be unilateral. Rarely is it due to carcinoma.

The diagnostic procedures are similar to those which are necessary in the diagnosis of Cushing's syndrome.

Adrenogenital Syndrome.—Patients suffering from this condition are virilized. The term virilization refers to masculinization of the female with the result that there is loss of head hair, recession of temporal hair, acne, hirsutism, lowering of the voice, enlargement of the thyroid cartilage, amenorrhea, and enlargement of the clitoris. This syndrome must be differentiated from masculinization of the female caused by tumors of the ovary, such as arrhenoblastoma, luteomas, or hyperfunctioning Leydig cells in the hilus of the ovary. Pelvic examination, if necessary under anesthesia, will assist in excluding an ovarian tumor. The 17-ketosteroid excretion is not as high when an ovarian tumor is present as it is with an adrenal tumor.

Pseudohermaphroditism.—This condition is due to adrenal cortical hyperfunction in utero. It is said to be always due to hyperplasia. It must not be confused with true hermaphroditism which is a genetic abnormality.

When female pseudohermaphroditism is present the child is born with excess hair and undetermined sex. The clitoris is enlarged to penislike proportions. Sex may have to be determined by laparotomy, although the need for such has been greatly lessened by sex chromatin study (Barr and Marshall). This includes skin biopsy and blood or sputum studies. It appears that the combination of two X chromosomes (male) leaves a spot of sex chromatin below the nuclear membranes.

If the child is a female, a clitoridectomy is then performed.

Due to the large amounts of androgenic substance secreted from the adrenal cortex, such children grow very rapidly, but unless treatment is instituted, there will be such rapid closure of the epiphyses that retarded growth will result.

Feminization in the Male.—Enlargement of the breasts and atrophy of the testicles are the outstanding features of this disease. In 11

recorded cases each had a malignant cortical carcinoma.

Treatment. When a definitive anatomic lesion is demonstrated by palpation or other means previously discussed, surgical removal is indicated. In many instances surgery is performed both as a diagnostic as well as a therapeutic measure.

Treatment of adrenal cortical hyperfunction by means other than surgery has not been successful except in the pseudohermaphrodite. In this disease 25-50 mg of cortisone administered orally each day will depress adrenal cortical androgenic function to normal.

X-ray therapy has been directed both at the adrenal and the pituitary. The administration of estrogens has likewise not proved successful.

Surgical removal of the adrenal gland may be done through (1) the usual lumbar incision as for exposure of the kidney, which gives relatively poor exposure; (2) thoracoabdominal approach by traversing the pleura and diaphragm, as described by Chute; or (3) a dorsal lumbar flap incision, in which the angles of the 11th and 12th ribs are removed, as described by Nagamatsu. This latter incision gives excellent exposure of the adrenal gland. It is felt that greater efficiency is attained if two teams of surgeons work simultaneously.

Preoperative and Postoperative Care.—If one adrenal is found to be normal, then the other adrenal must be explored. If an adenoma is found, extirpation is performed. If hyperplasia is present, then a bilateral adrenalectomy is the treatment of choice. It is well to assume that the patient will have no adrenal cortical function for at least 5-7 days postoperatively and therefore preoperative, operative, and postoperative treatment is necessary. Cortisone 25 mg is administered orally q 6h, beginning two days preoperatively. On the morning of operation 100 mg of cortisone is given intramuscularly. This is repeated in 8 hours' time. When the patient is able to swallow, cortisone may be given orally in 25 mg doses q 6h. for seven days postoperatively. If an adenoma has been removed, the cortisone is gradually reduced (12.5 mg. q d). If hyperplasia was found and bilateral adrenalectomy performed, the patient can be maintained on 25-50 mg of cortisone per day.

SURGERY OF THE ENDOCRINE GLANDS

If during the operative procedure the patient should collapse, 100 mg of compound F (hydrocortisone) may be given in the intravenous 5% glucose and saline



Fig 213 Gross specimen of adrenal cortical carcinoma and atrophied or congenital aplastic kidney

Blood Supply.—Branches from the inferior phrenic and renal arteries as well as the abdominal aorta maintain the blood supply. The veins empty into the inferior vena cava on the right and renal veins on the left.

Nerve Supply.—This is derived from the celiac and renal plexuses which in turn receive cholinergic fibers from T₅-L₁ via the splanchnic nerves. These fibers go through the cortex to the medulla. The secretion of epinephrine is greatly diminished when the splanchnic nerve is severed.

Physiology

Two hormones have been extracted from the adrenal medulla—Epinephrine and norepinephrine. A comparison of these two hormones is given in summarized form in Table 14

Diseases of the Adrenal Medulla

Hypoadrenalism.—This is not an established clinical entity, although when there is cortical destruction, the medulla may also be destroyed

Hyperadrenalism.—The only functioning tumor of the adrenal medulla is the pheochromocytoma. However, many other tumors occur, and these are discussed briefly. Considerable difficulty exists in understanding the nature of medullary tumors. This is due to the variety of different cell types which may be present in different parts of the same tumor or its metastasis. Cells may be found in all stages of development. The degree of malignancy depends upon the number of primitive cells present

Adrenal Medullary Tumors.—See Tables 15 and 17

Diagnostic Techniques.—These are essentially the same as those described for the adrenal cortex.

Treatment.—Surgical removal of the tumor is as described for the adrenal cortex. Preoperative and postoperative care of the patient in whom a pheochromocytoma is to be removed is important. Profound vascular alterations occur with surgical manipulation of the tumor. The patient may develop epinephrine

THE ADRENAL MEDULLA

Anatomy

At birth, the adrenal medulla is small and most of the chromaffin tissue exists as paraganglionic masses along the abdominal aorta. It is derived from ectoderm. The predominantly functional cell is the pheochromocyte. Sympathoblasts arise from sympathogonia which in turn arise from the neural crest. The subsequent development of the sympathogonia is as follows

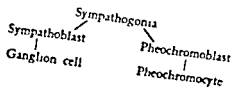


TABLE 14

	EPINEPHRINE	NOREPINEPHRINE
Chemical difference:	Tissues (enzyme)	Absent N methyl group
Site of inactivation	70-90%	Tissues (enzyme)
Ratio in man:	1 Vasodilatation	10-30%
Physiologic action:	2 Myocardial stimulation	1 Vasoconstriction
	3 Increased blood flow in muscle	2. No effect
	4 Increased splanchnic blood flow	3 Decreased
	5 Renal blood flow reduced	4. No effect
	6 Hyperglycemic effect	5 Same
		6 Less effect

TABLE 15
ADRENAL MEDULLARY TUMORS

EMBRYOLOGIC CELL (SYMPATHOGONIA)			
NERVOUS TYPE OF CELL (NORMAL DEVELOPMENT)	TUMOR	ENDOCRINE TYPE OF CELL (NORMAL DEVELOPMENT)	TUMOR
Sympathoblast	Sympathoblastoma	Pheochromoblast	Pheochromocytoma
Sympathetic Ganglion cell	Ganglioneuromata (usually benign —rare)	Pheochromocyte	(Synonyms: Paraganglioma Chromaffinoma)
	Neuroblastoma (very malignant)		

TABLE 16
METASTATIC SPREAD FROM ADRENAL MEDULLARY TUMORS

TUMOR OF RIGHT ADRENAL GLAND		TUMOR OF LEFT ADRENAL GLAND	
Pepper type of metastases to	<div style="display: inline-block; vertical-align: middle;"> <div style="font-size: 2em; vertical-align: middle;">{</div> <div style="display: inline-block; vertical-align: middle;">liver retroperitoneal lymph nodes</div> </div>	Hutchinson type of metastases to	<div style="display: inline-block; vertical-align: middle;"> <div style="font-size: 2em; vertical-align: middle;">{</div> <div style="display: inline-block; vertical-align: middle;">orbit skull</div> </div>
(Right lymphatics empty directly into the portal system, and the right adrenal gland lies in direct contact with the liver)		(Left lymphatics are connected with lumbar glands and pass downward to groin and aortic glands, and upward to the deep cervical glands and the skull)	

shock It is recommended that a continuous intravenous drip containing Regitine or Dibinamine be maintained in order to block the possible hypertensive episodes Postoperative noradrenalin is helpful in overcoming the hypotensive phase which so often follows extirpation.

Metastatic Medullary Tumors—The "Pepper type" of metastases is considered to be due to a primary neuroblastoma of the right adrenal gland, with the bulk of the metastases occurring in the liver (This has been explained as being due to the fact that the right adrenal lies against the bare area of the liver and that the lymphatics of the right adrenal empty directly into the portal system) The

"Hutchinson type" of metastases consists of a primary tumor of the left adrenal, with initial metastases to the skull and orbit. (This is explained on the basis that the lymphatics from the left adrenal gland ascend along the aorta to the thoracic duct and reach the skull sooner and more directly than when conveyed by the lymphatics of the right adrenal gland)

Some authorities disagree with this concept, and statistics reported show that in 56 examples of the "Pepper type," the growth was found in the right adrenal in 27, in the left adrenal in 21, and was bilateral in 8. In 83 examples of the "Hutchinson type," the primary tumor was in the left adrenal in 38 cases, in the right adrenal in 35, and bilateral in 10

SURGERY OF THE ENDOCRINE GLANDS

TABLE 17

	AGE GROUP	SEX	5 & 5	Due to metastases	Due to enlarging tumor	MALIGNANT	APPEARANCE
						Yes—fatal in 1 mo.-1 yr	
Neuroblastoma	Children	Equal					Yellow-gray soft nodular—contain both sympathogonia and sympathoblasts
Ganglioneuroma	Any age	Females					Not characteristic—grossly rounded nodular encapsulated—contain ganglion cells
Schwannoma							
Pheochromocytoma	Any age 1st 3rd decade	Equal				Rarely	Occurs twice as often in the right side; 5-10 cm in diameter—rounded encapsu- lated yellow-brown on section— contain large polyhedral cells
Attacks of Palpitation Tremor Anxiety Giddiness Headache Precordial pain Nausea and vomiting Skin is pale Perspiration Pupils dilated Pulse rapid Systolic B.P. raised Hypertension Glycosuria Provocative diagnostic tests*							

*These include two groups of drugs
1. Histamine, tetraethylammonium chloride, and methanoline.
2. Benzedrine, Roeline (Regitine) (5 mg in 1 ml. saline intravenously—a fall of systolic pressure of 35/25 mm. Hg is significant).

†Rarely occurs in nodules

ISLET CELL TUMORS OF THE PANCREAS AND HYPERINSULINISM

Islet cell adenomas were first described by Nicholls in 1902, but no mention of a functioning tumor was made until 1927, when Wilder et al. reported a case of islet cell carcinoma with liver metastases which produced hyperinsulinism and hypoglycemia. In 1929 Roscoe Graham successfully removed the first functioning benign islet cell tumor with a complete recovery of the patient.

Hyperinsulinism with its resulting hypoglycemia is caused by a hyperfunctioning tumor of the pancreas. It should not be confused with functional hypoglycemia which occurs as the result of a disturbance of the blood-sugar regulating mechanism. This latter syndrome usually occurs in patients with emotional instability and with an imbalance of the autonomic nervous system.

There are numerous causes of spontaneous hypoglycemia. The following classification developed by Conn gives a comprehensive outline of these causes:

- I Organic—recognizable anatomic lesion
 - A Hyperinsulinism
 - 1 Pancreatic islet cell adenoma
 - (a) Single
 - (b) Multiple
 - (c) Aberrant
 - 2 Pancreatic islet cell carcinoma
 - (a) Localized
 - (b) With metastases
 - 3 Generalized hypertrophy and hyperplasia of the islets of Langerhans
 - B Hepatic disease
 - 1 Ascending infectious cholangitis
 - 2 Toxic hepatitis
 - 3 Diffuse carcinomatosis
 - 4 Fatty degeneration or "fatty metamorphosis"
 - 5 Glycogenosis (von Gierke's disease)
 - C Pituitary hypofunction (anterior lobe)
 - 1 Destructive lesions (chromophobe tumors, cysts)
 - 2 Atrophy and degeneration (Simmonds disease)
 - 3 Thyroid hypofunction (? secondary to pituitary hypofunction)
 - D Adrenal hypofunction (cortex)
 - 1 Idiopathic cortical atrophy
 - 2 Destructive infectious granulomas
 - 3 Destructive neoplasms
 - E Central nervous system lesion (hypothalamus or brain stem, interference with nervous control of blood sugar)

- II Functional—no recognizable anatomic lesion but explicable on basis of unusual somatic function
 - A Hyperinsulinism (imbalance of the autonomic nervous system): hypoglycemic fatigue, nervous hypoglycemia; functional hypoglycemia; reactive hypoglycemia
 - B Alimentary hyperinsulinism (rapid intestinal absorption)
 - 1 After gastroenterostomy
 - 2 After gastric resection (partial or total)
 - C Renal glycosuria (severe degrees of low renal threshold for dextrose)
 - D Lactation
 - E Severe continuous muscular work
- III Miscellaneous
 - A Factitious (surreptitious insulin administration)
 - B Postoperative hypoglycemia
 - C Severe inanition
 - D Unknown

Islet Cell Tumors

These tumors are uncommon, although nearly 300 cases have been reported since 1929.

Incidence.—They are found in one out of every 800-1,000 autopsies. They occur in all age groups and are equally divided between males and females.

Pathology.—Islet cell tumors may be benign or malignant or so-called borderline malignant in type and may be functional or nonfunctional. The following classification of islet cell tumors of Lopez-Kruger and Dockerty describes the various types which have been encountered:

1. Adenomas of islet cells with hyperinsulinism and hypoglycemia
2. Adenomas of islet cells without hyperinsulinism
3. Carcinoma of islet cells with hyperinsulinism
4. Carcinoma of islet cells without hyperinsulinism
5. Borderline malignant islet cell tumors with or without hyperinsulinism

The adenomas most commonly occur in the tail of the pancreas, which contains the greatest concentration of normal islet cell tissue. Tumors, however, also occur in the body, the head, and uncinate process, and even in aberrant rests of pancreatic tissue.

The benign adenoma is small in size and measures in diameter from 0.5-1.5 cm. It is dusky, reddish-gray in color, and at operation

shows up as a darker area in the surrounding normal pancreatic tissue. Microscopically, the tumor cells resemble normal islet cell tissue. These tumors may be multiple, and two or more adenomas have been discovered in 12% of cases. Malignant tumors are distinguished by an invasive pancreatic mass with regional and liver metastases. *Occasionally, hyperinsulinism is caused by a diffuse hyperplasia of the islet cell tissue*

Clinical Features.—The symptoms are due to a low level of carbohydrate supply to the brain which results in decreased cerebral metabolism. The brain is unable to store glucose in any appreciable amount. As in anoxia, the higher brain centers show the most sensitivity to the hypoglycemia. Electroencephalographic studies will show typical tracings due to hypoglycemia which are almost identical to those of anoxemia. Pathologic changes in the brain, in the nature of a degeneration of nerve cells, have been found at autopsy.

The symptoms vary greatly from patient to patient and tend to progress in their severity. They occur after a period of fasting or after a period of exertion plus fasting, which tends to depress the blood-sugar level. In the early stages of the disease, the symptoms are subjective.

Moersch and Kernohan have divided the symptom complex into three stages. In the *first stage*, the patient is mentally and physically retarded with drowsiness and lassitude. There may be slight confusion, restlessness, and irritability. The *second stage* is one of excitement, as shown by a marked confusion, marked restlessness, agitation, pallor, and profuse sweating. The *third stage* is that of coma, with or without convulsions, which may be tonic or clonic, a positive Babinski may be present, and nystagmus and motor paralysis occasionally occur. The level of blood sugar that produces these symptoms varies from patient to patient. If the condition continues for a long time, permanent degenerative changes occur in the brain; the symptoms then become permanent and cannot be relieved by the administration of glucose. Many patients have been diagnosed as epileptics and have been treated as such for years. Hyperinsulinism may lead to obesity as the patient learns that

he can avoid or minimize an attack by frequent feedings of high carbohydrate foods.

Diagnosis.—Hyperinsulinism must be carefully distinguished from other causes of hypoglycemia, the chief of these being functional hypoglycemia. In *hyperinsulinism* the fasting blood sugar is low and to be diagnostic should be below 50 mg. per 100 ml. The symptoms occur after a period of starvation or exertion and increase in severity as time goes on. The 5-hour glucose tolerance curve is extremely variable and is of little value in the diagnosis of hyperinsulinism. This is possibly due to the fact that the hyperfunctioning islet cell tissue behaves in a similar fashion to a pheochromocytoma, having periods of marked activity with intervening periods of quiescence. Only after the liver store of glycogen is depleted does the typical hypoglycemic curve result.

In *functional hypoglycemia*, the patient is usually emotionally unstable. A long-standing anxiety tension state will very often produce a hypoglycemia of the functional type. In the majority of cases, the symptoms are subjective, but occasionally the objective signs of pallor, sweating, and agitation may occur. The attacks do not occur after fasting but usually occur 2-3 hours after a meal, particularly if it has been of a high carbohydrate content.

In contradistinction to the patient with hyperinsulinism, the patient with functional hypoglycemia has a *normal fasting blood sugar*. The 5-hour glucose tolerance curve shows a sharp rise in the first 2-3 hours which is followed by a drop to hypoglycemic levels. (Glycosuria may occur during the phase of the elevated blood sugar.) Before attempting a 5-hour glucose tolerance curve, it is essential that the patient be placed on a high carbohydrate intake for two days prior to the test. The symptoms of functional hypoglycemia tend to remain the same over a period of years. As in the case of hyperinsulinism, exercise will aggravate the symptoms. The patient with functional hypoglycemia often gives a history of poor dietary habits and frequently subsists on a small carbohydrate breakfast and lunch, leaving his major meal until evening. The symptoms can be prevented by an adequate balanced diet consisting of liberal amounts of protein.

The best means of differentiating the two conditions is a prolonged period of fasting which, at times, may need to be maintained for 48 or even 72 hours. If an organic hyperinsulinism is present the blood sugar will, in nearly every case, fall below 50 mg./100 ml.

Hypoglycemia due to hepatic disease can be distinguished by signs of severe liver disease and by liver function tests. The hypoglycemia of anterior pituitary or adrenal insufficiency can also be distinguished by a careful observation of the well-known stigmata of these diseases. Hypoglycemia is frequently observed after a subtotal gastrectomy and the 5-hour glucose tolerance curve shows hypoglycemic blood sugar levels, which may be accompanied by symptoms. This hypoglycemia is partly responsible for the late postprandial phase of postgastrectomy syndrome.

Treatment.—Once the diagnosis is certain, the tumor should be removed as soon as possible, principally because such a tumor may be malignant. Early operation is also important to prevent permanent brain damage and increasing obesity. Before abdominal exploration is attempted, the surgeon must be sure that the disease is of an organic nature and not a functional hypoglycemia.

The three points defined by Whipple should be observed before any operation is contemplated. (1) the attacks must come on after a period of fasting or after exertion, (2) the blood sugar level must fall below 50 mg./100 ml., (3) the symptoms must be relieved by glucose, given either orally or intravenously.

Preoperative Treatment.—The patient should receive a high caloric, high protein diet for several days prior to the operation. Intravenous glucose may be necessary the night prior to the operation and on the morning of the operation. Any electrolyte disturbances should be corrected by the appropriate measures.

Operation.—It is usually best to approach the pancreas through the gastrocolic omentum which is removed from the stomach, care being taken to conserve the gastroeiploic vessels. However, in the case of a low-lying stomach, the approach may be more simply made through the gastrohepatic omentum. The anterior surface of the gland should be carefully visualized and palpated, starting with the tail and working toward the body and

head. If no tumor can be visualized or palpated, the body and tail of the pancreas should be mobilized to allow palpation of the gland between the thumb and fingers. Frequently, an invisible tumor may be discovered by this means. If nothing is found in the tail or body, the head of the pancreas must then be mobilized by reflecting the duodenum and pancreas toward the midline to allow bidigital palpation. A careful search for multiple tumors must always be made. It is important never to neglect an examination of the common sites of aberrant pancreatic tissue, for occasionally tumors can be discovered in such areas. This is particularly so if nothing is found in the pancreas itself.

Once located, the benign tumor usually can be easily enucleated. However, if it occurs in the tail of the pancreas, it is sometimes preferable to resect a portion of the pancreas with the tumor. If any suspicion of malignancy exists, a wide area of excision should be carried out. If metastatic carcinoma is present, a radical pancreatectomy together with resection of all metastatic tissue should be performed.

If no adenoma can be found either in the pancreas or in aberrant tissue, a resection of the tail and body of the pancreas should be performed in the hope of removing an occult tumor or a sufficient amount of hyperplastic islet cell tissue to effect a cure. If after such a procedure the symptoms still persist, a total pancreatectomy may be performed.

Supportive therapy during the operation should consist of whole blood and physiologic saline. Glucose-containing solutions should be carefully excluded until the postoperative hyperglycemic response is evaluated.

Postoperative Treatment.—Following the removal of a functioning tumor, a period of hyperglycemia develops which is usually transient in nature but, at times, is sufficiently prolonged and severe to require insulin control. Once the residual normal islet cell tissue, which may be atrophic because of its functional depression by the hyperfunctioning tumor, regains its insulin secretory power, the hyperglycemia disappears. Only rarely does a permanent diabetic state ensue.

Results of Treatment.—The results of removal of benign and borderline malignant

SURGERY OF THE ENDOCRINE GLANDS

adenomas are good if the operation is carried out before permanent brain damage has developed. However, resection of malignant tumors with metastatic spread gives little, if any, hope of a permanent cure.

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Film References

Title	Running Time	Sound or Silent	Color	Procureable From
<i>Surgical Management of Primary Hyperthyroidism (Illustrates the author's technique of thyroidectomy for hyperthyroidism) (1950) (By Frank H. Lahey, M.D., Boston)</i>	21 min	Sound	Color	American Cyanamid Co Surgical Products Division Danbury, Conn.
<i>Primary Hyperparathyroidism Due to Parathyroid Adenoma (Diagnosis and surgical treatment) (1951) (By Joel W. Baker, M.D., and Randolph P. Pillow, M.D., Seattle)</i>	32 min	Silent	Color	American Cyanamid Co Surgical Products Division Danbury, Conn.
<i>Carcinoma of the Thyroid, Thyroidectomy, and Neck Dissection (Shows technique of total thyroidectomy and radical neck dissection for carcinoma of the thyroid with extensive metastatic involvement of lymph nodes) (1951) (By R. Lee Clark, M.D., Houston)</i>	32 min	Sound	Color	American Cyanamid Co Surgical Products Division Danbury, Conn.
<i>Resection of the Pancreas for Adenoma of the Islets of Langerhans (By Charles B. Puestow, M.D., F.A.C.S., Chicago)</i>	15 min.			American Cyanamid Co Surgical Products Division Danbury, Conn.

Chapter 14

The Breast

Ray Lawson, MD.

Breasts are characteristic of the mammalian class of animals. Their function is to secrete milk for the nutrition of the young; but in addition, as a secondary female sexual development, they serve, in the human at least, as an attracting stimulus to the male. They are composed of epithelial elements, which are surrounded and supported by fibrous and fatty connective tissue—the stroma. In the active state the epithelial elements form compound tubulo-alveolar exocrine glands. In the resting phase no alveoli are present, they do not secrete, and thus they cannot be considered true glands. No true specific internal secretions have so far been identified.

EMBRYOLOGY

It is remarkable that while the breasts are the last glands to function in the adult, they are the first of the epidermal glands to appear in the fetus. The glandular part of the breast is derived from ectoderm, which grows inward in strandlike fashion. The supporting stroma is evolved from mesodermal subcutaneous tissue. As early as the sixth week, an ectodermal thickening or outgrowth is apparent on either side of the trunk. In the second stage this *milk line* atrophies except in the pectoral region. This subsequently grows inward, branching radially into 15-20 solid cords. These ectodermal cords eventually become the milk ducts and each represents one unit of breast tissue. They branch and subdivide and

at 7 months begin to canalize. At birth the depression from the epithelial ingrowth becomes a prominence forming the nipple.

ANATOMY

The size, shape, and structure of the breasts are extremely variable. The breast is situated, except for the prolongation of the tail, in the superficial fascia on the front of the thorax. From the sternum it extends to the midaxillary line and vertically from the 2nd to the 6th ribs. There is no capsule and the 15-20 radiating lobes are firmly imbedded in the superficial fascia by strands of fibrous tissue. These form the framework of the organ and are known as the ligaments of Cooper. They ramify throughout the breast substance and connect the skin to the deep fascia. These ligaments are very important from the standpoint of the diagnosis of malignant disease because when breast tissue becomes edematous as a result of lymphatic permeation and obstruction, these inelastic bands cause fixation and dimpling of the skin.

Each lobe of the breast is an independent gland composed of lobules. A lobule is made up of branching lobular ducts, the smallest of which are termed ductules. The ultimate terminations of the ductules during lactation are the secreting alveoli. The stroma immediately surrounding the smallest ducts and acini is termed the periductal or intralobular connective tissue and is a relatively cellular type

of tissue subject to constant variations. The stroma associated with the nipple and lactiferous and larger ducts is much more dense since it is not subject to great change. This is termed the intralobar connective tissue. The fibrous connective tissue between the lobules is designated interlobular, while that separating the lobes is termed interlobar.

Relations.—The gland lies on the pectoral fascia. Posterior to the upper half is the pectoralis major muscle, while inferiorly are the serratus anterior, the external abdominal oblique, and upper part of the rectus sheath. The axillary tail (of Spence) is particularly variable in size and extent. It passes upward and laterally at the edge of the pectoralis major and on through the foramen of Langer of the deep fascia into the axilla.

Blood Supply.—The anterior perforating branches of the internal mammary artery, particularly the 2nd, 3rd, and 4th, supply the medial portion. The lateral aspect is supplied by the lateral thoracic from the second part of the axillary artery as well as by the lateral perforating branches of the corresponding intercostals. A point of surgical importance is the fact that the main arterial inflow is through the upper half of the breast. The venous drainage in general follows the course of the arteries. Enlarged bluish veins characteristic of pregnancy and lactation are often seen running independently in the subcutaneous tissues.

Lymphatics.—Detailed knowledge of lymphatic drainage of the breast is important, because the aim of cancer treatment is the removal not only of the breast itself but also of the associated lymphatic vessels and glands *en bloc*.

The lymphatic system of the breast originates in the interlobular spaces and on the walls of the lactiferous ducts and terminates in nodes in the axilla, thorax, and cervical region.

Superficial.—The plexus of small lymph vessels, which surround the acini, passes via the periductal lymphatics to the subareolar plexus. From here, efferent channels lead principally to anterior pectoral nodes along the lateral thoracic vein, at the edge of the pectoralis major muscle.

Deep.—Some vessels leaving the interlobular spaces and ducts pass posteriorly to the deep fascial plexus, in relation to the pectoral and serratus muscles. From the deep plexus, large lymphatic channels run to the regional lymph glands. The efferent vessels from fascial plexus can be traced in groups circularly like the spokes of a wheel.

1. Some vessels pass directly above, after perforating the pectoralis major muscle, and terminate in Rotter's and other interpectoral glands, which lie in relation to the pectoralis minor muscle. Efferents from here proceed to surgically inaccessible subclavian nodes.

2. From the medial aspect of the deep plexus, vessels pass inward, alongside the perforating arteries and enter the anterior mediastinal glands, particularly those along each side of the internal mammary arteries.

3. Some vessels from the deep plexus cross the midline and anastomose with the corresponding plexus of the other breast.

4. From the inferomedial part of the plexus vessels travel through the rectus fascia to the ligamentum teres and thence to the liver and abdominal lymph nodes.

5. On the lateral aspect, there are channels which pass inward with the intercostal blood vessels to terminate in a series of nodes which lie inside the lateral and posterior chest wall and also nodes in proximity to the aorta. This route may be responsible for some of the vertebral metastases so frequently seen in breast cancer.

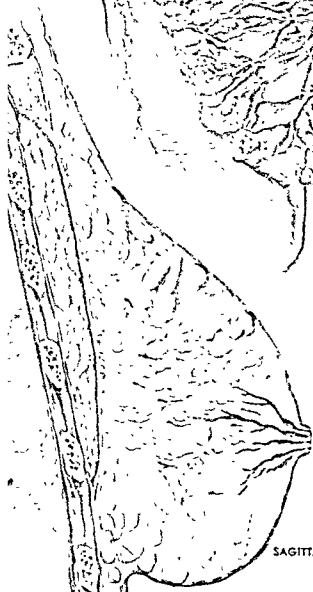
6. The most important lymphatic drainage of the deep plexus is upward and laterally to the lateral pectoral nodes and thence to the axillary groups. Direct connections exist, however, from the breast to the axilla.

The lymph nodes of the axilla lie in relation to the axillary vein and its immediate branches, inside the deep fascia.

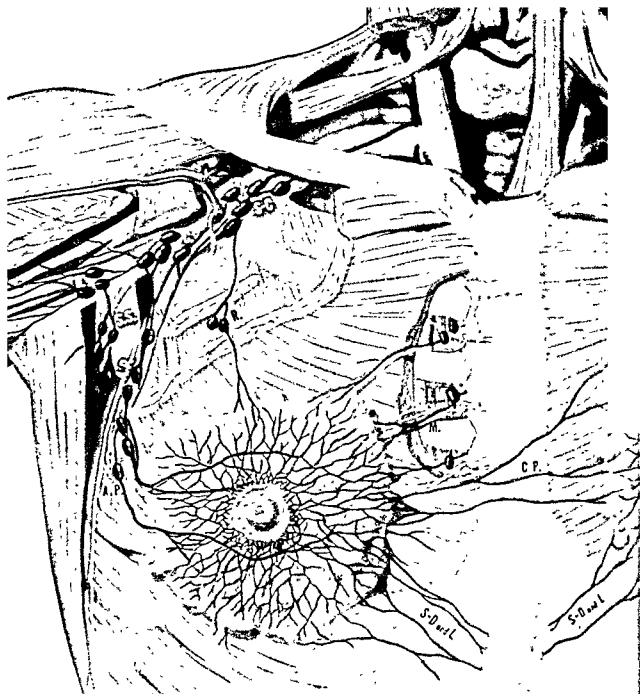
Both axillary and internal mammary nodes drain primarily into the base of the neck where the lymphatic vessels enter the confluence of the large veins. The internal mammary nodes can also drain by retrograde manner into the dome of the liver. It has been traditional to accept the premise that breast cancer is disseminated primarily by lymphatics and later by hematogenous routes after the



ANTERIOR DISSECTION



SAGITTAL SECTION



- A.P. — Anterior Pectoral Nodes
- C. — Central Axillary Nodes
- B. — Brachial Nodes
- S.C. — Subclavian Nodes
- R. — Rotter's Nodes
- I.M. — Internal Mammary Nodes
- M. — Pathway to Mediastinal Nodes
- C.P. — Cross Mammary Pathway to Opposite Breast and Axilla
- D and L — Pathway to Subdiaphragmatic Nodes and Liver
- S.S. — Subscapular Nodes

lymphatic vessels empty into the large veins. However, it is quite possible that a very large proportion of breast cancers spread primarily and directly by purely hematogenous routes, without lymph node invasion. Metastatic cells can easily pass through the lungs, so that all metastases can be explained by this route.

Nerve Supply.—The skin covering the breast is richly supplied with branches of the 4th, 5th, and 6th intercostal nerves. The glandular part receives autonomic fibers which run with the 2nd to 6th intercostal nerves. Smooth muscle fibers which erect the nipple are supplied by these autonomic nerves.

HISTOLOGY

The alveoli of the female breast are lined by cuboidal or columnar secreting epithelial cells which rest on the *membrana propria* in orderly fashion. During lactation, the mechanism of milk secretion involves constant exfoliation of these cells. Columnar epithelium lines the ducts as far as the lactiferous sinuses, and then it becomes squamous in character. In the ducts a reserve outer layer of low cuboidal cells supports the columnar epithelium. The stroma consists of fat and fibrous tissue and is contiguous with the surrounding subcutaneous structures, since the breast has no limiting capsule. Around the acini are so-called accommodative spaces consisting of loosely arranged connective tissue. Because the bulk of the breast is fat, the actual size bears no relation to the quantity of milk-producing structures. The normal male breast does not significantly alter throughout life but consists merely of a few small duct rudiments posterior to the vestigial nipple.

PHYSIOLOGY

Most breast derangements are in some way related to the varying cellular characteristics that result from constantly changing hormonal stimulations. A more detailed knowledge of breast physiology than space permits here should be acquired. With the onset of puberty, the female breast becomes subject to alternate hyperplasia and involution. The hyperplasia is characterized by a material increase in both the duct structures and stroma. In the cycles of

this process, which are coordinated with the menstrual periods, the phases of hyperplasia exceed the following involution phases until such time as the breast assumes adult size. When pregnancy occurs there is a tremendous wave of proliferative activity and many alveoli develop. In the later stages of lactation, regressive changes occur in which these racemose glands atrophy, and the acini disappear. Finally with the menopause, there is the onset of a gradual reduction in both parenchyma and stroma, until eventually nothing remains but a few dilated ducts and a small amount of fatty fibrous connective tissue.

EXAMINATION OF THE BREASTS

The student should adopt a routine method of examining all breasts, and the following outline is suggested.

The patient is undraped to the umbilicus and seated on the edge of the table or bed with the arms at the sides. The lighting must be adequate and free from distorting shadows. The breasts are first critically inspected and compared for size, contour, and symmetry. The arms are raised and the examination is repeated. Next, the nipples are examined for eczema and retraction. The examiner, standing behind the patient, palpates for supraclavicular glands, a sign of secondary spread.

Then with the pectoral muscles relaxed, by placing the patient's arm on one of his own, the axilla is gently palpated in all directions with the fingertips. Keeping the fingers here, the arm is swung back and relaxed as the axillary contents are palpated against the rib cage. The location, size, consistency, and fixity of any enlarged lymph nodes are noted. The fact that the result of a clinical search for involved nodes is unreliable is no excuse for neglecting this procedure, as positive findings are extremely significant.

Palpation of breasts should always be done gently and kept to a minimum for fear of spreading cancer cells. Only the flexor side of the extended fingertips should be used. The breasts are palpated with the patient sitting up and then again with the patient in a supine position with the side of the chest raised with a pillow. The whole of the breast structure

is investigated gently with the extended fingers. The most difficult area to palpate with accuracy is directly beneath the nipple.

The skin of the breasts is now examined for areas of retraction or fixation. Edema of the skin or "peau d'orange" from lymphatic blockage is a late sign and usually denotes an inoperable lesion. By gently pinching and lifting the skin, retraction or fixation of areas

iliac crest with the hand, or flexing the body on the hips with the neck extended.

Attention is again directed to the nipple, to test for the presence of discharge. This is done by stroking the breast with the index finger centrally over the skin, toward the nipple, in a circumferential manner. A milky discharge in a nonlactating woman usually does not have serious significance. About 5% of

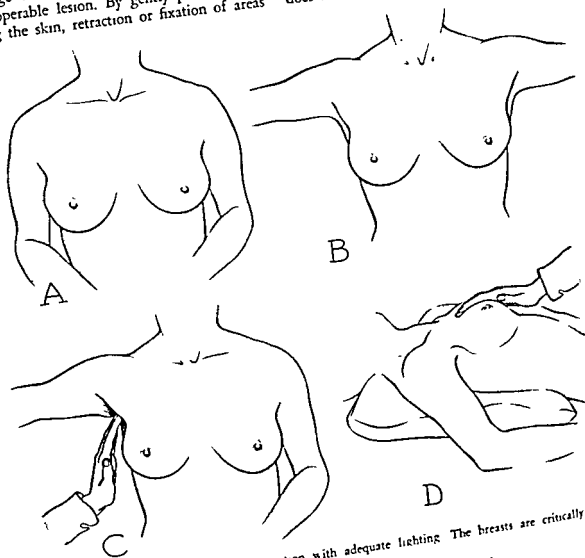


Fig. 214—A, Preliminary inspection with adequate lighting. The breasts are critically inspected for contour, symmetry, and changes in skin.
B, Examination repeated with arms raised to accentuate latent deformities.
C, Careful palpation of the axilla, with relaxed pectoral muscles.
D, Gentle palpation of the substance of the breast with the finger tips. The thorax is elevated on a pillow.

is often elicited—a most important sign in cancer. Retraction is also elicited in other ways, such as raising the arms above the head, pushing the whole breast in each direction, compressing the breasts from both sides, contracting the pectoral muscle by pushing on the

of all breasts which have a bloody discharge will harbor carcinoma. But almost invariably in these cases a mass is palpable close to the areola, and the clinical diagnosis is evident. About 90% of nipples discharging blood will be associated with either papillomatosis or

ulcerating granulations, secondary to chronic duct dilatation or stasis.

Local x-ray examinations are of little practical help in examining the breasts. Rapid pathologic examination following punch or needle biopsy of lumps is being more frequently done as a routine preoperative measure in well-equipped clinics. This procedure is useful when applied to sites of secondary lymphatic spread, such as supraclavicular or cervical lymph nodes, to determine the state of operability when the diagnosis of breast cancer has been made. Papanicolaou smears of nipple discharge, properly interpreted, are helpful in detecting intraductal papillomatosis, but so far this method has been of no real assistance in the diagnosis of breast cancer. Aspiration of large cysts may relieve the discomfort from increased tension and effect complete disappearance of such lumps. When using this method, one must be aware of the danger of overlooking malignant disease and have the cells examined by a competent cytologist if the fluid is bloody or cloudy. The risk of disseminating cancer cells by needle biopsy is not a practical hazard, since follow-up surveys where needle biopsy has been done on cancer patients do not show an increased spread of disease.

Applications of various technologic measuring techniques in attempts to evaluate biologic changes of a dynamic nature rather than static anatomic differences are currently undergoing evaluation by several methods. Echographs made by measuring the reflection of sound waves are being explored but so far are of laboratory interest only. The study of altered heat patterns radiated by the skin in various wave lengths and detected by electronic cameras will likely contribute helpful diagnostic information.

In describing a lump in the breast, the following points should be noted: the size, shape, position, consistency, mobility, overlying skin dimpling, skin edema, nipple retraction, and nipple discharge. Most breast cancers are not clinically palpable until 1 cm in diameter. It is a sad fact that at this stage less than half the cases have strictly localized disease.

Every patient should receive a thorough radiologic examination of the chest, spine,

and pelvis before radical mastectomy is contemplated, so that distant skeletal metastases may, in some measure, be detected.

Enlarged lymph glands in the neck or supraclavicular region should be biopsied by needle technique if involvement is suspected.

CONGENITAL ANOMALIES

Amastia

Complete absence of a breast is rare and is usually found in males. It may be associated with the absence of the pectoral muscles or other congenital aberrations. An interesting hereditary pattern may sometimes be deciphered when this abnormality appears.

Athelia and Polythelia

Athelia is a rare condition denoting absence of the nipple, whereas polythelia, which is more common, refers to the presence of accessory nipples without breast tissue. Accessory breasts may be found anywhere along the mammalian *milk line* which extends from the axilla to the groin. Tumors can arise in these accumulations of breast tissue. Thus a primary carcinoma is occasionally seen in the axilla.

Gynecomastia

Gynecomastia is a true hypertrophy of mammary tissue in the male. The cause is often unknown, although trauma may be a factor, as well as hormonal disturbances. It may appear in men receiving estrogenic treatment for carcinoma of the prostate. It is also seen associated with hormone-producing tumors such as adrenocortical carcinoma, embryonal carcinoma, and especially chorionepithelioma. Testicular biopsies and blood estrogen determinations are occasionally of diagnostic significance in these cases. In the male, excision of such breast tissue may be warranted to correct discomfort or to alleviate embarrassment. Painful enlargements of the breasts are not infrequently seen in males at puberty. Such lesions should be treated conservatively, since they are due to a temporary hormone disturbance. The majority of patients with gynecomastia do not present any gross hormonal change. Impaired metabolism of estrogens by the liver and the vitamin B complex dietary

deficiency sometimes seen in liver disease can lead to gynecomastia. The important problem is to differentiate it from carcinoma, since about 15% of cancers occur in males. The effect of hormonal stimulation through the placenta is sometimes seen in the infant at birth. This is designated *mastitis neonatorum*. The discharge known as *witch's milk* has given rise in the past to superstitious phantasies, as the involved baby is usually the first-born and beyond term.

Virginal Hypertrophy (Macromastia)

This type of diffuse enlargement is usually unilateral. The cause is unknown. An abnormal breast of this nature seldom functions efficiently and is particularly prone to structural disease. Operative treatment may be indicated to alleviate embarrassment in extreme degrees of enlargement. This consists of intricate plastic procedures to reduce the size or simple mastectomy.

INFLAMMATORY LESIONS

Acute Infections

Acute breast infections may occur at any period of life but are generally associated with lactation. With modern methods of prevention and management, infections have relinquished the important and frequent place they formerly held in the realm of breast surgery.

The causative organisms should always be identified and tested for proper antibiotic therapy. Staphylococci are common offenders. The bacteria invade the breast through a crack in the nipple and pass inward by way of the ducts or the periductal lymphatics. A diffuse cellulitis is first established, but continuation of the infective process leads to localization and suppuration.

Three varieties of abscess formation are formally described, depending on location.

1. Premammary abscess may be subareolar or subcutaneous in position. Because of the location, there is no difficulty in the diagnosis. The classical signs of inflammation are present and the localized pus may be fluctuant. The pus should be aspirated and the cavity filled with the antibiotic of choice or simple surgical drainage performed. Premammary abscesses have a tendency to become chronic, but most

chronic recurring abscesses which point at the areolar margin are secondary to duct dilatation, stasis, and chemical mastitis. The irritating material filling the ducts ruptures through the ducts and causes an abscess which usually erodes through the areolar margin. Many of these are found in cases of congenital inverted nipples. In the chronic type, therapy consists of excision of the involved ducts and inflammatory mass, with plastic repair of the remaining areola.

2. Intramammary abscess is the usual type of acute breast infection and is located well within the substance of the breast tissue. It practically always results from lactational mastitis. The cavity is diffusely located and may involve one or more lobes. There is marked systemic reaction with fever and malaise. The localized inflammatory reaction is associated with induration of the breast. If the abscess is well developed, the treatment consists of operation under general anesthesia. A large radial incision into the abscess cavity is made and a finger is inserted to thoroughly break down the honeycomb type localizations to ensure complete drainage. The cavity is then packed with gauze, and the incision is left wide open. Dependent drainage with soft rubber tubes through a separate stab wound is often advisable.

3. Retromammary abscess is a localized infection in the areolar tissue between the chest wall and the breast. It is extremely rare and may be acute or chronic. The acute cases are typified by a much more intense systemic reaction with less apparent local signs. Such infections are usually secondary to subjacent lesions, such as empyema, tuberculous osteomyelitis of the ribs, or infected hematomas. The typical abscess eventually points inferolaterally, pushing the breast forward. Treatment consists of surgical drainage in addition to correction of the underlying lesion. A curved incision is made along the inferolateral aspect of the breast. The abscess is located with a pair of artery forceps and digitally explored. Gauze is then loosely packed into the cavity after the causative lesion has been suitably treated.

The most important aspect in the treatment of all puerperal breast infections is prophylaxis by careful hygiene. This includes clean-

ness of the nipple, protection from trauma, and prevention of stasis by expression of the preliminary secretion. The use of antibiotics at the time of childbirth has been dramatically effective in reducing the incidence of these infections. If an acute infection occurs, the baby should be weaned, and the breast "dried up." The application of a firm protective breast binder and oral therapy with 5 mg. of stilbestrol 3 times daily will adequately serve this purpose.

Chronic Infections

Tuberculosis, while rare, is responsible for the majority of chronic infections. It is usually secondary and occurs in women having open pulmonary lesions. The organism may gain entrance through a cracked nipple. A large tender mass of variable consistency is present, and it may be absolutely indistinguishable from carcinoma. Certainly in the early stages, the clinical diagnosis is impossible. The axillary glands are characteristically enlarged and, as the lesion advances, multiple draining sinuses appear. Treatment consists of wide *en bloc* excision of the area, including the overlying skin of the breast and the underlying costal cartilages, if involved. This can usually be done extrapleurally and without breaking into the abscess cavity. Closure is obtained by undermining the adjoining skin and subcutaneous fat and, if necessary, by rotating a skin flap to cover the defect. The general treatment of tuberculosis must be enforced, which includes rest, diet, dihydrostreptomycin, and PAS.

Syphilis of the breast is now a curiosity. As a primary lesion it appears as a chancre in the region of the nipple. It is single, unilateral, painless, and associated with rapid enlargement of the axillary lymph nodes. Diagnosis is made by the typical appearance and by dark-field examination of the exudate. In doubtful cases biopsy is necessary. The treatment is that of syphilis in general.

Actinomycosis is an extremely rare infection and is usually secondary to involvement of the lungs, with extension through the thoracic wall. It is characterized by chronically indurated areas and weeping granulation tissue which discharges sulfur granules through secondarily infected sinuses. Treatment is surgical

excision of the lesions, massive doses of iodides, penicillin, and other forms of chemotherapy as described in Chapter 3.

Eczema of the nipple usually clears up if mechanical irritation is avoided and the part kept scrupulously clean and dry. Cortisone preparations and tar ointments have been found useful. Should the eczema persist, Paget's disease should be suspected, and a wedge biopsy may be indicated to rule out this condition.

Other types of chronic inflammation such as sarcoid, blastomycosis, filariasis, sporotrichosis, and leprosy have been reported.

CHRONIC MASTITIS (CYSTIC DISEASE)

The female breast is likely to be altered in structure in the first place, by fibrous involution with loss of parenchyma and, in the second place, by varying degrees of epithelial hyperplasia. These two types of changes often coexist in varying degrees and, so far as is known, are due to abnormal hormonal stimulations. When such structural changes are evident, we call the lesion *cystic disease*, or *fibroadenosis*, for lack of a better term. Despite the fact that the varied terminology so commonly encountered in reading descriptions of the different phases or degrees of these types of breast changes is extremely confusing, it is possible to orient and classify the progression of the abnormalities in structure in a more or less orderly fashion. Whether or not it is usual for cancer to evolve from the associated series of epithelial changes is a much debated point. Until this is settled the problem of properly treating the lesions called cystic disease remains difficult.

The fundamental changes consist of an increase in fibrous connective tissue with loss of parenchyma (epithelial elements). Obstruction of the lactiferous ducts by inspissated material, chemical irritation, or external pressure from fibrosis may result in the formation of cysts. Such cysts may reach considerable size and contain serous fluid under tension. According to a once popular pathologic classification, it was claimed that these cysts may be lined (in chronologic order) by compressed atrophic epithelium, a denuded fibrous mem-

brane, or by regenerated and hyperplastic epithelium. The lining epithelial cells when hyperplastic may form several layers inside the cysts or ducts, traverse the lumina, or fill them solidly. Further progression of epithelial changes has been described, in which the cells are considered malignant and the so-called *intraductal carcinoma* or *carcinoma-in-situ* is recognized. Since the diagnosis at this stage depends on cytologic changes alone, the borderline of malignancy cannot always be

tion of estrogenic substances. Warren's studies showed that women afflicted with cystic disease before the menopause were 11.2 times as likely to develop cancer as those in the general population. He found that after the menopause this danger was not appreciably increased. On the other hand, Bloodgood's school and other authorities, the weight of whose opinion cannot be overlooked, claim that the relationship between this condition and cancer is purely coincidental. Furthermore,

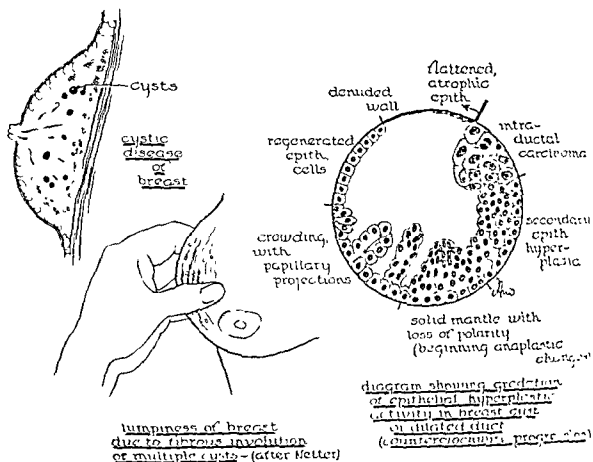


Fig 215—Chronic cystic disease of the breast

sharply drawn. However, once these cells break the law, i.e., the growth extends beyond its normal boundaries into periductal tissue and lymphatics, the diagnosis of carcinoma is readily made on a topographic basis.

There is laboratory evidence to support the interrelation of the epithelial activity in cystic disease and the development of malignancy. Experimentally, in male mice, a similar series of epithelial changes, progressing to malignancy, has been reproduced by the administra-

our own and many other follow-up studies on women who have had breast biopsies which showed benign hyperplastic epithelial changes do not show any impressive increased incidence of malignancy.

Diagnosis.—This is a very common disease of apprehensive and tense personality types. More and more cases are being encountered because an increasing number of women with cancerphobia are seeking medical counsel. Cystic disease is characterized by nodular or

thickened areas in the breast which become increasingly painful before menstruation. The findings on examination are variable. There may be no cysts, or the cysts may be so small as to be imperceptible, or they may be as large as an egg and may be single or multiple. Large cysts often have a bluish translucency on surgical exposure and have been termed by Bloodgood, blue-domed cysts. Most areas of nodularity consist of fibrous involution and are found in the upper outer quadrant. The pull of gravity due to the upright position has been suggested as a cause for this. Benign epitheliosis *per se* is not a palpable lesion and can only be diagnosed microscopically. Unfortunately there is little clinical and pathologic correlation of hyperplastic epithelial states, and a part of the breast severely involved may feel more normal than an area only moderately affected. The actual incidence of this fluctuating disturbance in the adult female population is impossible to ascertain but appears to be extremely high.

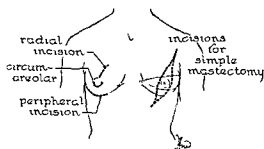


Fig. 216—Common incisions used in breast surgery

Treatment.—Local excision of the diseased area is far from satisfactory. As the lesion is more diffuse than clinically apparent and medical treatment inadequate, the only cure is simple mastectomy. As a general measure this is obviously far too radical but may be warranted in cases presenting severe degrees of this disorder. The pain and tenderness may frequently be relieved by 50 mg progesterone vaginal suppositories inserted every second night for 5 doses. Many clinicians use methyltestosterone 10 mg daily by mouth, especially during the premenstrual phase, and one capsule of vitamin B complex 3 times daily. Additional therapy in the form of fluid and salt

restriction with the use of nonmercurial diuretics is sometimes helpful. Judicious use of ataractics may be beneficial. Hot compresses for severe pain, adequate support, and, above all, reassurance round out the treatment which, it must be admitted, is most unsatisfactory. While the somewhat increased incidence of cancer with cystic disease is generally recognized, clinical statistics do not support a radical approach just for the sake of the surgeon's and patient's "ease of mind." The patient's age, marital status, and familial history of cancer, as well as personal attitude, must be given individual consideration. Any "dominant" lumps or suspicious areas must *always* be excised and microscopically examined. It may be argued that biopsy may miss an important area. There is no answer to this argument. If the biopsy shows atypical, benign epithelial hyperplasia, some surgeons unhesitatingly perform prophylactic simple mastectomy. The clinical differentiation between benign and early-stage malignant lesions is frequently impossible, and experienced surgeons are constantly distressed by the number of patients with inoperable cancer who have been treated conservatively for cystic disease until the real diagnosis has become obvious. Every breast lump *must* be excised, but the real difficulty lies in the clinical distinction between a lump and an area of nodularity. It is impractical and impossible to biopsy all areas of thickening and nodularity in every breast, as the disease is so characterized by relapses and remissions that few breasts would escape the surgeon's knife at some time or other. Cystic disease has a remarkable tendency to resolve during the first three months of pregnancy. One should be extremely careful to search for a co-existing carcinoma if epithelial hyperplasia is detected in a biopsy specimen. A close, conscientious six-monthly follow-up of these patients must be undertaken in order not to miss more serious lesions.

BENIGN TUMORS OF THE BREAST

Nonindigenous benign tumors such as lipomas, chondromas, leiomyomas, granular cell myoblastomas, dermoids, and sebaceous cysts may occur.

Fibroadenomas make up about 15% of all breast tumors. This type is most often seen as a firm, round, mobile, encapsulated, painless mass, about 1/2-2 cm. in diameter, in women under the age of 25 who have not yet nursed. Such tumors may be single or multiple and are often associated with either localized or generalized cystic disease. They seem to arise from an overgrowth of the intra-lobular fibrous connective tissue. Treatment is local excision of the tumor, preferably under general anesthesia. The incision should be made radially from the nipple or along the inferior fold of the breast. In rare cases, cancer can be traced to proliferation of the involved epithelial cells and occasionally sarcomatous degeneration occurs in a fibroadenoma.

Duct papilloma generally grows slowly within a cystic dilation of one of the main ducts near the nipple and is characterized by a nipple discharge which is often bloody. They are usually nonpalpable, but the dilated duct containing them may occasionally be felt to be firm and round. If the tumor can be accurately delineated (by transillumination and digital pressure producing nipple discharge over a specific area), local excision is satisfactory as it is not prone to malignant change.

Duct papillomatosis is in essence an advanced type of cystic disease, with multicentric epithelial proliferations proceeding to the formation of papillomas. This lesion is characterized by nipple discharge which is often bloody. No tumor masses can be palpated. The treatment is simple mastectomy or preferably local excision of the main duct system through an 180 degree circumareolar approach.

FAT NECROSIS

Fat necrosis, although it is uncommon, should always be considered in the diagnosis of a breast lump. Clinically, the lump cannot be differentiated from carcinoma. Occasionally following a blow, the fat liberated from ruptured cells is saponified by circulating enzymes. The lipogranulomatous reaction associated with proliferation of fibrous tissue results in a firm, hard, generally painless lump of indefinite outline. Many cases appear to be unrelated to trauma. The condition is sometimes seen in

the dependent part of pendulous breasts and in some cases seems to be related to nonpurpurative panniculitis. The treatment is local excision of the mass for diagnostic purposes.

GALACTOCELE

This rare lesion forms a smooth, tense, rounded swelling in the breast during the second or third month of lactation. It is the result of blockage of a milk duct by scarring or local inflammation. Its recognition is important insofar as it may be confused with malignant disease. The treatment is repeated aspiration and support of the breast by a compression bandage. Occasionally it is advisable to arrest secretory activity of the gland by hormone therapy.

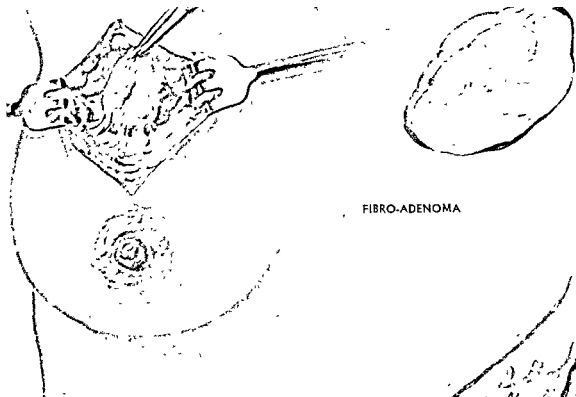
PLASMA CELL MASTITIS

Plasma cell mastitis is a rare lesion of the aging postlactational breast. It usually occurs in women 30-40 years of age. The exact etiology is unknown, but the condition is associated with advanced degrees of mammary duct ectasia. The inflammatory process probably represents a foreign body reaction to some chemical product of mammary secretion and is characterized by intense fibroplasia and the presence of large numbers of plasma cells. The onset is acute, with mild pain, tenderness, chills, and fever. A painless mass, 2-5 cm in diameter and resembling a malignant tumor, is usually palpable when the acute phase subsides. Biopsy is necessary to distinguish it from carcinoma. The condition may be a variant of chronic premammary abscess, as previously described.

MALIGNANT DISEASE

CARCINOMA

The understanding and control of cancer is the most urgent problem of medicine today. The collection of accurate data on such a complex disease is much more difficult than would at first seem apparent. Our information dates back to the Egyptian descriptions in 3000 B.C. Cancer was extensively discussed in the early literature of Persia and India. In 400 B.C. Hippocrates recognized abnormal growths which he called *Karkinoma*. Four hundred



FIBRO-ADENOMA



BENIGN
INTRACYSTIC
PAPILLOMA



stromal cells. Changes in cells of the pituitary glands have also been described as a common accompaniment to breast cancer. This further indicates the association of the disease with an abnormal hormonal background. Trauma, often related to the discovery of malignant disease, has not yet been incriminated as a definite etiologic factor. Lawsuits are sometimes dependent on this supposition.

Pathology.—The many diverse pathologic forms make precise classification difficult, but a relatively simple one is suggested. Often a combination of pathologic types is found in one section. It should be pointed out here that there is no definite histologic correlation between tumor type and long-term survivals. Where the nuclear structure is characterized by anisonucleosis, with many nuclei being greatly enlarged, the chromatin coarse and clumped, the nucleoli prominent, and mitotic figures frequent, the prognosis is particularly poor. On the other hand, where there is much lymphocytic infiltration into the primary tumor and also increased reaction of the sinus histiocytes in the regional lymph nodes, survival time seems to be increased.

Scirrhus carcinoma is the most common type of breast cancer. It forms a stony hard tumor in which the dispersed epithelial cells grow in solid nests and columns. The malignant cells appear to be so compressed by the greatly increased fibrous tissue growth resulting from host reaction that there is no attempt at glandular formation.

Medullary carcinoma has a marked glandular proliferation with relatively little connective tissue. The lesion is soft and friable and microscopically consists of masses of large, round, epithelial cells. Medullary carcinoma has a relatively better prognosis. It seems to spread more slowly with larger involvement of solitary nodes. However, it has less local inflammatory tissue reaction.

Adenocarcinoma may be divided into the *mucoid type*, with its tendency to papilliferous change and the deposition of mucoid material, and the *ductal type*. The latter arises from the ducts of the breast parenchyma. Adenocarcinoma, while a relatively uncommon form of breast cancer, offers a somewhat better prognosis than does the scirrhus carcinoma.

In **intraductal carcinoma (comedo cancer)**, malignant cells at first remain confined to the ducts, and in this state the differential diagnosis between frank malignancy and carcinoma-in-situ may be exceedingly difficult. The differential diagnosis between carcinoma-in-situ and lesser degrees of epithelial hyperplasia may tax the resources of the most astute pathologist and cannot be accomplished in frozen sections. Pathologists naturally tend to protect themselves by labelling doubtful lesions malignant. Simple mastectomy is indicated for carcinoma-in-situ, as it is claimed that half of these lesions will become clinical cancer (intraductal) within five years.

Paget's Disease of the Nipple

Paget's disease of the nipple is a chronic intractable eczematoid change in the skin and occurs when a carcinoma close to the nipple has invaded and locally blocked the superficial lymphatic return. Cancer cells, which are here termed Paget's cells, invade the epidermis and become swollen and vacuolated. It is doubtful that the malignancy in these cases ever actually arises in the overlying squamous epithelium, but this point is occasionally subject to argument. The disease is from the outset a malignancy, and the treatment is mastectomy. Paget's disease metastasizes as infiltrating duct carcinoma. It has a better than average prognosis. The important point is that an area of eczema in the region of the nipple lasting 2-3 weeks should be biopsied. Similar lesions may occur elsewhere in the skin of the body. Ordinary benign eczema of the nipple usually responds promptly to local treatment such as wet dressings, antibiotics, or hydrocortisone ointment. On the other hand, Paget's disease is progressive.

Breast Cancer—Clinical Features

The problem of *early* diagnosis is in many respects insoluble. It depends on the connotation of the term. In the so-called *early stages* it is impossible to distinguish carcinoma from other breast swellings. Thus every lump in the breast of a woman must be considered as cancer until it has been removed and the proper

diagnosis has been made by histologic examination. Unsuspected breast cancers found accidentally have a remarkably good prognosis. Nodularity developing in one section of an otherwise normal breast may mask an early malignant change. A typical breast cancer is stony hard and is best felt by the flat of the hand compressing the organ against the chest wall. The edge of the tumor is vague and indefinite. With extension of the disease, there is ultimately skin involvement and local lymphatic occlusion with the development of peau d'orange. If the tumor is in the subareolar area, the nipple becomes retracted. Later the mass becomes fixed to the skin and to the pectoral fascia and muscles. If the tumor lies within, or has invaded one of the main ducts, there may be a nipple discharge, which is usually clear, but may be bloody. In the more diffusely growing forms, the tumor may involve the whole breast.

To assist in standardizing treatment and assessing results, four principal stages of breast cancer have been defined by Portmann. The limitations of any such gross artificial grouping should be at once apparent since the metastatic cellular nature of the disease does not allow accurate assessment.

Stage 1:

Tumor—localized in breast and mobile

Skin—not involved

Metastases—none

The treatment is radical mastectomy. After such treatment, 5-year survival may be expected in 70-80% of cases, and about 40% will survive 10 years. X-ray therapy should be unnecessary in this group.

Stage 2:

Tumor—localized in breast and movable

Skin—not involved

Metastases—a few axillary nodes, none elsewhere

With treatment as in Stage 1 with the addition of deep x-ray therapy, one may expect 25-30% 5-year survivals and about 12% 10-year survivals.

Stage 3

Tumor—diffusely infiltrating breast, fixation to chest wall, edema of breast and/or secondary tumors

Skin—edematous, indurated, ulcerated and/or secondary nodules

Metastases—many axillary nodes involved or fixed but no clinical or x-ray evidence of remote metastases

While the outlook in this stage is hopeless, worth-while palliation is sometimes achieved by mastectomy, but patients in this category are usually treated with x-ray and hormones.

Stage 4:

Tumor—as in any other stage

Skin—as in any other stage

Metastases—axillary and supraclavicular nodes extensively involved, clinical and x-ray evidence distant metastases

X-ray or hormone therapy may afford temporary palliation.

Survival statistics indicate that carcinoma occurring in the second breast should be dealt with as a primary lesion if evidence of other areas of involvement is absent.

In assessing end results and determining the value of treatment, it is necessary to compare the statistics with untreated cases. Medical literature contains observations on some 800 untreated patients, and it is doubtful if this group will ever be enlarged. The 5-year survival was 20% and the 10-year survival, 5%. Twenty per cent of the patients died during the first year. The average survival was 38.5 months after the lump was first noticed. Several cases are on record of persons living over 30 years with breast cancer. Good results are sometimes obtained by the treatment of late cases and poor results from the treatment of so-called early cases. The disease is extremely unpredictable, and late metastatic spread is seen more frequently in breast cancer than in any other type of malignancy.

One way of conveniently explaining the extremely diverse growth patterns of breast cancer as well as the unpredictability of therapeutic results is on the basis of *biologic predeterminism*. This thesis has been championed by Ian MacDonald of California. He contends that the clinical course in the individual case is determined before the symptomatic phase is reached and depends essentially on the inherent growth potential of the original tumor cell. While it is difficult to prove or disprove this contention, it might, in unthinking clinicians, promote an unjustifiably fatalistic attitude instead of stimulating therapeutic en-

thusiasm. An equally satisfactory and more constructive philosophy is the concept of *host resistance*. Unfortunately this is as yet a non-specific term, but it is likely that some of the many investigative projects now being pursued will illuminate and emphasize this avenue of thought.

Spread of Cancer

Cancer starts as a localized disease and with the passage of time spreads throughout the body. Some tumors behave almost like a syphilitic chancre, with widespread blood stream dissemination of cells almost before the primary growth is apparent as such. Other carcinomas seem content to remain localized indefinitely. The tumor spreads by direct extension when cell production is occurring at a faster rate than cell destruction, in other words when the growth is enlarging. The cells move along the lines of least resistance, like the crab they are named after; that is, along the tissue spaces. Surface tensions of the cell membranes are low, and they break away easily from the main tumor. They are capable of ameboid movements. Oxygen, glucose, and special nutritive materials probably attract these cells by chemotaxis toward the capillary loops, which are increased in number in the vicinity of the cancer. Although cells of breast cancer are about twice the size of red blood corpuscles, capillaries are quite distensible. Thus it is plausible that showers of cells are constantly being disseminated, not only by the lymphatics but also, directly, by the blood vessels. Tumor cells in peripheral blood have in fact been reported by some cytologists in a high proportion of breast cancer patients. Cancer growing in the medial half of the breast has a much higher rate of mediastinal gland involvement. The intrinsic lymphatic drainage in the breast is primarily to the subareolar area, so this area should always be widely excised in any operation for breast cancer, regardless of the location of the primary tumor. From the subareolar plexus, tumor cells pass deeply to the lymphatics lying on the pectoral fascia, and thence peripherally. The most obvious route is to the axillary glands and, secondarily, to the supraclavicular, infraclavicular, and cervical groups. Probably the lymphatics of the other breast form a primary target for

spread. It is impossible to tell pathologically whether a carcinoma in the other breast is primary or metastatic. Involvement of the other breast occurs in about 5% of operable cases. The other axilla is invaded early in a significant percentage. Lymphatic spread to the liver may take place via the lymphatics in the sixth interspace or by retrograde spread through the internal mammary chain which normally drains the dome of the liver. Clumps of tumor cells in either the pleural or peritoneal cavity frequently seem to break loose and grow at random on distal serosal surfaces. Lymphatic spread may cause a deposition of cancer cells in the skin, particularly in the area of the operation. Cells from intrathoracic involvement may pass to abdominal nodes by the lymphatics of the posterior part of the diaphragm. Breast cancer has an affinity for red bone marrow, and any bone in the body may be involved.

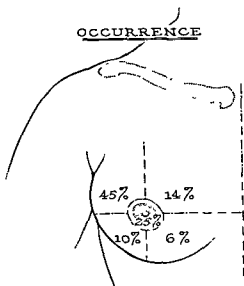
In patients dying from the disease, bone metastases are found in about 40%. The adrenals are invaded in half the cases and the other breast in at least 20%. In deaths from breast cancer, all patients have lymph node metastases, 65% have pleural invasion, and 60% liver metastases. Local skin occurrences are seen in about 40%. Brain metastases account for 12% of deaths. The actual dynamics of spread are not understood. A histologically normal endocrine system is rarely, if ever, seen in advanced breast carcinoma. It is likely that some chemical affinity for the cancer cells exists in specialized body tissues. For instance the pars nervosa of the pituitary gland commonly plays host to a metastasis, but this is not true of the pars anterior.

Although the majority of these tumor cells must be destroyed by unknown defense mechanisms or deteriorate after a sterile life, some must have the ability to lie dormant about the body for long periods of time ready to answer an appropriate summons for reactivation. Inanition, associated with disseminated pulmonary metastases, pleural effusion, anoxia, and pneumonia, seems to be the condition most directly related to death.

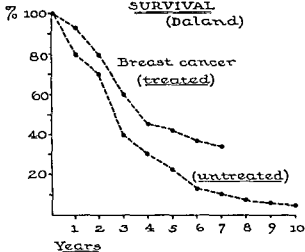
Treatment

The orthodox treatment of cancer has as its objective first the excision from the body of

OCCURRENCE

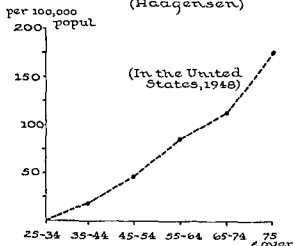


SURVIVAL (Baland)

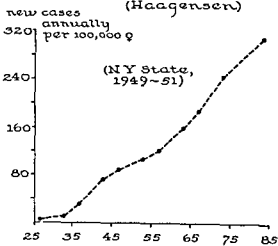


B.

MORTALITY BY AGE (Haagensen)

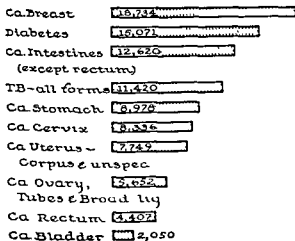


MORBIDITY BY AGE (Haagensen)

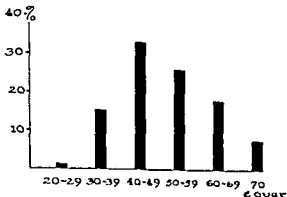


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COMPARATIVE MORTALITY (U.S WOMEN, 1950) (Haagensen)



AGE DISTRIBUTION (Haagensen)



F

FIG. 21. 11 Cancer statistics

cells that have taken on this new genetic behavior, and, if that is impossible, the use of any agent known or presumed to have a differentially destructive effect on the malignant cells. When this is impossible, there still remain medical and surgical measures for symptomatic relief which do much to make the final stages of this disease more tolerable.

Local disease is curable by adequate excision. In other words, if all the tumor cells lie within the tissue encompassed by surgical resection, cure is assured, and if not, failure is certain. The cornerstone of surgical treatment is a radical mastectomy as enunciated by Halsted in 1894. Although 5-year survival rates have shown improvement in some reports, it is a melancholy but undeniable fact that the average patient with breast cancer today does not seem to fare much better with respect to cure rate than did the patient of 60 years ago. This is due in some measure both to reluctance of the patient to seek early examination and to laxity on the part of the physician in insisting on thorough examination and early adequate treatment. No matter what personal philosophy we adopt about this disease, there is a substantially large group of cases that can be tremendously benefited by palliative surgery, even if cure cannot be obtained.

The clinical progression of disease in some patients is probably accelerated by growth of cells disseminated at the time of operative procedures, and some growths are so virulent that even the most careful surgery is not palliative but definitely harmful. Contraindications to radical surgery because of danger of spreading or causing acceleration of the disease and thus shortening survival time include acute (inflammatory) carcinoma, advanced disease with edema of the skin over the breast, and disease associated with satellite carcinomatous nodules in the skin. Where distant metastases exist, such as involvement of the supraclavicular glands, bones, lungs, and liver, operations to remove or prevent skin ulceration may occasionally be indicated, but x-ray and hormonal therapy are usually preferable. Inability to withstand surgery due to poor physical condition is an obvious contraindication to any operative attack on cancer.

Although a critical look at survival statistics indicates that about four out of five women with breast cancer will inevitably die from the disease, there are no grounds for adopting an attitude of therapeutic nihilism, since the palliative value of extirpative procedures has been clearly demonstrated to be very real. The term *operable* is often loosely used and means different things to different people. I should infer that the lesion may possibly be cured and at least the expected survival time of the patient materially prolonged, with relief of suffering and restoration of normal function.

X-ray Therapy

X-ray therapy is not curative, but tumor growth appears sometimes to be temporarily arrested and the life of the patient prolonged when it is properly administered. There is great variation in the radiosensitivity of breast tumors. The best response is from the more anaplastic types, despite the fact that they in general carry the worst prognosis. Exposure to radiation results in local arteritis, thrombosis, atrophy, and necrosis of tumor cells. Objections have been voiced from some quarters about the inadequacy of x-ray therapy alone and about the unpleasantness of radiation sickness and local skin reactions. However, its benefits should always be secured wherever possible in the treatment of all types of breast cancer.

X-ray therapy may be combined with surgery either before or after operation. Preoperative irradiation is administered with the intention of destroying malignant cells which may have spread from the primary tumor and of lessening the risk of spread at operation. In some advanced cases it makes surgical intervention feasible by reducing the growth of a relatively fixed lesion. The suggestion that valuable time may be lost and efficient wound healing jeopardized is probably overemphasized. In most centers, postoperative irradiation only is given in an attempt to destroy malignant cells which have been left in the operative field and those locally disseminated as a result of the operation. X-ray therapy is often effective in inhibiting the growth and pain of metastatic lesions. Pathologic fractures and osseous lesions occasionally rectify following its judicious application.

Hormonal Therapy

From the vast amount of experimental work since World War II and the barrage of papers describing clinical observations, certain useful clinical data are coming into focus. We are becoming more aware of the greater importance of classifying tumors according to their biologic properties rather than on the basis of the unrelated histologic attributes. Thus has evolved the classification of estrogen and pituitary-dependent (or stimulated) tumors as opposed to hormone-independent tumors.

Estrogens.—

Deprivation.—About 40-50% of breast cancers in premenopausal women show a definite degree of estrogen dependency. In other words, these tumors will respond by increased growth to an increase in the level of circulating estrogens or with a temporary retardation or regression of the disease on removal of this growth stimulus. Unfortunately, methods of estrogen assay have not yet been developed to a satisfactory state for easy practical use. However, oophorectomy in premenopausal women is a useful adjunct to therapy. This may be done as a prophylactic measure at the time of the mastectomy, but most clinicians prefer to wait until the onset of metastatic disease, at which time it is termed a therapeutic castration. Although no difference in survival times can be proved, the reason for delaying is to better observe the effects of the procedure, since a patient who responds to therapeutic castration will likely respond subsequently to hypophysectomy. Irradiation of the ovaries is not nearly as satisfactory as surgical removal.

Administration.—Diethylstilbestrol 15 mg. is given daily until a total of 4 Gm is taken. This treatment is reserved for patients 5 and preferably 10 years past the menopause. The tumor response improves with increasing age. Thirty per cent of patients show temporary regression of soft tissue lesions. Certain unpleasant side reactions can be expected. Temporary nausea and vomiting are encountered and are relieved by reducing the dose until tolerance is acquired. Uterine bleeding is the most troublesome phenomenon produced by estrogens. Outward signs such as pigmentation of the areola and axillary skin with breast en-

gorgement are often seen. While edema and hypercalcemia may develop in any patient with extensive bone metastases, these complications are sometimes precipitated by the administration of estrogen. They can be very serious and lead to anuria and death unless recognized and treated promptly. Patients placed on estrogen therapy should be conscientiously and closely watched for complications. The mode of action of estrogen is unknown, but the effects are considered to be mediated in some way by pituitary inhibition. Many authorities dogmatically state that estrogens should not be given for menopausal symptoms to women who have had breast cancer. The trained clinician uses the clinical response as the logical guide to hormone administration.

Androgens.—

Deprivation.—Following radical mastectomy for male mammary carcinoma, orchiectomy should be carried out. This is the situation in which hormone interference gives its best results in cancer therapy. Not only is the mechanism of action unknown, but no reasonable theories have yet been postulated to explain the effects.

Administration.—Androgens are given to the younger age groups of women in the form of testosterone propionate 150-300 mg/week, intramuscularly, for a period of 3 months. Although there are many unpleasant side reactions, over 50% of the patients are improved subjectively, with weight gain and relief of pain, and about 20% show recalcification of bone lesions. The chief unfavorable effects in order of frequency are hoarseness, hirsutism, increased libido, loss of scalp hair, acne, and the development of a ruddy complexion. The masculinization that androgens produce is so unpleasant that this hormone should not be given prophylactically. Androgens are more effective than estrogens in causing recalcification of bone lesions regardless of age, but there are cases where the growth rate of osseous metastases has been accelerated by androgens, with resulting hypercalcemia.

Adrenal Hormones.—

Deprivation.—Bilateral total adrenalectomy for advanced disseminated breast carcinoma has been studied and advocated by Huggins. It should be reserved for patients shown to have a proved hormone-dependent tumor. Sub-

jective response characterized by relief of pain and increased feeling of well-being has been obtained in 65% of patients and objective regression in 40%. The operative mortality is only 2.5%, but the patient must be maintained indefinitely on cortisone therapy in the range of 50 mg. daily. The good results from adrenalectomy are said to be due to more complete removal of estrogens from the body.

Administration—Cortisone therapy causes some inhibition of both pituitary and adrenal secretions. This substance probably also acts peripherally by displacing estrogens at tumor target sites. Recommended dosage is 150 mg orally per day for 3 days, then 50-100 mg. daily, depending on the degree of response. Hydrocortisone in four fifths the dosage is biologically equivalent. However, the delta steroids are preferable because of their freedom from salt-retention effects. Prednisolone 10 mg. is as effective as 50 mg. of cortisone and is slightly more active than prednisone. Worth-while remissions frequently occur even after the disease has become refractory to other forms of endocrine interference. Salt and water retention with pulmonary edema and cardiac failure, Cushing's syndrome, aggravation of diabetes, peptic ulceration, and osteoporosis, must be anticipated when using this therapy.

Hypophysectomy.—Removal of the pituitary gland for metastatic breast cancer was first performed in 1931. The rationale is that removal of the source of trophic hormones eliminates the release of steroid hormones from target organs. It is postulated also that a cancer-stimulating substance, possibly the somatotrophic hormone, would also be eliminated. However, the role of hypophysectomy in the treatment of advanced cancer of the breast is still in the process of being defined, and no satisfactory method of selection of cases is currently available. Premenopausal women responding to therapeutic castration and postmenopausal women demonstrating improvement from estrogen or androgen administration appear to respond favorably to hypophysectomy. It is not yet known whether this measure can produce an effect equal to the sum of oophorectomy and adrenalectomy. Although the evidence is not convincing, current trends favor hypophysectomy over adrenal-

ectomy. Practically all hypophysectomized patients show some degree of diabetes insipidus, and many require vasopressin. Nearly all require thyroid supplements. Temporary objective improvement is obtained in slightly more than 50%.

Hormonal therapy for breast cancer still remains a weak crutch on which to lean after surgery and irradiation are no longer of value. The dramatic effects sometimes encountered demonstrate the investigational value of these procedures and stimulate the search for new facts relevant to the nature of the disease.

Chemotherapy.—

Alkylating Drugs.—These are chemicals which are capable of the addition or substitution of an alkyl group in a compound. An alkyl group or radical is generally considered to be that unit which remains when one hydrogen atom is eliminated from a hydrocarbon, for example, methane (CH_4) \rightarrow methyl (CH_3 -) radical; ethane (C_2H_6) \rightarrow ethyl (C_2H_5 -) radical. It is alleged that some of these chemicals are particularly toxic to cancer cells during the phase of cell division, and certainly some show clearly demonstrable, though temporary, tumor-inhibiting effects in human beings.

Nitrogen Mustard, Sulfur Mustard, Triethylene Thiophosphoramidate (Thio-TEPA), Tetramine, Triethylene Melamine (TEM), Busulfon.—This group of drugs, together with thousands of other cytotoxic chemical agents, is being studied, but all are of research interest only and should not be considered in routine therapy. Prophylactic chemotherapy at the time of cancer operations is currently under investigation. For instance, nitrogen mustard is being given by some groups intravenously with the idea of destroying dislodged cancer cells before they can take root.

Radioactive Phosphorus.—Radioactive phosphorus is given orally or intravenously to some advanced cases in the form of NaH_2PO_4 with the phosphorus made radioactive. Some bone lesions have an affinity for activated phosphorus which directly irradiates the tumor cells. Testosterone is used concurrently as a so-called matrix-fixing agent for the P^{32} .

Colloidal Gold.—Colloidal gold¹⁰⁰ is administered by direct injection for the local treat-

ment of pleural or peritoneal effusions where the tumor seedings are small and diffuse. The colloidal particles are small and effect local irradiation. If there are appreciable pulmonary components, this treatment must be supplemented by through-and-through external radiation therapy.

It is doubtful if any presently known compounds will have a permanent place in the chemotherapy of breast cancer

the diagnosis is confirmed, the instruments, drapes, and gloves are changed to lessen the danger of dissemination of tumor cells. A blood transfusion is started. The incision begins over the insertion of the pectoralis major muscle on the humerus, then proceeds inward and downward over the anterior chest wall on the lateral aspect of the breast, staying at least 2" from the edge of the tumor. Another skin incision passes medially to encircle

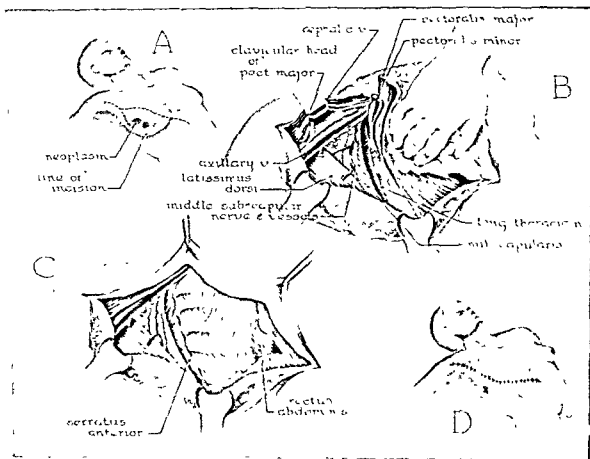


Fig. 218—Radical mastectomy

Radical Mastectomy

Radical mastectomy still remains the cornerstone of breast cancer treatment. The classical operation was first described by Halsted and is performed as follows. The patient is anesthetized, in a supine position, and draped with the appropriate arm abducted to 90 degrees. Unless previous needle biopsy has been done the mass is totally excised and examined microscopically to validate the diagnosis. If

the tumor. The skin flaps are developed with a sharp scalpel, keeping them as thin as is consistent with adequate circulation. This can be facilitated by lifting and stretching the skin edges with towel clamps.

The division of the clavicular and pectoral parts of the pectoralis major muscle is found and separated with the scalpel handle and followed down to the humerus. The head of the pectoral part is then detached at the hu-

meral insertion, and the muscle is reflected forward, exposing the pectoralis minor. The insertion of the latter is detached from the coracoid process and removed together with the surrounding clavipectoral fascia. The axilla is now accessible and its contents of lymphatic glands, fascia, and fat are carefully and thoroughly excised, working from the medial angle laterally. The axillary sheath must be opened as it contains many lymphatics. If possible, the long thoracic nerve to the serratus anterior and the nerve to the latissimus dorsi muscle are spared. Dissection is now carried down until both pectoral muscles are detached at their origins. The upper part of the fascia over the rectus abdominis is also removed. The mass of breast tissue and muscle is now discarded. A stab wound is made in the lateral skin flap for a long soft rubber drain. Difficulty may be encountered in skin closure because of the large amount of skin which must sometimes be removed. If closure is impossible without undue tension, a split skin graft should be taken from the thigh and sutured in place. A firm, well-padded dressing is now applied. The mortality resulting from this operation should not exceed 5%.

Arm movements are begun on the 5th or 6th day following inspection of the wound. On the 2nd or 3rd day, according to the amount of discharge, the drain should be removed, and by this time the patient is usually ambulatory. Sutures are removed from the 8th-12th day if the healing process has been normal.

A distressing complication of the operation of radical mastectomy is edema of the arm. This is caused by disruption of lymphatic pathways, low-grade infection, thrombosis of the axillary vein, or vascular fibrosis from x-ray therapy. It is treated by elevation, massage, or the use of an elastic arm bandage.

Simple Mastectomy

Since 1911, R. J. McWhirter, an Edinburgh radiotherapist, has been advocating simple mastectomy followed by x-ray therapy as a general treatment for breast cancer. His arguments have been based on two contentions. First, that while surgical intervention gives excellent results when the disease is confined to the breasts, the poor results from radical

mastectomy where the axillary nodes are involved may be due to surgical dissemination of cancer cells. Second, applying Dahl-Iversen's figures of metastatic spread, which showed 33% positive nodes in the neck and internal mammary invasion in 48% when the axilla was involved, a remarkably small group was left which was potentially curable, having only axillary node involvement. However, his results in over 2,000 cases show a 42% 5-year survival which is not out of line with the results of the radical procedure. This approach has met with much opposition on the grounds that it ignores the established principles of cancer surgery and depends on a lymph node survey that included a high proportion of advanced cases. There is, however, universal agreement that this technique should be applied to poor-risk patients or those with borderline or inoperable lesions.

Superradical Mastectomy

Extension of the scope of the radical operation has received much extensive investigation, and ingenious methods have been designed to extirpate increased quantities of anatomic structures contiguous with the breast. Wangersteen's superradical mastectomy has been planned to attack the lymphatic nodes of the neck. His operative mortality and morbidity rates preclude the general adoption of this technique.

Jerome Urban of the Memorial Hospital for Cancer has applied a procedure permitted in his hands a safe *en bloc* resection of the chest wall including the internal mammary nodes. Although his series commenced in 1951, the rationale is soundly based and the results from the first 275 cases are promising but more time is necessary for its accurate appraisal. It is a logical type of extirpation when the primary growth is situated medially and the cases properly selected.

It is unlikely that any significant advance in the management of breast cancer will occur until such time as more fundamental information pertaining to the specific nature of the neoplasm is available. The student should be cautioned about the part played by survival gymnastics and the careful selection of cases.

where any optimism is shown in articles dealing with breast cancer, particularly in the competitive reporting of results. Constant suspicion leading to early diagnosis and the prompt institution of adequate surgery cannot be too strongly emphasized, for therein lies the only hope of cure.

SARCOMA

Sarcoma of the breast is uncommon and may assume a great variety of forms, varying in malignant propensities from relatively benign lesions to extremely malignant ones such as hemangioendotheliomas (angiosarcomas) for which all treatment even in the earliest stages is apparently hopeless. When cystosarcoma phylloides or giant fibroadenomas become malignant, 96% will become sarcomatous, while only 4% will show carcinomatous degeneration. Breast sarcomas occur mainly following malignant degeneration of benign

fibrous tumors. Treatment consists of simple or radical mastectomy. For lymphosarcoma, x-ray therapy is a valuable adjuvant.

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Film Reference

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Breast Cancer: The Problem of Early Diagnosis (1950) (By National Cancer Institute, U. S. Public Health Service, Bethesda, and American Cancer Society, New York)	36 min	Sound Color	American Cancer Society 47 Beaver St New York 4, N. Y.

Chapter 15

Thoracic Surgery

Stewart Baxter, MD., and Darrell D. Munro, M.D.

INTRODUCTION

World War II and the years following have seen major developments in the field of thoracic surgery, the fundamental principles of which were established when diseases of the lung and pleura were first remedied by surgical means. Indeed, this specialty has matured and become parent to its offspring, the present-day cardiac surgery. Today every intrathoracic and chest wall structure is amenable to operative correction.

The modern concept of chest surgery includes the elimination or correction of the disease process, the preservation of maximum pulmonary function, and the eventual complete rehabilitation of the patient to an active and productive life. To accomplish this aim, the surgeon must completely understand the detailed anatomy of the pulmonary segment, which is the smallest functional and anatomic unit, and also the pulmonary and ventilatory function with the departures from normal caused by various disease processes.

ANATOMY

The lungs are subdivided into lobes, three in the right and two in the left, and each lobe is composed of primary units or segments. The key to understanding the segmental anatomy lies in the bronchial pattern of each lobe.

The air-conducting system, or bronchi, may be likened to a deciduous tree. Its leaves

are comparable to the clusters of alveolar sacs attached to the terminal twigs or branches of the bronchial system and oxygen is absorbed by these terminal structures. The complexity of this system is obvious, and variations from the normal occur. Nevertheless the pattern is remarkably constant when the intricacies of the branching systems of bronchi, arteries, and veins in the lung are considered.

Each bronchopulmonary segment possesses its own bronchial system, its own central pulmonary artery branch transporting unoxygenated blood to the segment, and its own peripheral vein draining oxygenated blood from the segment. Each segment is a separate functional and anatomic unit capable of performing independently and therefore amenable to surgical removal.

The most consistent anatomic structure in the lung is the bronchus with its branches. The most variable is the pulmonary venous system, while the arterial pattern is intermediate in this respect. The arteries are central in location and closely accompany the bronchi. The veins are peripheral, lying between and draining adjacent pulmonary segments, and are called intersegmental veins.

The standard nomenclature for denoting the branches of the bronchi is the *Jackson-Haber* classification. The trachea usually divides into a right and left mainstem bronchus at the level of the T4-T5 intervertebral disc. *There are 10 segments in the right lung. Likewise there are, in effect, 10 segments in the left lung.*

but on this side, two segments in each lobe originate from a single or common segmental bronchus, thus creating what is known as a compound segment. It follows then that on the left side, the number of primary segmental bronchi is reduced to eight. The next important point to realize is that the *lingula* of the left side corresponds to the middle lobe of the right side and constitutes the lower division of the left upper lobe.

The 10 segments in the right lung are distributed in the upper, middle, and lower lobes, respectively, according to the formula 3-2-5. These segments are named as one would expect, according to their anatomic location in the lobe with respect to the chest. Therefore, the three segments of the *upper lobe* are top, front, and back, or apical, anterior, and posterior. In the *middle lobe*, the segments lie side by side and therefore one is medial and one is lateral. In the *lower lobe*, the group of basal segments, four in number, is crowned by a single segment which is perched on top of these like a cone. This segment is uppermost in the lower lobe and is known as the superior segment, which must not be confused with the apical segment of the upper lobe. Below this, the basal segments are in anterior, posterior, medial and lateral positions, hence are so named. Thus, by consulting Fig. 219, the student can readily fix in his mind the segmental anatomy of the right lung, which is the key to the left.

The segmental anatomy of the left lung is now readily understood. You will recall there is a compound segment in the upper lobe and one in the lower lobe. In the *upper lobe* the apical and posterior segments arise from a common stem bronchus before its division, and therefore this compound segment is designated apical-posterior. In the lower lobe it is the anteromedial segment that is compounded. The one remaining difference is that in the *lingula* of the left upper lobe, which corresponds to the middle lobe of the right side, the segments are placed one on top of the other instead of lying side by side and are therefore called the superior and inferior segments. Nomenclature of the left side is as follows: the *left upper lobe* is divided into two main divisions, the upper division and the lower division. The upper

division, corresponding to the right upper lobe, is subdivided into the apical-posterior segment and the anterior segment. The lower division, which is the *lingula*, has two segmental divisions, superior and inferior segments. The *lower lobe* is quite similar to that on the right side. The superior segment of the lower lobe again sits on the basal segments which consist of the compound anteromedial and the posterior and lateral basal segments.

One should visualize rather than memorize this segmental anatomy, which will facilitate the segmental localization of disease seen on x-ray. The internal topography of the openings of the segmental bronchi is an important study which must be mastered before diagnostic bronchoscopy is attempted. This will be described in detail in the section on bronchoscopy.

The branches of the pulmonary arteries are named in exactly the same manner, for they accompany the segmental branches of the bronchi. Thus on the right side there are 10 main subdivisions of the pulmonary artery, each accompanying the bronchus. The pulmonary artery and its branches are mainly a transporting system and serve to carry unoxygenated blood from the right ventricle to be distributed to the dense capillary network in the alveolar walls. The pulmonary arteries are nutrient for only a minor fraction of the pulmonary parenchyma, and therefore one may ligate and sever the main pulmonary artery without subsequent necrosis of the lung. The pulmonary veins begin in the distal branches of the pulmonary capillaries and coalesce with larger branches, eventually reaching the hilum. They carry oxygenated blood to the left side of the heart for distribution to the remainder of the body.

The bronchial arteries are nutrient to the bronchi and a major portion of the lung parenchyma, and generally one is on the right and two are on the left side. They originate directly from the thoracic aorta on the left and usually from the first intercostal artery on the right, supplying oxygenated blood and nutrition to the bronchial system as far as the peripheral respiratory bronchiole and alveolar ducts. They also nourish the bronchial glands, visceral pleura, pericardium, nerve

plexuses, esophagus, and the coats of the pulmonary vessels. Most of the blood brought to the lungs by the bronchial arteries returns by way of the pulmonary veins; the rest returns by way of the bronchial veins which, in general, correspond to the bronchial arteries

there is a free peripheral anastomosis between the bronchial arterial capillaries and the pulmonary venous capillary bed.

The lymphatic drainage of the lung provides an interesting anatomic study, particularly with respect to primary neoplastic disease. The

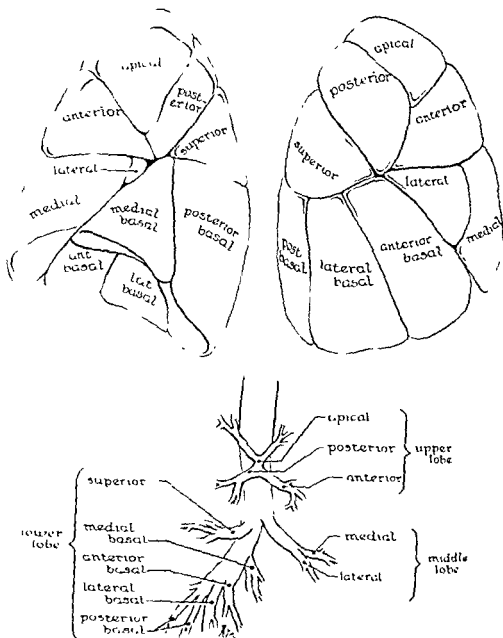


Fig. 219—Lobes and bronchi of right lung according to the Jackson-Huber terminology

but are not well-defined anatomic structures. On the right side they empty into the azygos vein and on the left side either into the superior intercostal or the accessory hemiazygos vein. There are no anastomoses between the bronchial and pulmonary arteries. However,

vessels originate in two plexuses, the superficial and the deep. The *superficial plexus* lies on and beneath the visceral pleura and fans out beneath this membrane, draining toward the hilum to empty into the hilar glands. The *deep plexus*, consisting of a bronchial and a

vascular group, is responsible for draining most of the lung parenchyma. They follow the terminal twigs and branches of the bronchi and vessels along their segmental divisions to the larger nodes at the root of each lung. For practical purposes, no anastomosis occurs

that are distributed along and adjacent to the branching bronchi. Characteristically, these small peripheral nodes are located in the angles at the points of bronchial division. As the efferent lymphatic channels pass toward the hilum, the nodes are progressively larger.

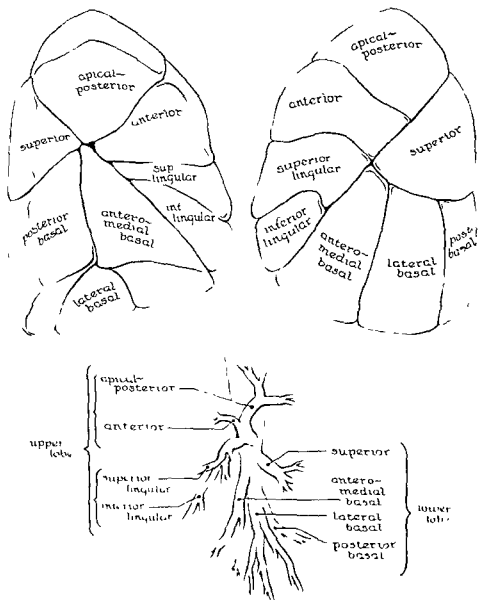


Fig. 220—Lobes and bronchi of left lung according to the Jackson-Huber terminology

between these two systems. They both drain centrally, the superficial around the rim of the wheel and the deep along the spokes to the central hub or root, where anastomoses may occur. From the lung parenchyma the deep channels drain successively into lymph nodes

At the main lobar bronchi, the nodes are grouped as a ring around the bronchus. The lymph nodes along the roots of the smaller bronchi are known as the *pulmonary nodes* and those around the main lobar bronchi as *bronchopulmonary nodes*. From the broncho-

pulmonary nodes, efferents pass to nodes at the main bifurcation, the *interbronchial nodes*, and also to nodes around the roots of the great vessels. From here they drain centrally to the *tracheobronchial nodes* situated in the angle between each mainstem bronchus and the trachea. Thence, efferents pass upward

fact has been established. A cross-drainage exists from the left lower lobe by which the lymphatic channels drain directly upward into the lymph nodes in the deep left cervical chain or can cross over via the subcarinal channels and drain into the right cervical chain. This, of course, has a direct influence

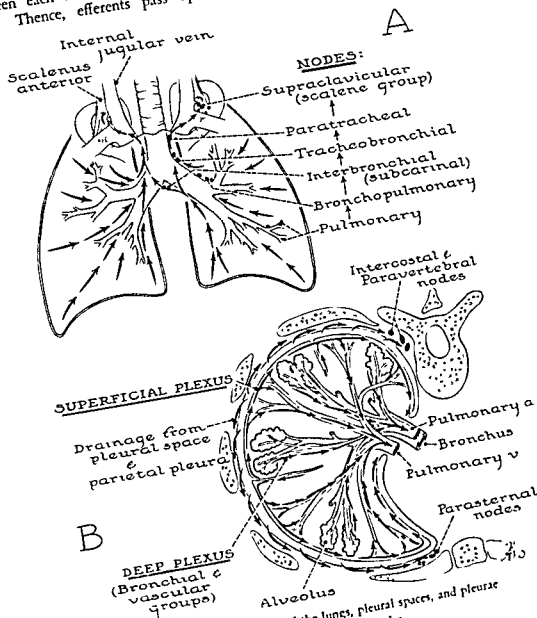


Fig. 221.—The lymphatic drainage of the lungs, pleural spaces, and deep cervical nodes
A, Centripetal lymph flow to hila and deep cervical nodes
B, Diagrammatic cross-section showing details of lymph plexus drainage

along the trachea, where they are known as the *paratracheal nodes*. The lymphatic drainage from the lungs eventually empties into the deep cervical group (*supraclavicular nodes*) at the root of the neck and from here into the venous system. One important anatomic

on the route of lymphatic spread in cancer of the left lower lobe. Therefore it must be remembered that all the lymphatic drainage of the right lung drains into the right deep cervical nodes; the lymphatic drainage of the left upper lobe drains into the left deep cer

vical nodes; but lymphatic channels from the left lower lobe may drain into either side. A better understanding can be obtained by a study of Fig. 221.

It is appreciated that there is an unusual anatomic situation in the lung whereby all the lymphatic drainage must necessarily pass through a very small neck on its route centrally and upward to the mediastinum and base of the neck. This anatomic site exists at the primary hilum, in the area immediately surrounding each mainstem bronchus. Therefore, despite the widespread prevalence of pessimism surrounding cancer of the lung, there is an unusual anatomic advantage in the surgical excision of a primary tumor and the ablation of its lymphatic drainage at its site of exit from the lung involved. It must also be mentioned that while the lymphatic drainage of the lung itself is always upward, that of the pleural space and parietal pleura is along the intercostal channels, posteriorly to nodes in the posterior intercostal spaces, anteriorly to nodes in the parasternal area along the internal mammary chain, and downward to nodes in the paravertebral areas and beneath the crura of the diaphragm.

The innervation of the lung is still far from understood and contains many mysteries. However, the nerves are derived from the autonomic nervous system, from both the parasympathetic (vagus) and the sympathetic divisions. These systems supply anterior and posterior pulmonary nerve plexuses which send fibers outward along the root of the lung to the bronchial walls, to the alveolar sacs, and to the pulmonary vessels. The nervous control of the pulmonary capillary bed has not been elucidated and is one which may ultimately prove to be of extreme importance. It is here that a buffer of the pressures between the pulmonary and systemic circulatory systems exists.

PHYSIOLOGY OF THE THORAX

The outstanding feature which distinguishes the pleural space from all other body cavities is the negative subatmospheric pressure which is normally present. This is produced by (1) the changes in the intrathoracic volume brought

about by the excursion of the chest wall during inspiration and expiration, and (2) the inherent elastic recoil of the lung. This negative pressure averages minus 6-9 mm of mercury at the end of inspiration and minus 4-6 mm at the end of expiration.

Pulmonary ventilation, on which life depends, cannot occur unless expansion of the thoracic cage is followed by inflation of the lung. If the pleural space is open to the atmosphere, an inspiratory effort will cause the suction of air into the pleural space with resultant lung collapse, adequate ventilation then becomes impossible.

It is important to emphasize that a unilateral pneumothorax will result in a reduction of the negative pressure in the opposite pleural space. This is caused by movement of the normally mobile mediastinum toward the intact side, the resultant reduced pulmonary volume of which decreases the vital capacity and tidal volume.

During inspiration the thorax is enlarged by the upward and outward expansile movement of the ribs and by the descent of the diaphragm which create a lower pressure in the alveoli, and air flows from the atmosphere through the tracheobronchial airway into the alveoli.

Since the lungs are normally elastic, the work of inspiration is mainly expended in overcoming the resistance of the lungs to expansion. At the end of inspiration the chest wall relaxes, and expiration occurs as a passive phenomenon. During any respiratory cycle the work involved to cause lung ventilation may be divided into (1) the work used to expand the lung against its elastic recoil (compliance), (2) the work necessary to overcome functional losses within the system (a very small component), and (3) the work necessary to overcome resistance to air flow throughout the tracheobronchial tree.

Open pneumothorax implies the presence of free air in the pleural space, with entry and exit through a chest wall defect with each phase of respiration. If the defect equals or exceeds the tracheal airway in size, more air will enter the pleural space than the lung

Closed pneumothorax implies the presence of free air in the pleural space, the amount of air remaining constant. Such a condition is usually well tolerated, since the movements of the chest wall still produce a swing in intrapleural pressure. The ventilation which is possible depends on the degree of pneumothorax present.

In *tension pneumothorax* a valve action exists whereby air enters the pleural space on inspiration and cannot be expelled during expiration. This situation is the result of a flap valve wound. Air continues to be sucked into the chest with each inspiratory effort, the negative pressure becomes completely abolished, and in a short time a high positive pressure develops. Complete collapse of the lung results with marked mediastinal shift and consequent compression of the opposite lung. Serious dyspnea ensues, and as respiratory effort increases, more air enters the space and a vicious cycle is established. This is an emergency requiring immediate decompression by needle aspiration or catheter drainage and the prevention of further air entry through the defect by surgical closure or suitable dressing.

PULMONARY FUNCTION TESTS

A number of physiologic tests have been developed for the qualitative and quantitative assessment of pulmonary function which have proved to be of great value in diagnosis, assessment, and treatment of surgical patients with cardiopulmonary disorders.

A specific enquiry with regard to *dyspnea* is of fundamental importance, and this provides good evidence of the patient's cardio-respiratory powers. The ability to climb stairs and the mobility of the thoracic cage on deep breathing should be studied.

Radiologic evidence of parenchymal disease, pleural thickening, and spinal and chest wall deformities generally signifies some limitation of function. The diaphragmatic and chest wall mobility should be noted on fluoroscopy.

The *maximum breathing capacity* (MBC) is the simplest and most universally applicable and provides a measure of the ability of the patient to move air in and out of the lungs with maximal respiratory effort. It is expressed as the number of liters of air per minute.

Comparisons are made of the result obtained with average normal values for persons of similar age and stature. The accuracy of this test depends largely on the patient's cooperation, the skill of the technician, and the type of apparatus.

The *vital capacity* is much less useful as a test of pulmonary function and is a measure of the greatest amount of air that can be exhaled after the deepest possible inspiration. It may be a misleading indicator of pulmonary function; e.g., in *emphysema* a relatively normal vital capacity may be present with a low maximal breathing capacity.

The *time vital capacity* is a measure of the maximal amount of air that can be exhaled in relation to time. Normally 95% is expelled in the first 3 seconds; lower values indicate an obstructive element in the expiratory phase e.g., in bronchospasm as occurs in asthma and chronic emphysema.

Bronchspirometry enables the respiratory function of each lung to be measured separately. Using this method the oxygen uptake, carbon-dioxide elimination, vital capacity, and ventilation can be determined. Of these factors, the oxygen uptake is the most useful indicator of the contribution each lung is making to the over-all pulmonary function.

Further information of value may be obtained by calculating the *ventilation equivalent*, i.e., the number of liters of air breathed per 100 ml of oxygen consumed.

In complicated problems more complex measurements of pulmonary function may be required. These include the measurement of the *pulmonary diffusing capacity* and analysis of the *blood gas content* of an arterial blood sample. The first of these measurements will reveal whether there is any barrier to the passage of oxygen from the alveoli into the blood and whether the surface area for oxygen exchange within the lung is adequate. This measurement is important in problems of pulmonary fibrosis and in chronic emphysema. The *oxygen saturation of arterial blood* will help to reveal the presence of any considerable shunt of blood within the lung or whether there is inadequate oxygenation of blood passing through lung alveoli. It is important to

realize that there may be considerable impairment of pulmonary function and disabling dyspnea without any gross change in arterial saturation

It has become increasingly apparent that the determination of *carbon-dioxide tension of the arterial blood* is vitally important. This is particularly true in the immediate postoperative period, when the patient may fail to ventilate the lungs adequately. This hypoventilation will very rapidly bring about carbon-dioxide retention. It must be emphasized that this potentially dangerous condition may be present although the patient's color when breathing oxygen is satisfactory. An elevated arterial carbon dioxide, if progressive, will result in stupor, progressing to deep coma and ultimate death. It is likely that many postoperative deaths, particularly in the older age groups, terminating in coma, are due to this sequence of events. To reverse the carbon-dioxide retention, one must provide adequate and efficient ventilation by cleaning the bronchi and improving all mechanical factors to assist air exchange. It may be necessary to use a mechanical respirator for this purpose, and a tracheostomy may be indicated. This diminishes the dead respiratory space, improving alveolar ventilation and, in addition, facilitates bronchial aspiration.

INVESTIGATION AND DIAGNOSIS OF A CHEST CASE

History and Physical Examination

A few leading questions may indicate the nature of the underlying disease. *Cough* is one of the commonest symptoms. Normally, this is a reflex mechanism for the expulsion of secretions and foreign material irritating the bronchial mucosa. It may be a dry, nonproductive cough which is produced by an intrinsic tumor in the lumen of the bronchus, or by extrinsic pressure as a result of enlarged mediastinal and peribronchial lymph nodes such as occurs in Hodgkin's disease.

A cough which is productive is most important, and the character of the *sputum* must be carefully noted. Sputum may be measured by collecting it in a single container to estimate the daily volume. In severe bronchiectasis

there is copious mucopurulent secretion. Blood expectorated in the sputum is as serious a sign as hemorrhage from any other mucosa-lined tract of the body. The production of bloody sputum or hemoptysis indicates a break in the integrity of the mucous membrane of the respiratory tract and may result from severe infection such as occurs in bronchiectasis, foreign body erosion, ulceration in endobronchial tuberculosis, rupture or erosion of a large blood vessel in a tuberculous cavity, or, finally, from the friable vascular surface of an ulcerating neoplasm. One must not accept the patient's statement that he has not expectorated blood, for often his observations are not reliable since, to him, blood in the sputum usually means a gross amount thereof. The patient should be specifically asked: "Have you ever noticed any streaks or specks of blood in your sputum?" Have you paid particular attention to this detail?" If not, he is instructed to do so.

Chest pain may be of the typical pleuritic type which is aggravated by deep breathing or coughing. Another type is a constant, boring, severe pain due to malignant invasion of the chest wall itself. Referred pain around the chest wall from nerve root pressure and other extraneous sources must be elucidated.

The presence or absence of varying degrees of *shortness of breath* must be ascertained and carefully assessed as to the extent of disability it produces. Another significant sign, and one which is often neglected, is the presence of a *persistent unilateral wheeze*. To elicit this sign, one must ask the patient to forcibly expire the maximum amount of air, because frequently this sign is present only at the end of forced expiration. Sometimes there is a history of a persistent unilateral wheeze which had been present for a number of weeks and then completely disappeared. In such a case there has been a narrowing of the bronchial lumen to the critical point where air passing through the narrowed orifice is set in vibration, producing the wheeze which will disappear when the occlusion becomes complete. A persistent unilateral wheeze, or one which has been transient and disappeared, is generally due to an organic obstruction in the bronchus.

As in any examination, the *patient's color* is of importance. *Hoarseness* is another significant sign. It denotes invasion or compression of the recurrent laryngeal nerve by tumor. The presence of *fever* indicates an infection. *Weight loss* has been a time-honored symptom indicative of lung cancer, and if such has occurred, it usually is a late sign of widespread metastases. Apart from the purely metabolic effect of the carcinoma, there is no particular reason why a patient should lose weight unless such a cancer is producing obstruction with pneumonitis and chronic suppuration or widespread metastases. *Acute swelling and pain in the joints* are of special significance. Diseases of the chest are prone to be accompanied by a form of rheumatism known as *pulmonary osteoarthropathy*. It occurs occasionally in carcinoma of the lung. Recently, it has been recognized that the nervous system may be affected in cases of pulmonary carcinoma; a neuropathy with muscle weakness and sometimes frank paralysis occurs. In such cases no organic lesion can be demonstrated in the central nervous system. The mechanisms for neuropathy and osteoarthropathy are unknown.

In the physical examination, besides the findings by percussion and auscultation, a few points are of extreme importance. Particular attention should be paid to the lymphatic *drainage stations of the lung*. The accessible and palpable areas are the supraclavicular fossae. To be properly examined, the patient must be seated, with the examiner standing behind with one hand on the patient's head and the other palpating the supraclavicular and deep cervical chain. With the head turned toward the side being examined, the strap muscles can be relaxed, and deep palpation in the fossa may reveal the presence of hard nodes. If such is the case, a biopsy with microscopic examination may reveal the nature of the disease process. This is particularly true for cases of Boeck's sarcoidosis, Hodgkin's disease of the mediastinum, and cancer of the lung. The presence of digital clubbing, cyanosis, and swelling of the joints should be noted. The general examination should exclude the presence of enlargement of the liver and any other palpable masses.

Radiologic Examination

A good radiologic examination, with films of high quality, and a radiologist familiar with the detailed appearances of chest x-rays are absolute prerequisites to diagnostic work of high quality. *Routine plain x-rays* of the chest often indicate a diseased state. *Fluoroscopy* is used to determine the movements of the diaphragm and chest wall, the entry and exit, the movements of the heart and of its chambers, as well as the configuration of the barium-filled esophagus. The presence of a paralyzed diaphragm can be verified by this method. The *tomogram (planigram)* is a method of examination whereby for predetermined planes, at depths usually 1 cm apart, films in sharp focus for the set level are obtained, the remainder being blurred. This is achieved by a specialized technique whereby the rays cross one another at a critical focal point, which can be adjusted at varying depths throughout the chest. With such a series of films it is possible to show in detail the structures at each particular level, the exact anatomic site of disease, and also the degree of lymph node involvement.

The *bronchogram* is valuable in outlining the bronchial system. A radiopaque substance is instilled into the bronchial tree and flows with inspiratory movements and by gravity along the mucosal lining into the peripheral branches. Abnormalities such as dilatation, bronchiectasis, obstruction, the presence of anomalies, and enlarged lymph nodes can thus be accurately identified. The newer water-soluble dyes are preferable because they are completely absorbed and do not leave residual opacities that obscure later x-ray studies.

Barium swallow may be indicated in any disease of the mediastinum to outline the position of the esophagus and its relation to the disease process, in addition to any intrinsic esophageal disease. It is also used to outline the posterior border of the heart to demonstrate chamber enlargement. In many cases of bronchogenic carcinoma it is employed to exclude involvement of the esophageal wall by direct invasion and to demonstrate the presence of enlarged lymph nodes producing compression defects.

Pulmonary angiography may be employed to diagnose and assess such conditions as arteriovenous aneurysm, abnormalities of the pulmonary vascular bed, the extent of the spread of bronchogenic carcinoma, displacement caused by mediastinal tumors, and secondary node invasion of the heart and major vascular structures

Endoscopy

In some cases, although the chest x-ray is reported normal, bronchoscopic examination will reveal the presence of serious disease. The direct observation of the air passages and their ramifications presents a more complex picture than that found in any other part of the body accessible to endoscopy. The bronchoscope is a speculum; its rigid structure and size and the anatomic design of the tracheobronchial tree restrict the extent of the air passages that are accessible to inspection. Employing the standard bronchoscope, only the trachea, larger bronchi, and some major orifices to different lobes are visible. The small segmental bronchi and peripheral radicles cannot be seen. However, telescopic inserts with different lens systems, introduced through the bronchoscope, permit inspection of otherwise hidden portions of the bronchial tree. These attachments have greatly increased the scope of inspection and permit direct examination of segmental orifices and divisions. In spite of this, some portions still remain beyond our vision. However, these areas are amenable to an indirect method of examination by the collection of secretions from segmental bronchi for bacteriologic and cytologic studies. This material should be collected from the precise subdivision leading to the disease. The examiner must therefore be thoroughly conversant with the segmental anatomy and internal topography of the tracheobronchial tree and should be accustomed to interpreting chest x-rays with respect to segmental distribution of disease. No examination of the bronchial tree is adequate by present-day standards unless the telescopic inserts are employed.

Bronchial bleeding demands bronchoscopy just as unexplained rectal bleeding demands proctoscopy. Adequate topical anesthesia is essential for successful bronchoscopy, and a

routine method of examination must be developed. There are special features which are important. One should determine whether the major component of the lesion under vision is endobronchial, intrabronchial, or extrabronchial. Compression of a bronchus either on one side or in a circumferential manner so that it is concentrically narrowed, is an endoscopic sign of grave significance, usually denoting malignancy, and this same finding may also occur in Hodgkin's disease and in Boeck's sarcoidosis with marked lymphadenopathy.

A bronchoscopic diagnosis is made in one of three ways (1) visualization, (2) direct tissue biopsy, and (3) examination of secretions or lavage material aspirated from the site of the disease. Bronchoscopy, if properly performed, is acceptable to the patient under local anesthesia and can often be carried out as an outpatient examination. Early detection of bronchogenic cancer is essential, and bronchoscopy is indispensable to this end.

Esophagoscopy is the second endoscopic procedure used in the diagnosis of chest lesions. Many of the principles of bronchoscopy are directly applicable to the use of the esophagoscope. These are procedures requiring caution in their performance, as perforation of a bronchus or of the esophagus may result.

Examination of the Sputum

The sputum obtained from the productive cough is a mixture of transudate and exudate from the air-conducting system, and microscopic and bacteriologic studies are often rewarding. It is apparent that bacteriologic smears and culture will identify the offending pathogens responsible for many of the suppurative lung diseases. The identification of an organism gives the precise etiologic factor and, furthermore, cultural methods using the sensitivity-determining techniques will indicate the drug or chemotherapeutic agent of choice. The proof of tuberculosis rests on the identification of the tubercle bacillus in the sputum or bronchoscopic washings. Some uncommon forms of lung infections such as the fungus group may be identified by special cultural techniques.

Equally important is cytologic examination which permits the identification of malignant cells that have exfoliated from a cancer of the bronchial mucosa. Such microscopic examination requires a cytologist familiar with the appearances of malignant cells, who can interpret the slides accurately. Deep cough specimens are necessary for the examination in suspected cancer, and often direct bronchial washings will contain clumps of tumor cells. Clinics where competent cytologists are employed report an accuracy of 85% or better, false positives being rare.

Diagnostic Biopsy

Recently the cervicomediastinal node biopsy (scalene node biopsy) has proved to be of great value. The underlying anatomic fact is that the lymphatic drainage is to the deep cervical node chain on each side. The technique is a simple approach through a short incision above the clavicle. The deep cervical fascia is incised and the scalene fat pad exposed. This lies over the scalenus anticus muscle and invariably contains a group of small lymph nodes. Palpably enlarged nodes may be found and excised for section and identification. If enlarged nodes are not found, the scalene fat pad is removed for microscopic examination of its contained nodes. These nodes are often the mirror of the pathologic intrathoracic process and give an accurate tissue identification of any disease that may be present in the lungs or mediastinum. It is also possible to enter the superior mediastinum, in the paratracheal area, and palpate any gross tumor or lymph node enlargement in this region. If a node is palpated, a specimen may be obtained with a laryngeal biopsy forceps. It is possible to reach the level of the 6th rib posteriorly, and, on the right side, to feel the right upper lobe bronchus. Scalene node biopsy is valuable in diseases possessing an affinity for lymph nodes, such as Boeck's sarcoidosis, Hodgkin's disease, and other lymphomas, as well as cancer of the bronchus. Histoplasmosis, tuberculosis, some of the fungus diseases, and the pneumoconioses may also be diagnosed in this way. In addition to the tissue sections of the nodes, a portion of the freshly removed node

may be sent for bacteriologic culture. One other application is the submission of a portion of the biopsy specimen for mineral assay, to identify the pneumoconioses such as asbestosis and silicosis.

Its greatest use is in cancer of the lung, as often two facts are immediately established. In the first place, tissue diagnosis of metastatic carcinoma in the lymph node removed, and, second, this finding automatically contra-indicates any form of surgical intervention for excision of the primary neoplasm. A cervicomediastinal biopsy may be indicated even though no palpable enlargement of the lymph nodes is present, since they are situated beneath the deep cervical fascia and may not be easily palpated. In their series of cervicomediastinal biopsies, Harken and Black have demonstrated the usefulness of this procedure, as they obtained a positive diagnosis in 30% of cancer of the lung, they found that 40% yielded positive nodes in this area. In other words, 40% of the cases of carcinoma of the lung were at once proved to be inoperable. Therefore, in every case of this disease, either proved or suspected, a cervicomediastinal biopsy should be performed. Lesions of the right lung require only a right-sided biopsy, lesions of the left upper lobe require only a left-sided biopsy, but lesions of the left lower lobe, because of crossed lymphatic drainage, require a biopsy in both the supraclavicular areas before positive assessment can be obtained.

Direct lung biopsy has been advocated recently to establish the nature of certain intrathoracic diseases. This is a useful method to determine the identity of an obscure disease process and is generally used for lesions that are diffusely distributed through both lungs. Such conditions are found in Boeck's sarcoidosis, the pneumoconioses, peripheral bronchiolar carcinoma, obscure fibrotic diseases of the lung, and the Hammond-Rich syndrome. The biopsy is performed through a small thoracotomy incision in an anterior interspace. The patient in the supine position. An adequate representative wedge of tissue is removed from the edge of a lobe for histologic study. The cut lung edges are repaired, the lung is re-expanded, and the wound is closed.

From such a biopsy the exact identity of these bilateral pulmonary lesions is often revealed, whereas the x-ray appearance is not usually diagnostic. The operation is performed under endotracheal positive pressure anesthesia, requires a minimum of exposure, and has a low morbidity and mortality rate.

Aspiration biopsy is used only when all other methods of diagnosis have failed, and only for peripherally located lung tumors with adhesions or invasion of the chest wall or for some mediastinal masses.

Exploratory Thoracotomy

In a fair percentage of cases it is inevitable that despite a thorough investigation, an accurate diagnosis cannot be reached. Such cases require an exploratory thoracotomy, which is particularly applicable to the peripheral round lesion (coin lesion) in the lung. Actually, for such cases most of the aforementioned tests may be eliminated and a thoracotomy performed to approach the problem directly. This establishes its exact nature by excision and pathologic study and institutes definitive treatment at the time. Thoracotomy carries no greater risk than laparotomy and can be used for the same reasons.

In this category we should also mention the *idiopathic pleural effusion*. In spite of all methods of investigation, accurate diagnosis of some effusions is impossible, and there should be no hesitation in performing a thoracotomy to establish their identity and causative factor. These cases are prepared with antituberculosis drugs because a considerable percentage have a tuberculous basis. During exploratory thoracotomy, it is usually possible to obtain a section of tissue for immediate frozen section and diagnosis. If the lesion is located deeply within the parenchyma, a segmental wedge or lobe resection may be necessary.

CONGENITAL ANOMALIES OF THE THORACIC CAGE

Certain somewhat rare congenital anomalies of the thoracic structures or contents require recognition and, occasionally, operative correction.

Anomalies of Ribs and Pectus Excavatum ("Funnel Chest") (See Chapter 30.)

Hernia of Lung

Hernia of the lung may be congenital or acquired, due to trauma or infection involving some portion of the thorax. A true hernia exists when a portion of lung covered by parietal pleura protrudes through an abnormal opening in the thoracic wall, mediastinum, or diaphragm. *Prolapse* of lung is a protrusion of lung through the parietal pleura.

Etiology.—Most of the cases are traumatic, but rare congenital supraclavicular herniation through Sibson's fascia is encountered. Herniation may be due to large infected wounds of the chest.

Diagnosis.—The diagnosis is usually evident from the history and physical examination, there being a resilient, resonant tumor which appears on respiration, coughing, and straining.

Treatment.—Small supraclavicular hernias may be completely asymptomatic, and no operative treatment is indicated. Repair of Sibson's fascia can be performed in more severe cases. Repair of the traumatic types is more difficult, requiring frequently the use of fascia, periosteal flaps from adjacent ribs, and even the transplantation of ribs above and below the defect.

TRAUMA OF THE THORAX

Trauma of the thorax may involve injury not only to the structures of the chest wall but also to its contents. Several traumatic mechanisms are recognized, e.g., direct and indirect violence, blows, and blast injury. There may be a break in the continuity of the chest wall, including the pleura, e.g., stab and gunshot wounds, or there may be tears and ruptures of the intrathoracic organs, lungs, heart, pericardium, esophagus, and great vessels. Consequently, for the sake of clarity, this subject will be considered under the headings:

1. Nonpenetrating trauma of the thorax and viscera
2. Penetrating wounds of the thorax
3. Blast injuries of the thorax

Nonpenetrating Trauma of the Thorax and Viscera

This may result from direct blows as well as crushing injuries. *Contusions and hematomas* of the chest wall rarely require surgical treatment. *Rupture of an intercostal artery* from stab wounds or rib fractures, not penetrating the pleura, may occasionally require open exploration and ligation.

Fracture of Ribs

Etiology.—Direct blows, crushing injuries and, occasionally, muscular strain and severe coughing or sneezing may fracture the ribs. Pathologic conditions such as cysts or tumors predispose to fracture. The most common sites

pleura and lung, but the absence of this finding does not rule out lung damage. If the lung has been punctured, there may be bloody expectoration. Careful x-ray study will confirm the diagnosis.

Treatment.—The treatment of simple fractures involves some form of strapping, sedatives, and intercostal nerve block. *Strapping* can be done with adhesive tape, the conventional method of strapping the affected side to the unaffected side being most widely used. However, some find that complete circular strapping of the lower ribs is more effective, regardless of the site of fracture. A well-fitted and well-applied chest binder has been found to be just as effective and does not require

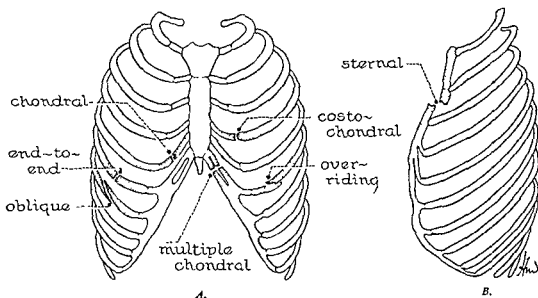


Fig. 222—A, Types of rib and chondral fractures B, Fracture displacement of sternum

are from the 4th-8th ribs, along the anterior and posterior axillary lines and at the angles. These usually result from compression of the chest. The 1st rib may be fractured in downward trauma to the clavicular region. Types vary from simple, undisplaced, to grossly comminuted fractures which may be complicated by damage to the deeper structures.

Signs and Symptoms.—Pain at the site of injury and on respiration is characteristic. Marked voluntary splinting of the abdominal muscles on the affected side is usual. Localized tenderness and crepitus may be felt at the site of fracture. Tissue emphysema is only present if there has been rupture of the parietal

shaving of the chest. It can be readjusted and removed for x-ray and physical examination of the chest.

Regional nerve block is useful in relieving severe pain and respiratory embarrassment early in the condition. One or usually two intercostal nerves are infiltrated with 1% Novocain above and below the site of fracture or fractures, 3-5 ml. being injected in each space.

Fracture and Dislocation of Costal Cartilages and Sternum

This is the result of either a direct blow or crushing injury. Injury to the intrathoracic

organs is quite common. Reduction of the dislocation may be necessary, with fixation by strapping or by hyperextension plaster mold. Open reduction and fixation by suture or Kirschner wire are occasionally necessary if gross displacement is present.

"Stove-in-Chest"

This refers to a severe crushing injury when one side of the thorax has been driven in. Numerous ribs are usually fractured, frequently in two places (e.g., the axillary line and angles), plus fracture of the sternum and other bony parts. Serious complications such as tension pneumothorax, mediastinal emphysema, injury to major vessels, and mediastinal flutter, and later, empyema, lung abscess, and mediastinitis may result.

General supportive treatment such as oxygen, blood or plasma for shock, removal of bronchial secretions or blood is indicated.

Other complications will be discussed separately.

Penetrating Wounds of the Thorax

Penetrating wounds may be internal from comminuted fracture of ribs or external from stab and gunshot wounds. Intrathoracic complications result from injury to the lung, heart, and great vessels. Among the conditions that may be encountered, the following are the most important and will be discussed individually:

1. Collapse from massive hemorrhage
2. Tension pneumothorax, with or without hemothorax
3. Mediastinal emphysema
4. Pneumopericardium and hemopneumopericardium
5. Laceration or rupture of diaphragm, with injury to the abdominal organs and immediate or subsequent herniation into the thorax

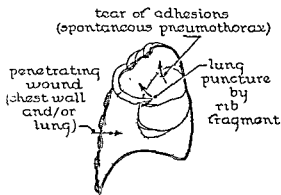
Collapse From Massive Hemorrhage

This may be difficult to distinguish from primary traumatic shock. As a rule, collapse from shock is more rapid than that caused by hemorrhage, unless from some major source. In primary shock, the rapid weak pulse,

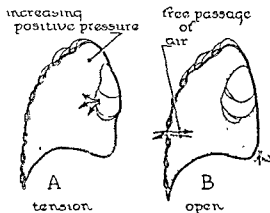
clammy skin, and low blood pressure are present immediately following injury.

Tension Pneumothorax and Hemothorax

These conditions result from any injury perforating the lung tissue when there is a nonadherent pleura. Comminuted rib fractures, with sharp spicules of bone driven into the lung, and stab and gunshot wounds are the most common causes. Tension pneumothorax is the term used to describe air in the pleural



Causes of pneumothorax (after Netter)



Types of pneumothorax

Fig 223.—Causes and types of pneumothorax

cavity under increasing pressure due to a "ball valve" wound of the lung or bronchus, when air leaks out during inspiration but cannot escape during expiration. In this way increasing positive pressure pneumothorax is built up, causing progressive collapse of the lung and mediastinal displacement. Blood or bloody fluid tends to collect in the pleural cavity, adding to the compression.

Symptoms and Signs.—The symptoms and signs are usually diagnostic. There may be subcutaneous emphysema as an indication of lung damage, dyspnea, frothy bloody sputum, cyanosis, rapid weak pulse, lowering of the blood pressure, and hyperresonance with absent breath sounds over the affected side. With accumulating blood or fluid there will be dullness at the base. There may also be evidence of mediastinal shift. All these symptoms and signs will become more acute if the condition is unrelieved, and the patient may go into general cardiorespiratory collapse and die.

In an emergency, this can be simply achieved by inserting a large needle, No. 18 or 16 gauge, through an intercostal space, and the air will be heard to escape under pressure. The needle is then attached to a pneumothorax apparatus, the pleural pressure measured, and further air removed until pressures approach normal. Positive pressures of 15-20 cm of water are often found.

If, after the removal of a significant amount of air (500-1,000 ml), the pleural pressure rises again, a more continuous form of pleural decompression is indicated. An intercostal

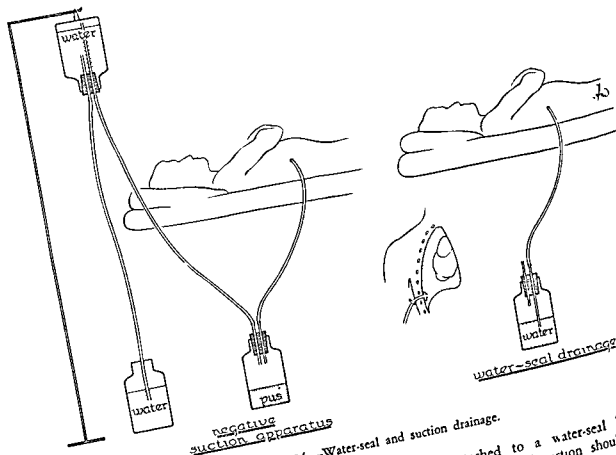


Fig 224—Water-seal and suction drainage.

Diagnosis.—The diagnosis is made by the history and physical examination and confirmed by x-ray, if necessary, but in an acute stage this delay is not justified.

Treatment.—The treatment is to remove the air from the pleural cavity and restore mediastinal pressure relationships to normal.

catheter attached to a water-seal bottle or Wangenstein type of suction should be inserted. In this way positive pressure cannot recur, the opening in the lung will soon seal over, and the lung will be encouraged to expand, at which time the catheter is removed.

Traumatic Hemothorax

This may occur, with or without pneumothorax and from the same general causes as pneumothorax. Wounds of the major vessels such as the aorta, vena cava, or hilar vessels cause such exsanguinating hemorrhage that the actual hemothorax is of secondary importance. However, hemothorax as an established condition requires separate consideration

Pathology.—The presence of a fairly large amount of sterile unclotted blood in the pleural cavity may be relatively asymptomatic unless it compresses the lung or causes enough mediastinal shift to produce respiratory or cardiac embarrassment. However, if allowed to remain in the pleural cavity, two complications may occur: organization and fibrothorax. These are caused by infection and clotting of the blood.

It has been well established that blood in the pleural cavity contaminated with bacteria or exudate from damaged tissues will clot, and soon layers of fibrin over the lung surface are formed. This leads to the condition of *organized fibrothorax*, where the lung is encased in a tough fibrous membrane which seriously impairs its respiratory function.

Diagnosis.—The diagnosis of small amounts of blood in the pleural cavity is difficult. Larger amounts give the characteristic signs of dullness and absent or distant breath sounds. X-ray shows the fluid level if a degree of pneumothorax is present as well.

Treatment.—Immediate aspiration of unclotted hemothorax is only indicated as an emergency measure to relieve respiratory distress, since increasing the negative pressure in the pleural cavity at this stage may only result in continued bleeding. However, in 24-48 hours, when conditions have likely become stabilized, aspiration can be started with safety, about 500 ml being removed daily or every other day until the pleural cavity is dry. Air should not be replaced, since the object is to keep the lung expanded. Penicillin 200,000-500,000 units in 5-10 ml of saline should be instilled at the end of the first aspiration and repeated if infection is suspected.

Clotted hemothorax may require rib resection and removal of clots, as in the open treatment of empyema. If the underlying lung

does not show a tendency to expand after removal of clots, a catheter should be left in the wound, or placed through a separate stab wound, and connected to negative suction, to encourage expansion of the lung. In the late case if the lung is encased in firm fibrous tissue and shows no ability to expand under positive pressure applied by the anesthetist, *decortication* should be undertaken. This is the peeling from the surface of the lung and visceral pleura of organized fibrous layers, thereby freeing the lung and permitting re-expansion. Fissures of the mediastinal and diaphragmatic surfaces should be freed as well.



Fig 225—Marked emphysema of face and neck, resulting from postpneumonecctomy bronchial leak.

Mediastinal Emphysema

This may occur as a result of closed or open thoracic injuries, and is due to a tear in the mediastinal pleura. Straining, coughing, and concussion may also cause it. It is diagnosed occasionally by the presence of crepitation felt above the sternal notch extending into the neck and face and may be detected by x ray. If sufficient pressure is produced, damming back of blood in the great veins occurs, and the patient expires from asphyxia.

The treatment is symptomatic and supportive unless localized pockets of air are demonstrated beneath the sternum, when aspiration often gives great relief.

Pneumopericardium and Pneumohemopericardium

These rare conditions result from lacerations of lung tissue close to the hilus, the air dissecting the fascial planes around the vessels and entering through a tear into the pericardial cavity. The heart shadow is enlarged by x-ray, and air may be demonstrated within the pericardium.

Laceration or Rupture of Diaphragm

Since the dome of the diaphragm rises to the level of the junction of the 4th or 5th rib with the sternum during expiration, the possibility of its being perforated during penetrating wounds of the chest must be considered when they occur below this level. Gunshot and shell fragments are responsible for many wounds of this type in warfare. The possibility of injury or perforation of abdominal organs must always be considered.

Pathology.—The possibilities of abdominal injury vary according to which side of the diaphragm has been penetrated. On the right side the liver covers its undersurface and prevents herniation of the intestines or other structures, and bleeding is usually not severe. On the left side, however, the stomach, kidney, and spleen are all in close contact, and perforation of these organs usually gives rise to serious complications.

Diagnosis.—It is frequently quite difficult to determine whether penetration of the diaphragm and intra-abdominal organs has occurred in combination with wounds of the thorax. Abdominal splinting and rigidity may be present with thoracic injuries alone or may take several hours to appear from abdominal causes such as perforation of the colon. Perforation of the stomach is likely to cause early and acute splinting if the contents are spilled into the peritoneal cavity, also free air may be demonstrated by x-ray if perforation has occurred. However, in the presence of marked abdominal rigidity and rebound pain it is wiser to explore, since the mortality

from well-conducted early exploration is considerably less than that accompanied by late exploration when extensive peritonitis has developed.

Injury to the spleen presents the usual left-sided symptoms, and kidney damage accompanied by blood in the urine. Widespread laceration of the left side of the diaphragm is present, herniation of viscera may take place immediately or at a later date and can be ascertained roentgenologically.

Treatment.—Surgical exploration of both thoracic and abdominal regions is usually indicated. The decision to explore through the chest and diaphragm depends greatly on the individual case.

In the face of serious thoracic wounds with extensive hemothorax, the transthoracic approach will, of course, be indicated in order to restore maximum pulmonary function. Later, exploration of the abdominal organs can be performed. With minor chest symptoms, such as from stab wounds, the abdominal route is usually indicated. Right-sided thoracoabdominal wounds are better approached through the thorax due to the anatomic relationship.

The most convenient double exposure can be obtained by entering the thorax through the 8th interspace and continuing the incision on to the abdomen as far as necessary. The rectus muscle can be retracted medially or sectioned as required. An adequate exposure of all the upper abdominal organs can be obtained in this way.

Open Wounds of Thorax

Etiology.—By open wounds, we mean those in which there exists an opening into the pleural cavity so large that the soft tissues cannot close it off, and there is free exchange of air through the opening. For this reason they are often referred to as sucking wounds. War wounds, shrapnel and shell fragments, severe automobile and industrial accidents are the common causes. Most stab and bullet wounds are not true sucking wounds, as the tissues of the chest wall seal the opening.

Pathology.—With one side of the thorax open to atmospheric pressure, several deleterious events occur to upset the normal cardio-respiratory physiology.

Collapse of lung on the affected side. With an unclosed opening in the pleura, and if there are no adhesions to support the lung, the collapse is usually complete, i.e., 50% of the respiratory capacity will be lost.

Mediastinal flutter. When one lung is collapsed, due to atmospheric pressure in the pleural cavity, the inspiratory pull of the thorax will be unbalanced, and the mediastinum, if mobile, will be drawn across to the good side during inspiration and return to the normal position during expiration. This continual shift of the mediastinum is called *mediastinal flutter* and is a large factor in the production of shock and respiratory embarrassment. It produces impaired function of the remaining lung and further tends to reduce the vital capacity.

Interference with cardiovascular function. Atmospheric pressure in the pleural cavity also impedes the return of blood to the heart by pressure on the vena cava and hilar vessels. Also, shift of the mediastinum occurring suddenly causes a kinking of the great vessels which may further contribute to respiratory distress.

Stagnation of air in the bronchial tree. Since the collapsed lung does not expand in unison with the good lung as in normal respiration, air tends to be sucked out of it during inspiration and forced back again during expiration. Thus there is a backward and forward shift of a certain amount of stale air in the bronchial tree.

Treatment.—Emergency treatment consists of immediate closure of the wound by tightly applied moist pads, a measure which in itself may produce marked improvement in the respiratory difficulty and general condition. Later care under proper conditions includes effective debridement of the wound under positive pressure anesthesia, control of bleeding vessels (intercostal), removal of foreign bodies or rib fragments, and repair of laceration of the lung. The chest wall is closed lightly, with negative suction drainage, after instilling penicillin and streptomycin into the pleural cavity. Tetanus antitoxin and polyvalent antipneumococcus antitoxin are given in accordance with routine surgical principles. The wound must be watched for evidence of infection in the muscle planes and reopened

if necessary. Hemothorax, tension pneumothorax, pneumonitis, lung abscess, and empyema must also be kept in mind as possible complications.

Some surgeons prefer to close the wound tightly and aspirate accumulated fluid and air, instilling penicillin and/or streptomycin with each aspiration. However, negative suction or water-seal drainage removes the fluid continuously and tends to keep the lung expanded; this is a definite advantage.

Blast Injuries of the Thorax

A blast explosion produces a momentary marked increase in air pressure immediately followed by a negative or suction wave. Injury may be sustained during both phases, but it is generally agreed that the initial pressure wave is the more destructive. The same principle acts in immersion blast, affecting, of course, only the immersed parts. The effect of bomb blast varies with the size of the missile and is most severe in the atomic bomb blast.

Pathology.—There is a sudden excessive increase in the intrapulmonary and mediastinal pressures. In the lungs, rupture of the vessels and alveoli fills the area with bloody exudate which seeps into the bronchi and is expectorated as a bloody, frothy sputum. The heart experiences a tamponade effect, squeezing the blood out of the thin-walled auricles into the pulmonary veins and vena cava.

Signs and Symptoms.—There may be a latent period before signs of shock, dyspnea, cough, and bloody expectoration begin. There may also be signs of an acute abdominal or cerebral condition. Atelectasis may be produced by retained secretions.

Treatment.—This consists of complete bed rest, efforts to clear the bronchial tree, and support of shock. Fluid and morphine should be given sparingly. Intranasal oxygen or an oxygen tent is helpful. Occasionally intrabronchial suction will be indicated. Wounds or injury to other systems, of course, should not be overlooked, and appropriate treatment should be given.

Death occurs from hemorrhage, respiratory insufficiency, cerebral injury, cardiac failure, and air embolism.

ACUTE PYOGENIC INFECTIONS OF PLEURA, LUNG, AND MEDIASTINUM

Empyema

Empyema is defined as pus in the pleural space (see also Chapter 30).

Etiology.—The incidence of empyema has markedly decreased since the widespread use of antibiotics in the pneumonias. The most common etiologic organisms are the pneumococcus, streptococcus, and staphylococcus. Other organisms found in mixed or putrid empy-

(5) via the blood stream as part of a septicemia; (6) infection of hemothorax due to any cause.

Pathology.—The pathology produced by an empyema depends on the infecting organism, the amount of effusion produced, and the site of the effusion. Mixed empyemas are far more toxic than those due to a single organism.

Anatomically, an empyema may be diffuse, i.e., involve the whole pleural cavity, or encysted, e.g., interlobar, diaphragmatic, mediastinal, apical, etc.

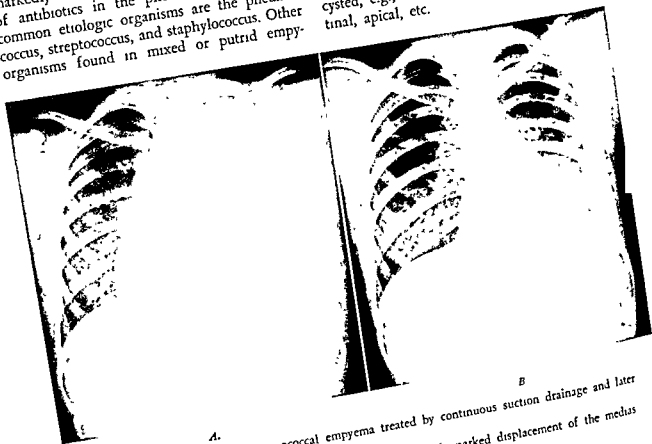


Fig. 226—Massive pneumococcal empyema treated by continuous suction drainage and later by rib resection and open drainage.
A, X-ray before drainage shows massive effusion with marked displacement of the mediastinum to the right.
B, Final result after drainage tube removed.

emas include Vincent's group, Friedlander's bacillus, *Esch coli*, and various anaerobic streptococci. The routes by which the pleural cavity may become infected are (1) from disease of the underlying lung, e.g., pneumonia of all types, lung abscess, bronchiectasis, infarction of the lung, (2) secondary to mediastinal infection, (3) introduction from outside, e.g., stab wound, pneumothorax, etc., (4) extension from a subdiaphragmatic abscess, liver abscess, or chest wall infection.

Empyema necessitatis indicates the relief of an empyema by spontaneous perforation of the chest wall.

The degree of respiratory embarrassment caused by an empyema depends largely on the amount of lung tissue collapsed by the fluid and the degree of mediastinal shift.

Signs and Symptoms.—Empyema usually follows a previously known pneumonia and is diagnosed by continuing fever, dyspnea,

dullness, absent breath sounds and voice sounds, and usually some tympany and egophony in the compressed lung above the pleural fluid.

Diagnosis.—The diagnosis of empyema, apart from the history and physical signs, is confirmed by aspiration and x-ray. Bacteriologic examination of the fluid removed should always be made, as well as notation of the physical characteristics of consistency, color, and odor.

Treatment.—The treatment of empyema has three aims. (1) to relieve the respiratory distress; (2) to reduce toxicity, (3) to pro-

drainage may be continuous or intermittent. *Continuous drainage* is performed by introducing an intercostal catheter into the empyema cavity and attaching it to a Wangenstein type of suction apparatus. In this way the fluid may be removed completely under controlled pressure and speed, forcing the lung to re-expand to fill the empyema space.

In general, the open method is indicated in all thick, purulent empyemas with localized margins and stable mediastinum, while the closed type of drainage is of value in early, massive watery effusions without localization or fixation of the mediastinum.

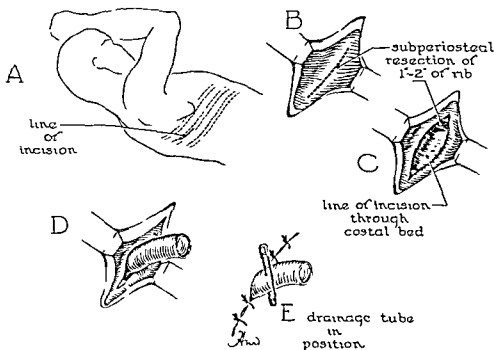


Fig. 227.—Technique of rib resection and open drainage, for empyema

note re expansion of the collapsed lung and maintain it until adhesions have formed and obliterated the empyema cavity. These aims can be accomplished in a variety of ways, depending on the amount and type of fluid present. The various procedures are discussed under the heading of *open* and *closed* drainage.

Open drainage refers to a thoracotomy and the placing of a drainage tube directly into the empyema at its lowest level, thus producing open dependent drainage. *Closed*

Intermittent closed drainage is merely a periodic aspiration of fluid without air replacement, using a closed system. This procedure is usually combined with the instillation of a suitable antibiotic according to the bacterial flora. In a percentage of cases this form of treatment alone may abort or cure an early empyema. However, the late empyemas do not respond well to this form of treatment, and late re-infection and reaccumulation of pus, multiple localization, and organizing fibrothorax are possible complications.

Chronic Empyema, Early and Late

Early chronic empyema may occur from 2-6 weeks after the effusion has become frankly purulent. It is characterized by organization of fibrin layers on the pleural surfaces, and of ordinary treatment in this stage may not bring about complete re-expansion of the lung. At this time, exploration of the thorax, decortication (or peeling off the fibrin layer from the visceral pleura) followed by continuous closed drainage will usually bring about complete re-expansion of the lung.

Late chronic empyema generally results from an improperly treated original empyema. Inadequate drainage, drainage too late in the disease, too early removal of tubes, retention of foreign bodies in the pleural cavity (tubes, gauze, sponges, etc.) and bronchopleural fistula are common causes. In these cases the degree of fibrosis is so marked that decortication is often impossible or too hazardous, and some other means of obliterating the cavity must be attempted. Saucerization of the cavity by removal of overlying ribs and thickened parietal pleura, as in the Schede type of thoracoplasty, is frequently indicated. The use of flap muscle grafts has been advocated to fill deeper cavities.

Lung Abscess

A lung abscess is a localized suppurative focus situated in the lung parenchyma, whereas gangrene of the lung is a more acute process with massive necrosis.

Etiology.—Acute lung abscess may be single or multiple and depends on a variety of etiologic factors

1. Acute pulmonary infections account for a large percentage of all lung abscesses, e.g. all types of pneumonia

2. Aspiration of septic material during operations on the mouth, nose, and throat, may aspiration of foreign bodies by children, may produce obstruction, atelectasis, and abscess.

3. Septic emboli in the bloodstream from other suppurative disease or septicemia, such as osteomyelitis or abdominal infection, may precipitate a lung abscess

4. Lung abscess may be a complication of other diseases, such as bronchiectasis or carcinoma of the lung.

5. Trauma of the lung occasionally results in abscess formation.

A wide bacterial flora is found in most lung abscesses and includes ordinary pyogenic organisms such as streptococci, staphylococci, pneumococci, Vincent's organisms, *H. m. influenzae*, colon bacilli, and numerous anaerobic organisms.

Pathology.—Usually two clinical types are recognized: the putrid and the nonputrid lung abscess. This classification really refers to the acuteness of the pathologic process responsible for the formation of the abscess as a result of the etiologic factors mentioned above. The segment of lung involved becomes atelectatic, and the alveoli are filled with serum and pus cells, and the parenchymal tissue is liquefied and destroyed. The blood vessels are thrombosed; this leads to further death of tissue, and a fan-shaped wedge of inflammation typically spreads out to involve the pleural surface and causes a pleuritis and obliteration of the pleural space over the area. Eventually the infective process may extend to involve the interlobar planes, mediastinal, or diaphragmatic surfaces.

During this phase there is a marked general reaction, and the condition is termed *cavitati*. This stage lasts from 4-6 weeks after formation at this time due to expectoration of necrotic lung tissue through a bronchus. Necrotic lung tissue tends to localize, a firm fibrous ring is formed around it, toxicity lessens, and the state is described as *chronic*. Death may occur from overwhelming toxemia, or perforation into a free pleural cavity may produce a lethal infection of an unprepared pleura. The chronic stage may persist for months or years unless properly treated, producing clubbing of fingers and toes, low-grade devitalizing toxemia, and amyloid degeneration of the liver, spleen, and kidney.

Diagnosis.—The diagnosis is made on the basis of the clinical history and physical signs. The latter, however, may be extremely slight, as in the case of a deep-seated abscess. The sputum is usually characteristic and copious, 75-150 ml daily.

Localization by x-ray is easy, especially when the cavity contains air and fluid. Bronchography is usually not of much value in the diagnosis of lung abscess since Lipiodol rarely

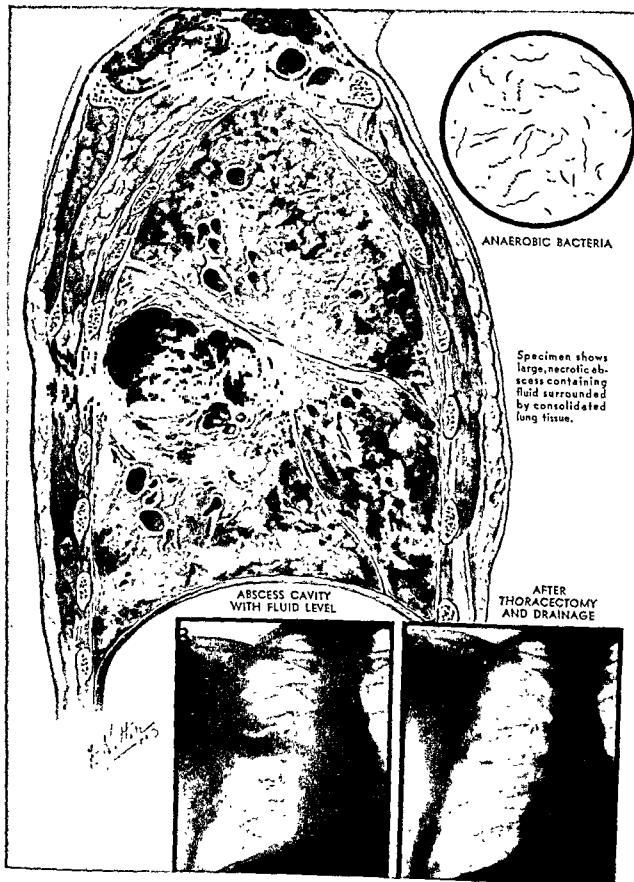


Plate 12.—Putrid Lung Abscess.

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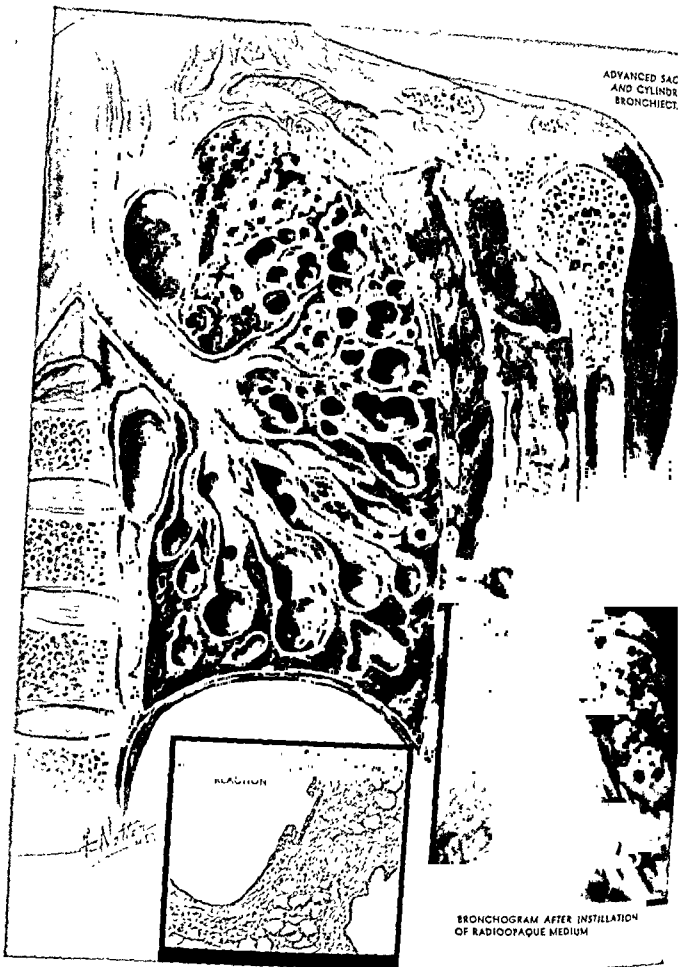


Plate 13.—Marked Saccular Bronchiectasis.

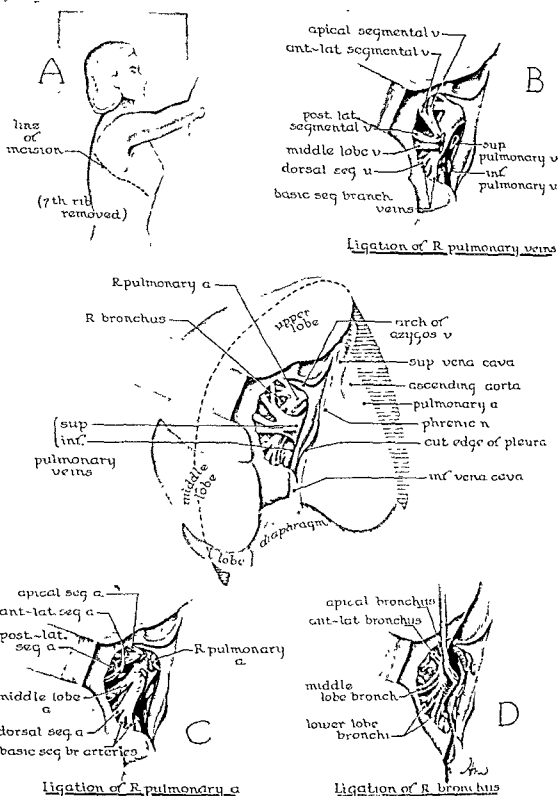


Fig 228 —Hilar anatomy and technique of right pneumonectomy.

third decades of life. There is often a history of repeated attacks of pneumonia or of long-standing sinusitis. The earliest symptom is morning cough and expectoration. This gradually progresses until the patient may bring up several ounces of increasingly fetid sputum during the day. Clubbing of the fingers develops and constitutional symptoms of malaise, cachexia, loss of weight, and pleural pain now appear. Hemoptysis may occur from time to time and may be mistaken for the hemoptysis of tuberculosis. Life becomes a burden with incessant coughing of foul sputum. Fever, chills, and progressive emaciation lead, in the advanced untreated cases, to death during an acute flare-up. Amyloid degeneration is often seen.

Diagnosis.—The history including the diseases of childhood is important. The physical signs are those of impaired resonance at the bases, distinct breath sounds and showers of coarse, bubbling râles. Bronchography is necessary to outline the exact extent and clinical pattern of the condition. Bronchoscopy is usually performed. The blood picture is not characteristic but reflects the toxic and nutritional changes. Tests for amyloid disease may show abnormal retention of dye; the kidney function may be impaired.

Treatment.—In view of the underlying pathologic process, it is obvious that in well-established cases of bronchiectasis medical treatment is only palliative. However, it plays an important part in the preoperative preparation. Postural drainage, bronchoscopic aspiration, high-caloric and high-vitamin diet will improve the general condition. Autogenous vaccines are of value in certain cases. The antibiotics have been a great factor in the treatment of bronchiectasis. Aerosol penicillin and streptomycin will reduce the sputum in many cases and, carried on postoperatively, lessen the incidence of postoperative pneumonia and empyema.

Lobectomy, segmental resection, and pneumonectomy, as indicated in the individual case, offer the only hope of a permanent cure. These operations, in capable hands, now carry a mortality rate as low as that in any other branch of major surgery. Many thoracic clinics are reporting consecutive series of 100 or more lobectomies without a fatality.

The modern technique of this operation is well standardized, the individual ligation of the pulmonary arteries, pulmonary veins, and bronchi being performed at the site of resection. Endotracheal anesthesia is most commonly used.

The posterolateral approach, resecting the 7th or 8th rib, is used for lower lobe lobectomy, and this or the anterior approach between the 3rd and 4th ribs in front for pneumonectomy. Negative suction drainage is generally employed for 24-48 hours postoperatively to remove the serosanguineous effusion and to encourage the remaining lobe or lobes to expand.

Complications.—The complications which may follow lobectomy are (1) early—hemorrhage from a major vessel, shock, atelectasis and postoperative pneumonitis, embolism, and pulmonary thrombosis, and (2) late—bronchopleural fistula, empyema, and wound infection. Rigid attention to operative technique and careful postoperative handling will eliminate many of the complications.

THE SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS

Introduction

This aspect of thoracic surgery originated at the beginning of the present century. For many years progress was slow and erratic, the end results uncertain and discouraging. Finally, the operation of thoracoplasty became firmly established and was responsible for the arrest of many cases of active tuberculosis. In the past 10 years the surgical resection of a tuberculous focus in the lung has become, in most instances, the operation of choice. The future outlook for the complete control of this disease by public health measures is encouraging, but the need for detection and medical and surgical therapy will continue for many years. Tuberculosis will probably remain endemic in our population indefinitely, although there is now less cause for the great fear that has existed in the past. With modern diagnostic aids, medical management, antituberculosis drugs, and surgical procedures, one can expect an excellent chance of cure within a reasonable period of time. Furthermore, the ravages of this disease, exemplified

by some of the tuberculous derelicts in sanitariums, will eventually disappear. Tuberculosis need not reach the stage of total lung destruction and disseminated disease

General Considerations

A number of factors have been responsible for the improvement in management and treatment in recent years. Undoubtedly, the most important development has been the discovery of antimicrobial drugs which are effective against the tubercle bacillus and which control and localize the infection. Streptomycin, the first practical antituberculosis drug, was discovered by Shtatz and Waksman in 1944. Para-aminosalicylic acid (PAS) was introduced in 1946 and is usually used in combination with streptomycin or some of the drugs discovered later. Its greatest advantage is that it can delay or prevent drug resistance by the tubercle bacillus. In 1952 isonicotinic acid hydrazide (isoniazid or INH) was found to be effective against the tubercle bacillus, and this has proved to be a very useful drug. Several other drugs have appeared since the discovery of isoniazid, such as viomycin (1951) and pyriznamide (1952), but all have either toxic or other unfavorable properties which complicate their use.

Treatment with these drugs combined with bed rest and other supportive measures prepares the patient for surgery if his disease process will not heal by medical therapy alone. The antituberculosis drugs localize the main focus of disease, control or eliminate satellite areas of involvement, minimize the amount of pulmonary parenchyma involved, clear the zone around the main focus of exudative reaction, reduce the magnitude of the operation, and preserve a greater percentage of functioning pulmonary tissue.

Pulmonary tuberculosis almost invariably begins as a focal area of disease. Extension may occur from this starting point either by direct invasion or more generally by way of bronchogenic spread. The initial disease should be controlled and confined by the antituberculosis drugs to as small an area as possible. The destructive changes caused by the original infection can be surgically treated when the primary infection has been controlled. Un-

questionably, the antituberculosis drugs have been largely responsible for the marked reduction in mortality and morbidity following resection for this disease and for the increased percentage of sputum-negative cases following surgery.

Many of these patients chronically debilitated by tuberculosis present disturbances in electrolyte balance and in blood volume; such persons require correction in the preoperative and postoperative periods. Likewise adrenal insufficiency may occur, requiring replacement therapy.

It is necessary to stress the importance of the anesthesiologist in the surgery of tuberculosis, as he is not only responsible for the controlled narcosis and relaxation of the patient but must also be expert in replacement therapy and in the maintenance of vital functions and must understand the pharmacologic action of the different agents employed. During the operative procedure he must prevent the contamination of other portions of the same or opposite lung by the spread of tubercle bacilli which occurs when the cough reflex is absent. In this respect, the technique of blocking the bronchus to a diseased lung, lobe, or segment is now a prerequisite for administration of an anesthetic in the presence of tuberculous suppurative disease. This is accomplished by using special blockers that are positioned to exclude the bronchus leading to the diseased site or by introducing special catheters so that the inflated balloon effectively seals and confines the secretions to the diseased area to be removed. Very often the anesthesiologist is called upon to block an entire lung and carry the patient through a major operation on the other lung. At the conclusion of the operation the patient should be conscious and possess an active cough reflex which expels contaminated secretions from the airway and prevents postoperative atelectasis.

The critical assessment of the functional capacity or reserve of the lungs is particularly important in cases of extensive disease. It is necessary to know the amount each lung is contributing to the over-all breathing capacity of the patient. It would be impractical to remove a lung or a lobe and leave the patient so short of breath that normal activity is

seriously restricted. The respiratory physiologist has become an indispensable part of the team for the assessment of these cases. With this knowledge, poor-risk patients with markedly diminished function may be accepted, as many difficulties can be circumvented or alleviated by special measures.

Improved surgical techniques and understanding of the segmental distribution of disease have enabled resections to be confined to the actual site of the disease, thus preserving a maximum amount of normal lung tissue. Complete familiarity with the chest and its mechanics and physiology and the increase in the understanding of cardiovascular lesions and their management have decreased the operative risk markedly in the past few years. Recently there have been newer techniques developed for the collapse therapy of this disease, which have received additional impetus from the use of synthetic materials inserted to provide direct, localized collapse with preservation of maximum pulmonary function.

Pathology

The position of surgery in the treatment of this disease is based on an understanding of the pathologic processes involved. Tuberculosis must always be considered a *systemic* disease in which we recognize and treat certain local lesions that manifest themselves by becoming clinically demonstrable. It follows that the general care of the patient remains fundamental. Surgery is used to assist the healing process or to extirpate disease which cannot be controlled by conservative therapy. Since it must be considered a systemic disease, the recognition of extrapulmonary foci, such as bone and renal tuberculosis, tuberculous meningitis, and miliary tuberculosis, is important.

The profession is indebted to Medlar, the pathologist, who elucidated the different patterns of pulmonary tuberculosis and established the pathologic concepts on which modern treatment is based.

In the lung, tuberculosis invariably begins as a localized area of consolidation, i.e., *tuberculous pneumonitis*. This may be manifested only as a slight chest cold or may simulate a severe toxic pneumonia. The lesion may be so small as to be missed on chest

x-rays, or it may show varying degrees of severity up to complete consolidation of a lobe or a diffuse bronchopneumonia. A tuberculous infection having started as a pneumonitis may terminate in three ways: (1) complete resolution of the pneumonia may occur, leaving no residual x-ray evidence; (2) a more severe infection in a patient with natural resistance may show slow healing with residual scar formation leaving the so-called spot on the lung, or (3) a still more severe infection may result in local death of tissue, associated with the particular type of inflammatory reaction and pus formation which are characteristic of tuberculosis. This is designated *caseous necrosis* and results in the formation of a chronic tuberculous abscess.

The student can easily appreciate that surgical treatment is not concerned with the first two sequelae quoted above. These are healed by the patient's natural resistance, in some cases without treatment and without his awareness of the disease. Many cases with the aid of medical treatment are brought to a state of permanent scar formation and quiescence. Caseation necrosis, persisting or progressing to cavity formation or to solid areas containing tuberculous pus, becomes a surgical problem.

A tuberculous abscess in the lung is of a chronic nature. The contents vary from semisolid to liquid, and the wall is composed of tuberculous granulations which mature slowly into scar tissue. The very nature of the pathologic process involved in the formation of these cavities often results in mechanical problems in healing and so may require a surgical solution. Similar to nonspecific lung abscesses situated near the surface, there is an overlying area of pleuritis which leads to local obliteration of the pleural space by adhesions. The semisolid contents of the cavity may liquefy, gradually erode, and point into an adjacent bronchus and be evacuated by coughing. At this point the patient produces contaminated sputum for the first time and becomes a so-called *open case of tuberculosis*. Sputum examination at this stage will reveal the presence of tubercle bacilli. During this time, heavy contamination may lead to bronchogenic spread to other parts of the same or opposite lung. This spreading may occur by means of cough propulsion, by gravity flow

during sleep, or during periods of severe debility. The closed tuberculous lesion containing liquid or inspissated pus, which appears on x-ray as a completely opaque well-circumscribed shadow, is known as a *tuberculoma*. When such a lesion empties into a bronchus, it becomes partially or completely evacuated, air enters the area previously occupied by the pus, and the presence of a cavity is then easily demonstrable. If air and pus are present in the same cavity, there will be an air-fluid level in the upright position, visible on x-ray. If it is completely evacuated, an air-filled cavity will be noted. A cavity is never stationary, and if the bronchial communication remains open and the walls have not become rigid, the cavity wall moves as the patient breathes. The actual method of healing of cavities is not settled, but for practical purposes, healing occurs only when the space becomes filled and obliterated with granulations. This in turn becomes scar and the walls approximate and fuse, obliterating the infected surface and preventing the continued growth of organisms.

Another factor of importance is the state of the bronchus. *Tuberculous bronchitis* influences the fate of every cavity. The small bronchus which drains the cavity is inevitably involved in the infection. Tuberculous endobronchial disease heals slowly with scar production. The scar contracts, and stenosis develops which may progress to complete bronchial obstruction resulting in atelectasis and failure of drainage of the involved lung. This stenosis may involve segmental, lobar, or main bronchi. If complete obstruction of a draining bronchus does occur, there is no exit for the accumulated caseous material, and a serious chronic condition is created. The failure to recognize active endobronchial tuberculous disease may result in failure of a resection due to the development of a *bronchial fistula*. It is difficult to sever an infected bronchus, suture it, and expect it to heal by primary intention. Many of the bronchial fistulas following resection can be attributed to unrecognized bronchial disease.

It must be realized that all three types of reaction commonly coexist in the same patient, one or more areas of caseation necrosis being

surrounded by zones of fibrosis and exudative reaction. Such areas may clear considerably or completely with medical treatment. Many open positive cavities may persist, but no tubercle bacilli can be found in the sputum despite diligent search. These *open negative* cases require resection, since examination and culture of the cavity wall and contents have usually revealed large numbers of *tubercle bacilli*.

These pathologic changes can be modified and sometimes cleared completely by the administration of antituberculosis drugs. Under their influence pneumonic areas can resolve, tuberculous ulcers in bronchi disappear, and cavities may even heal soundly. In the most advanced lesions, the infective element may be brought under control or even abolished, but the structural defects of the infection remain. Larger cavities persist either open or filled with caseous material, bronchi remain stenosed by scar tissue, and bronchiectasis persists. Such irreversible changes are a constant source of future reactivation and spread of the disease. When this state is reached and the patient's condition is satisfactory, resection is the logical step to remove the major focus of disease.

Surgical Treatment

The function of surgery in the treatment of pulmonary tuberculosis is to create by mechanical means a situation that encourages healing or to remove diseased areas that are damaged beyond repair. Surgical treatment should be deferred until the disease process has been stabilized by medical means and has remained so for a reasonable period. Recently it has been realized that this may be safely performed at an earlier stage with good results. However, this is a matter of judgment, and treatment must be adapted to the individual case.

General Considerations

The proper preparation of the surgical patient is important to prevent serious complications. The following general measures apply to all cases.

1. Systemic measures
2. Local measures
3. Specific measures

Systemic Measures.—The general nutritional state is important. The blood picture should be checked for hemoglobin content, hematocrit, red cell determinations, and NPN. Blood studies should also include the glucose levels, as diabetes and tuberculosis frequently coexist. Determination of the blood volume is important, especially in the debilitated or poor-risk patient. This will serve as a baseline for the control of blood loss and replacement throughout the procedure and in the postoperative phase and will assist greatly in avoiding overtransfusion which is equally as serious as undertransfusion. The electrolytes must be checked regularly when indicated and a stable state of water balance maintained. Antituberculosis drugs should be administered in proper doses and after sensitivity tests. Vitamin therapy is indicated in most cases. The psychologic preparation for the operation and postoperative phase is an accessory measure. Many centers have trained psychiatrists for such purposes. In the postoperative phase, rehabilitation programs and occupational therapy will speed recovery.

Local Measures.—These consist mainly of treatment designed to decrease the amount of sputum, to increase the efficiency of breathing, and to eliminate any obstructive factor in the minor bronchi. Bronchodilators may be administered to decrease the congestion in the mucosa, eliminate bronchospasm, and provide better aeration and drainage for exudative and cavity disease. Postural drainage, suitable for the location of the cavity, is instituted to render the patient as dry as possible before operation. Secondary infecting organisms in the sputum should be cultured, tested for sensitivity, and controlled by suitable antibiotics. The patient should discontinue smoking for 2-3 weeks prior to surgery, to diminish bronchospasm and cough. An adequate preparation of the operative site is necessary to minimize the tendency to wound infection.

Specific Measures.—These include the localization of the major focus of disease by the different x-ray techniques, namely, by plain x-rays in different projections, tomography, and bronchography. Cardiac assessment is obtained if the patient's symptoms and signs so warrant or if he is in the older age group.

Pulmonary function studies are necessary in planning the type and extent of resection and help to prognosticate the result. Prior to resection and every collapse procedure, each patient should have a detailed bronchoscopic examination. Finally, trained physiotherapists should visit the patient preoperatively, to explain that certain muscles are to be cut and to instruct the patient in shoulder girdle movements and in breathing exercises, emphasizing the use of the diaphragm and chest wall. This program continued in the postoperative period reduces the incidence of atelectasis and the failure of expansion, diminishes residual spaces, and minimizes the resultant deformity. An efficient recovery room is desirable for proper postoperative management.

Surgical Procedures

The operations may be grouped into three categories:

1. Drainage operations
2. Collapse or relaxing operations
3. Resection operations

Drainage Operations.—The larger cavities fail to heal because of the mechanical factors present, such as a thick-walled cavity or a bronchial stenosis. Monaldi, an Italian surgeon, popularized a method whereby a catheter was inserted through the chest wall into the cavity which was decompressed by continuous suction. This operation proved unsatisfactory and today is little used. *Empyema* complicating pulmonary tuberculosis may require drainage. This may be accomplished either by the closed water-seal or open methods; and such procedures may be supplemented later by decortication. This procedure removes the thick, external peel on the surface of the lung and lining of the chest wall and allows the underlying lung to expand, become adherent, and thus obliterate the space.

Collapse or Relaxing Operations.—In order to circumvent some of the mechanical factors preventing closure of a cavity, methods have been devised to promote approximation of its walls. *Artificial pneumothorax* is the introduction of air into the pleural space to negate the normal intrapleural pressures and provide concentric collapse of the lung. How-

ever, this is little used today. Likewise, *artificial pneumoperitoneum* has been largely abandoned.

Crush and avulsion of the phrenic nerve, used extensively in the past, is rarely employed. When the phrenic nerve is crushed, recovery from paralysis usually occurs in a period of 4-6 months, although a certain percentage of patients never regain function of the nerve. Complete avulsion of the nerve is no longer practiced. Even simple crushing should be discouraged, because permanent paralysis of the

a thoracoscope, which permits collapse of the lung. This operation along with artificial pneumothorax has become obsolete.

Extrapleural pneumonolysis was designed to produce a selective collapse over the desired area by dissection outside the parietal pleura, to create a space between the inner aspect of the endothoracic fascia and the outer aspect of the parietal pleura. Once the space is created by surgical dissection, it is then maintained by introducing artificial materials such as fat, paraffin wax, paraffin oil,

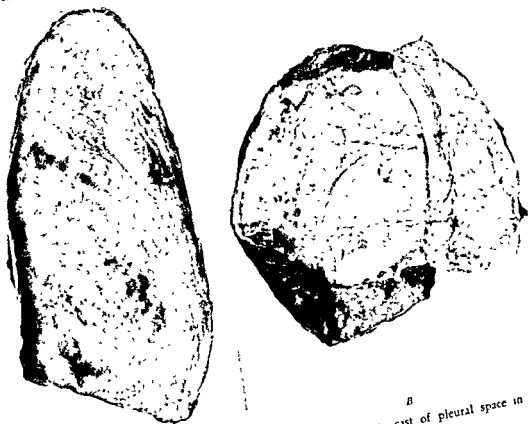


Fig. 229.—Decortication specimen which formed complete cast of pleural space in a case of chronic pleural effusion due to tuberculosis.
A, External surface showing imprints of ribs
B, Opened specimen showing shaggy fibrinous wall after fluid was evacuated

diaphragm deprives the patient of respiratory function which may be required later, particularly if his disease becomes bilateral.

Intrapleural pneumonolysis is used in conjunction with artificial pneumothorax where adhesions between the visceral pleura and the chest wall prevent adequate collapse. The adhesions are cut by a cautery introduced through

and various synthetic materials. The use of such materials for this purpose is known as *plombage*. Air may be introduced under positive pressure into this surgically created space and is known as *extrapleural pneumothorax*. Complications caused by the presence of a foreign body and the difficulties of maintaining the space limit the field of this operation.

Extraperiosteal or extrafascial pneumonolysis with plombage is a method of collapse therapy which involves stripping the periosteum and muscle from the ribs over the required area, so that the soft tissues of the chest wall, including the costal periosteum, collapse inward, depressing the underlying lung. The denuded ribs are not resected, and a packing or plomb is introduced to occupy the space between the ribs and collapsed soft tissues, to maintain the collapse. Regenerated ribs eventually form from the displaced periosteum in the collapsed position. The original ribs may be resected later and the packing removed, or if the packing is inert, it and the original ribs may be left in place. As in the extrapleural plombage, the material employed may be the same, but the newer synthetic materials are preferable. Lucite spheres were popular a few years ago and are still in use. However, some of the newer synthetic spongelike materials, such as Ivalon, seem to be the best agents for these procedures. Special indications for this operation exist where the disease process is limited to the apex of the lung in patients with low pulmonary reserve and where bilateral cavitory disease is present with reduced respiratory function. Contraindications include large peripheral cavities, marked fibrosis at the apex, densely adherent lung at the periphery, and cavities located below the apex. This procedure offers particular advantages. It produces a localized selective collapse, there is a retention of maximal function of normal lung tissue, there is minimal deformity of the chest wall, maximal preservation of chest wall motion without paradoxical movement, and the advantage of a one-stage operation. Complications may occur, such as infection of the extraperiosteal space by tuberculous and nontuberculous organisms, especially in the presence of the foreign body. Bronchopleural fistula and secondary hemorrhage may occur. When used in selected cases, this operation will provide a high percentage (85%) of arrested cases.

For many years, thoracoplasty of various types was the mainstay in the surgical treatment of pulmonary tuberculosis. Since 1950, thoracoplasty has been virtually abandoned in favor of excisional therapy. Thoracoplasty implies the permanent collapse of a portion

of the chest wall by removal of port ribs. The resection of the ribs is subperitoneal, leaving the detached periosteum on the collapsed soft tissue outside of the lung and parietal pleura. This periosteum will eventually regenerate irregular, bony plates in the position of deformity, thus establishing a permanent and irreversible collapse. The amount of collapse varies with the extent of the disease and the number of ribs resected. Usually at least 5 ribs must be resected to obtain useful collapse. However, the number may vary from a maximum of 11 ribs to 6 or 7 as the average. Extensive rib resection results in instability of the affected portion of the chest wall, which reduces the efficiency of respiration because of the resultant paradoxical movement. It interferes in part with the effectiveness of cough, and for these reasons the operation must be performed in stages so that the degree of paradoxical interference with cough efficiency is not so great as to endanger the patient's condition. The usual 6- or 7-rib thoracoplasty is usually accomplished in two stages, resecting 3 or 4 ribs at the first, and the remainder at the second operation. The interval between the procedures must be sufficient to allow stabilization by healing fibrosis of the chest wall from the preceding operation but not so long as to permit rib regeneration which would restore the chest wall and prevent additional collapse following the next stage. It is important to allow two weeks, with a maximum interval of three weeks between stages. It is important to remove the entire posterior portion of the ribs and the transverse processes. Failure to do this leaves a residual deformity beside the bodies of the vertebrae, and the transverse processes. Here, a cavity remains open without complete collapse and results in *thoracoplasty failure*. A refinement to aid the collapse is a procedure known as *apicolysis* which may be added to the thoracoplasty. During the first stage the upper ribs have been resected, the lower portion of the lung is freed by dissection in the extrapleural or the extrafascial plane, and displaced downward toward the hilum in accordance with the principle of combining apicolysis with thoracoplasty to enhance the concentric collapse.

TABLE 18
COLLECTED MORTALITY STATISTICS

SURGEON OR INSTITUTION	YEAR REPORTED	NUMBER OF RESECTIONS			MORTALITY
		STREPTOMYCIN COVERAGE NOT STATED	WITHOUT STREPTOMYCIN COVERAGE	WITH STREPTOMYCIN COVERAGE	
Sweet (Boston)	1946	63			33.0%
Barley (Philadelphia)	1947	80			27.5%
Gale (Madison)	1949	80			2.5%
Jones (Los Angeles)	1950		35	88	28.6% 5.7%
Day, Tuttle, O'Brien (Detroit)	1950			202	6.9%
Munz (Cleveland)	1950			53 consecutive	7.5%
Overholt (Boston)	1952		224	184	10.9% 3.9%
Royal Edward Laurentian Hospital, Montreal	1957			400 consecutive	1.5%

toward the hilum was first advocated by Semb and is widely practiced.

Bilateral tuberculosis may be treated by bilateral thoracoplasty; however, operations of this magnitude may produce a marked reduction in respiratory reserve. Before excisional operations and antituberculosis drugs were employed, a sputum conversion rate of 70-80% and a mortality rate of 2-5% were considered satisfactory.

Thoracoplasty, however, is not indicated for the treatment of solid lesions which cannot be collapsed, tuberculous bronchiectasis, atelectatic lobes, or diseased segments distal to stenosed bronchi. Giant cavities may be closed by thoracoplasty, but not infrequently they are merely reduced in size and still contain tubercle bacilli. Thick-walled cavities may be merely displaced without evidence of closure. Thoracoplasty is wasteful of functioning lung tissue, since it compresses not only the cavity but also adjacent normal tissue. In cases of thoracoplasty for bilateral disease, the resultant loss of healthy lung tissue is poorly tolerated. The inevitable deformity resulting from thoracoplasty and the necessity for multiple operations are other objections to this form of treatment. Thoracoplasty still has a limited use in the treatment of tuberculosis but in the majority of cases has been replaced by newer techniques.

Resection Operations.—In spite of isolated successful attempts to remove tuberculous segments of the lung (Tuffier, 1891; Lowson, 1893), resection operations were not generally adopted because of the excessive morbidity and mortality. Consequently, the safer collapse procedures were employed for many years. Recently, with the discovery of antituberculosis drugs and the improvement of surgical techniques, resection therapy has become the method of choice. The early mortality rate was 25-30%, whereas recent series report a rate of 3% or less (See Table 18.)

Resection surgery includes segmentectomy or wedge resection, lobectomy, and total pneumonectomy if indicated. A unilateral or bilateral combination of lobes or segments may be removed.

Preparation and Assessment for Resection

These patients require preoperative assessment and preparation. The essentials are as follows:

- 1 General measures, including bed rest, nutrition, elimination of secondary infection, and improvement of breathing function by physiotherapy.
- 2 Uninterrupted treatment with antituberculosis drugs in adequate dosage, for a minimum of three months.
- 3 Bronchoscopy is essential. The condition of the bronchial tree and particularly the condition of the mucosa at the proposed line of resection are carefully assessed.
- 4 Preoperative study of pulmonary function is required in patients with far advanced or bilateral disease.

5. Vital functions such as cardiovascular, renal, and adrenal must be assessed. Reduction in the amount of sputum by the use of postural drainage and bronchodilators in aerosol form is indicated.

6. Patients with low pulmonary function may require preoperative or postoperative tracheostomy and/or artificial ventilation.

The *indications* for resection in pulmonary tuberculosis are still controversial. Absolute and relative indications are as follows:

Absolute Indications

- 1 Destroyed lung
- 2 Destroyed lobe
- 3 Thoracoplasty failure
- 4 Bronchostenosis
- 5 Bronchiectasis
- 6 Thick-walled cavities
- 7 Multiple cavities
- 8 Lower lobe cavities
- 9 Tuberculoma
- 10 Residual open cavities

Relative Indications

- 1 Multiple solid foci which are anatomically suitable for resection
- 2 Undetermined lesions where neoplasm cannot be excluded
- 3 Encysted chronic tuberculous empyema
- 4 Bilateral disease (with adequate pulmonary function)

The *operation* may be performed with the patient in the supine, prone, or lateral position. The advantage of the *prone position* is that secretions do not gravitate into other parts of the same or opposite lung. The *supine position* is preferred by some but fails to give as good exposure. The *lateral position* with a posterolateral or an anterolateral incision is generally used. Two venous infusions should be started for forced transfusion of blood if required.

The technique of the operation varies and will not be discussed. Adequate postoperative drainage must be instituted to remove accumulated air, blood, and serum. This is usually accomplished with two chest tubes, one anterior and one posterior, connected to a water-seal drainage or to a suction apparatus.

The postoperative care includes (1) determination and recording of blood pressure, pulse, and respiration, (2) administration of oxygen if required, (3) physiotherapy to encourage respiratory and shoulder girdle movements; (4) sedatives adequate to provide

relief of pain but not to interfere with the cough mechanism; (5) maintenance of the patency of the drainage tubes; (6) administration of antibiotics for tuberculous and pyogenic organisms; (7) care for the bladder and bowel function and gastrointestinal distention; (8) early ambulation.

Convalescence is generally rapid and the patient is usually sitting on the edge of the bed on the 2nd or 3rd day, out of bed on the 3rd or 4th day, and walking and able to go to the bathroom by the 5th day. The skin sutures are removed on the 7th or 8th day, and by this time the lungs should be completely expanded.

As a rule, in the case of minimal disease, a period of three months of antituberculous drug therapy after operation is sufficient, but when there is a fair amount of nodular palpable disease, a minimum of six months is preferable. In cases in which bilateral disease is present, uninterrupted drug therapy should be continued until the second side is operated upon or until the disease is in a state of permanent control.

The *complications* fall into two groups:

General Complications

- 1 Hemorrhage
- 2 Shock
- 3 CO₂ retention (respiratory acidosis)
- 4 Hyperventilation (respiratory alkalosis)
- 5 Gastrointestinal distention and paresis
- 6 Wound infection
- 7 Bladder infection

Specific Complications

- 1 Spread of disease
- 2 Atelectasis
- 3 Hemothorax
- 4 Pneumonia
- 5 Bronchial fistula
- 6 Persistent air leak
- 7 Residual space
- 8 Empyema (tuberculous, pyogenic, or mixed)

Hemorrhage and shock are prevented by adequate estimation of the blood loss and its replacement throughout the procedure. *CO₂ retention* must be anticipated and should be recognized if the typical picture presents. However, this is an uncommon complication, as is also *respiratory alkalosis* from hyperventilation. *Gastrointestinal distention* following chest operations occurs frequently but is generally not as pronounced or prolonged as that

following abdominal operations. *Acute gastric dilatation* requires immediate decompression. *Wound infections* should be minimal. *Bladder infections* may occur, particularly if the patient requires catheterization

To avoid *atelectasis*, particular attention must be paid to the adequate maintenance

of the airways and the encouragement of coughing. Endotracheal suction and bronchoscopy should be employed without hesitation, if an atelectasis persists. In severe exsanguinating internal hemorrhage, it must be suspected that a ligature has slipped from, or cut through, a major vessel. The patient must be

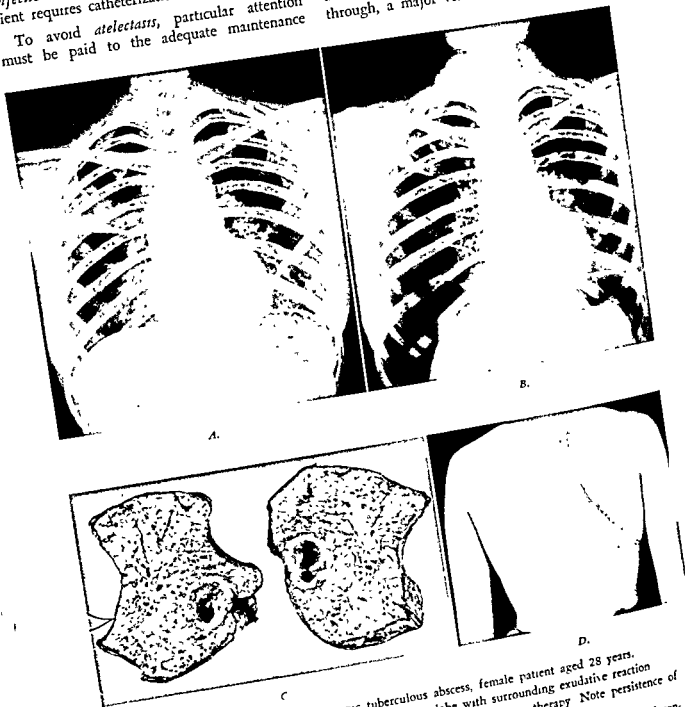


Fig 230—Lobectomy for chronic tuberculous abscess, female patient aged 28 years.
 A, Initial x-ray showing cavity in right upper lobe with surrounding exudative reaction
 B, X-ray 58 weeks after sanatorium and antituberculous drug therapy. Note persistence of cavity. Sputum remained positive. Resection recommended.
 C, Sectioned resected specimen showing large, irregular, thick-walled cavity. Smears of contents were positive.
 D, Scar, 10 days postoperatively.

immediately returned to the operating room, the wound opened, and the bleeding point identified and controlled. In delayed hemothorax, repeated aspirations and the use of enzymatic digestion may be employed. If this is not successful and the lung fails to expand because of the formation of fibrous adhesions, decortication may be necessary.

The *postoperative spread of tuberculosis* should be prevented by adequate preoperative preparation and postoperative management. If it occurs, intensive antibiotic treatment and local measures to ensure adequate evacuation of secretions will usually secure resolution. A *nontuberculous pneumonia* may develop during the postoperative phase and is handled in the usual manner.

Fistula of a main bronchus is a complication and early recognition is necessary. If discovered early the patient is returned to the operating room and the leak located and resutured. Delayed or unrecognized fistulas may be complicated by empyema and are best treated by closed catheter drainage until the acute phase is over. Then some method of eradicating the fistula and empyema space must be undertaken. A *persistent air leak* from a small peripheral bronchus or bronchiole is a troublesome complication following segmental resection. If closure does not occur spontaneously, it is advisable to locate and suture the leak or apply a muscle or flap graft to the opening. At the same time it may be necessary to perform a localized thoracoplasty or plombage to eradicate the residual space. *Tuberculous empyema* is treated by closed catheter drainage in the early phase, followed by later open drainage and thoracoplasty, and/or decortication, if indicated.

The common postoperative complications in a series of cases are listed below

Complications of Resection Surgery

- 1 Progression of pulmonary disease
 - A Early—bronchopneumonia, tuberculous, nontuberculous, or mixed
 - B Late—reactivation of latent or presumably stable disease
- 2 Bronchial fistula
 - A Early—due to inadequate closure or failure to obtain primary healing
 - B Late—due to late ulceration of bronchial stump, generally a sequela of endobronchial tuberculosis

3. Empyema

- A. Early—may be tuberculous, nontuberculous, or mixed
- B. Late—due to latent infection or progression of pleural tuberculosis

4 Miscellaneous

- Immediate, early, and late following resection
 - a. Exsanguinating hemorrhage
 - b. Shock—mild to severe
 - c. Airway obstruction
 - d. Cardiac—arrest, arrhythmia, or failure
 - e. Atelectasis
 - f. Wound infection
 - g. Paralytic ileus or acute gastric dilatation
 - h. Persistent pleural effusion
 - i. Persistent air leak and residual air space

The complications of persistent air leak and residual air space occur particularly after segmental resections and may require secondary operative procedures

Results and Prognosis

The immediate mortality rate following resection for pulmonary tuberculosis should be in the neighborhood of 3.5%. Permanent sputum conversion should occur in 85% or more. Negative reports of direct smear and culture from three direct sputum specimens, three gastric washings, and three laryngeal swabs, carried out over a period of one year postoperatively, are desirable as proof of permanent sputum conversion.

Complete patient rehabilitation should be about 75%. Approximately 15% of the remainder are rehabilitated to part-time work, leaving less than 10% who, although sputum-negative and able to carry on ordinary activities, are not able to earn their livelihood. The relapse rate is uncertain. Late deaths have occurred and are frequently attributed to causes other than active pulmonary tuberculosis. It is possible that in some cases of total lung removal, late deaths may be due to the development of right heart failure subsequent to removal of one lung and the existence of healed fibrosed disease in the other. It would appear that resection for pulmonary tuberculosis is, in most cases, the operation of choice. Its aims are (1) permanent arrest of the disease, (2) preservation of maximum pulmonary function, (3) elimination of residual postoperative deformity, (4) economy of time and expense to the patient, and (5) complete and full rehabilitation to an enjoyable and productive existence.

INTRATHORACIC CYSTS AND TUMORS

Cysts and Tumors of the Pleura

Cysts of the pleura are so rare that they have no practical significance.

Tumors of the pleura may arise from the secreting membrane itself (endotheliomas) or from the subserous layer. The latter are sarcomatous.

It may be difficult to differentiate tumors arising in the periphery of the lung from those of the pleura and chest wall. Pneumothorax may be of value. Bloody pleural effusion usually accompanies malignant tumors of the pleura. Tumor cells may be found on examination of the sediment. Metastatic involvement of the pleura is common.

Mediastinal Cysts and Tumors

Mediastinal cysts and tumors are frequently discovered by survey films and may be asymptomatic. Symptoms are usually caused by pressure on other mediastinal structures, viz., great vessels, esophagus, and nerves. The following classification, modified from Bradford, indicates the wide etiologic range

I. Congenital cysts

1. Dermoid
2. Teratomas
3. Pericardial cysts
4. Bronchial cysts
5. Gastric and esophageal cysts
6. Cystic lymphangioma

II. Acquired cysts

1. Parasitic
2. Neoplastic
3. Cystic hematoma

III. Connective tissue tumors

1. Fibroma
2. Lipoma
3. Leiomyoma
4. Chondroma, chondromyoma
5. Sarcoma

IV. Neurogenic tumors

1. Neurofibroma
2. Ganglioneuroma
3. Neuroblastoma

V. Thymic tumors

1. Benign thymoma
2. Malignant thymoma
3. Thymic cysts

VI. Primary tumors of lymph nodes

1. Lymphosarcoma
2. Hodgkin's
3. Sarcoidosis

VII. Primary and secondary sarcoma

VIII. Primary and secondary carcinoma

IX. Intrathoracic goiter

X. Aneurysm

The signs and symptoms of mediastinal cysts and tumors vary greatly with the etiology, and many are discovered accidentally during routine chest films. However, large tumors or cysts produce congestion of neck veins, substernal pain, dyspnea, dysphagia, cough, hoarseness, and Horner's syndrome from pressure on the trachea, esophagus, great vessels, and nerves, respectively.

The diagnosis may be extremely difficult and only decided at thoracotomy and exploration. X-rays in various diameters, barium swallow, and pneumothorax assist in localizing the tumor. Fluoroscopy, kymography, bronchoscopy, and bronchography give added information in appropriate cases.

Venography and angiocardiology have been advocated recently as a distinct aid in diagnosis, particularly of aneurysms of the aorta or great vessels. Therapeutic x-ray therapy is useful in differentiating lymphosarcoma, which shrinks considerably following this treatment, from benign tumors and cysts which remain unchanged in size.

Treatment.—It is generally agreed that all mediastinal tumors and cysts should be removed surgically if there are no serious contraindications. Many of the benign tumors and cysts undergo malignant changes in later years. Perforation of an infected cyst into a bronchus adds bronchiectasis or lung abscess to the existing condition, and many require lobectomy or pneumonectomy as well.

Anterior mediastinal tumors are exposed by the anterior approach through the appropriate interspace, dividing the costal cartilage above and below. Occasionally, splitting of the sternum vertically and laterally into the 2nd or 3rd interspace is necessary to expose anterior mediastinal tumors such as thymomas.

Posterior mediastinal tumors are reached by the posterolateral approach.

Tumors and Cysts of the Lung

Cysts of the Lung

Cysts of the lung are relatively rare but are being found with increasing frequency, due to wider use of the x-ray. They may be *congenital or acquired*

Congenital Cysts.—

(1) Dermoid cysts and teratomas of the lung are reported but are extremely rare. (2) Simple cysts containing air and lined by various types of epithelium are the most common. There is considerable controversy as to the origin of these cysts, but they may be solitary, bilateral, or multiple. The multiple type may be associated with bronchiectasis or honeycomb lung

Signs, Symptoms, and Diagnosis.—Many cysts remain asymptomatic for years. Infection or rupture with spontaneous pneumothorax may cause the *initial symptoms*. With large multiple cysts, increasing respiratory distress is inevitable

The diagnosis is made by the history and x-rays of the chest. Lipiodol will only occasionally enter a cyst, since the communication with the bronchi may be extremely minute or tortuous

Treatment.—Removal of the solitary cyst by dissection is feasible in some cases. The bronchial communications may be difficult to close. Lobectomy or segmental resection can be performed in certain cases

Acute pneumothorax may be a recurring symptom and is treated by suction drainage maintained for 2-3 weeks if necessary. A mild empyema if it results may cause adhesions and prevent subsequent ruptures

Acquired Cysts.—

Acquired cysts are believed to follow lung infection, pneumonia, and pneumonitis and are not to be confused with cavitation of lung abscess, bronchiectasis, or cancer

Emphysematous bullae or blebs are lung cysts without any epithelial lining and can present the same clinical picture as the true lung cyst.

Benign Tumors of the Lung

Histologically benign tumors are found in about 2% of the total cases of tumors of the bronchial tree. Fibroma, lipoma, adenoma,

chondroma, and hamartoma are some of the benign tumors occasionally reported. However, these tumors may undergo malignant change or cause bronchial obstruction with the sequelae of atelectasis, pneumonitis, lung abscess, and bronchiectasis.

Diagnosis.—Diagnosis of many of these tumors can be made by bronchoscopy and biopsy as they tend to originate in the large bronchi

Treatment.—Local removal by the bronchoscope is only rarely advisable, and lobectomy or pneumonectomy is generally performed, especially if infection of the lung has occurred distally.

Bronchogenic Carcinoma

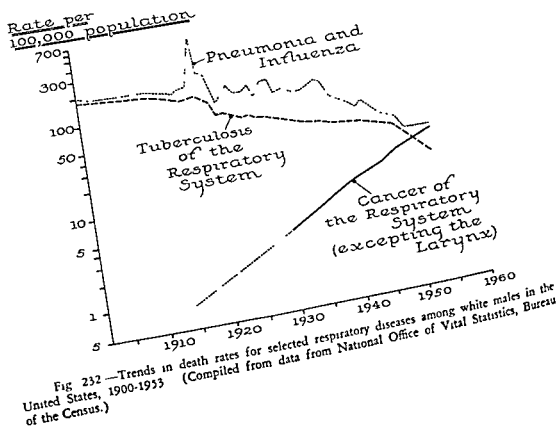
Incidence.—Cancer of the lung is now the commonest visceral cancer in the male population of the United States, where it causes more deaths than any other malignant tumor. Approximately 85% of cases occur in men. It is responsible for approximately 1 in 18 of all male deaths. Today the death rate has more than doubled that of 10 years ago and each year continues to show an increase, as evidenced by clinical investigations, vital statistics, and autopsy records. The incidence is 2-3 times higher in urban than in rural areas, but the relative increase is the same. In females, although the death rate is still comparatively low, the incidence shows a definite rise. The rate of increase among males is $2\frac{1}{2}$ times as great as that in females.

Etiology.—The exact etiology of carcinoma of the lung is not definitely established, but certain theories have recently been advanced which indicate that chronic irritants such as cigarette smoke and industrial pollution may be causative factors

Tobacco Smoking.—During the past 18 years, 16 independent studies in five geographically separate countries have shown that there is a statistical association between smoking and the occurrence of lung cancer. In Britain, investigations in progress for more than five years resulted in the following definite conclusions endorsed by the Medical Research Council with regard to lung cancer in men



Fig 231 —Bronchial adenoma of left lower stem bronchus causing obstructive bronchiectasis pneumonitis





RADIOGRAM BEFORE AND
AFTER ATELECTASIS CAUSED
BY BRONCHOSTENOSIS

Plate 14.—Advanced Bronchogenic Carcinoma With Partial Obstruction of the
Right Main Stem Bronchus and Metastatic Spread.

1. There is a higher mortality in
 - (a) smokers than in nonsmokers (5-15 times)
 - (b) heavy than in light smokers
 - (c) cigarette than in pipe smokers
 - (d) those who continued to smoke than in those who gave it up

2. Heavy smokers had a death rate of more than 40 times that of nonsmokers

3. The current death rate for lung cancer is approximately 1 in 8 for smokers and 1 in 300 for nonsmokers

4. Men who cease to smoke in their early 40's may reduce the likelihood of developing the disease by at least one half

Experimentally, five known carcinogenic substances have been extracted from tobacco smoke which consist largely of microscopic oily droplets held in suspension in the air. The droplets are of such size that they may be taken into the lung tissues and retained there. However, these carcinogenic agents are present in tobacco smoke in such small amounts that there is some doubt that they could be an etiologic factor. Some workers have succeeded in producing tumors in animals by painting concentrated tobacco tar extracts on the skin.

Atmospheric Pollution—Lung cancer is greater in urban than in rural districts. Carcinogenic substances have been identified in coal and industrial smoke and automobile fumes, and therefore it has been postulated and supported by investigation that atmospheric pollution may be a cause of the increase in lung cancer. Nonsmokers in cities have a definitely higher death rate from lung cancer than those in rural areas. In comparison with cigarette smoking it seems likely that atmospheric pollution plays a minor part in causing this disease.

Pathologic Considerations—There are three cell types of bronchogenic carcinoma:

1. Squamous cell carcinoma
2. Adenocarcinoma
3. Anaplastic or undifferentiated carcinoma
 - (a) large cell carcinoma
 - (b) small cell (oat cell) carcinoma

The commonest type encountered is the squamous cell and its anaplastic variant which comprise 80-85% of the whole group, adenocarcinoma constituting the remainder

Alveolar cell carcinoma is an unusual type. In the past, the site of origin has been considered the lining of the alveolar sacs. At present, the evidence favors its origin from the terminal bronchiolar epithelium. For this reason it is now called *peripheral bronchiolar carcinoma*.

All lung cancers arise from the bronchial mucosa. The site in the bronchial tree determines whether the lesion will be centrally or peripherally placed. The tumor may project into the lumen as a polypoid mass, infiltrate the submucosa, or grow extrabronchially. The two latter types produce narrowing or occlusion by either circumferential invasion or extrinsic pressure on the wall by involved lymph nodes. The commonest sites are at the points of bronchial division.

These cancers spread by direct extension, by the lymphatic channels, and by the blood stream. *Involvement of the lymph channels and nodes* is usually the first sign of extension beyond the primary growth. This metastatic process permeates centrally toward the hilum and thence along the previously described routes of lymphatic drainage to the deep cervical nodes. The pleural space drainage is to the anterior and posterior intercostal nodes and to the paravertebral nodes beneath the crura of the diaphragm.

Direct invasion of the chest wall, the diaphragm, the heart, and other mediastinal structures may occur. Involvement of the phrenic nerve results in paralysis of the diaphragm and that of the recurrent laryngeal nerve, in vocal cord paralysis. Direct infiltration of the pleura produces a pleural effusion, which may be sanguineous and reveal exfoliated malignant cells on cytologic examination. When direct extension occurs at the apex, it produces a group of symptoms known as *Pancoast syndrome*, consisting of Horner's syndrome, shoulder girdle and arm pain, and the presence of an apical mass on x-ray examination.

Some authorities believe that definite correlation exists between cell type and prognosis. Squamous cell types carry the best prognosis, with adenocarcinoma in second place. Of the anaplastic group the large cell has a better prognosis than the small cell type.

Symptoms and Diagnosis.—The essential points in the diagnosis of a case of broncho-

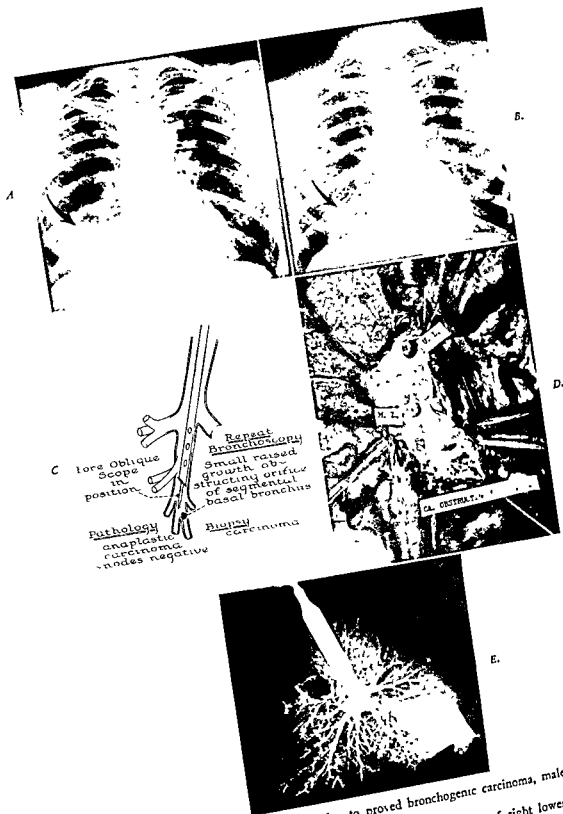


Fig 233—Unresolved pneumonia due to proved bronchogenic carcinoma, male patient 35 years
 A, X-ray showing pneumonic area involving posterior segment of right lower lobe. Cough, chills, and fever present
 B, Persistence of shadow 10 days later despite adequate antibiotic and local measures
 C, Bronchoscopic findings
 D, Resected right lung, medial view, with main bronchi opened. Note small raised growth obstructing posterior basal bronchus U.L., Upper lobe bronchus, M.L., middle lobe bronchus
 E, Injected specimen, medial view, demonstrating obstruction of the corresponding segmental bronchus with distal consolidation.

genic carcinoma have been outlined on pages 441-445. Early diagnosis, which implies a time factor, is essential. Patients who present symptoms of only 2-3 weeks' duration may have widespread metastases from a small primary carcinoma. Others may be treated for weeks or months for virus or atypical pneumonia or possibly pulmonary tuberculosis. Failure of a pneumonic infiltration to clear completely when treated adequately by drug therapy should arouse the suspicion that some mechanical factor may be responsible. A pneumonic process that fails to resolve under medical treatment promptly (3 weeks) should be submitted to intensive investigation, to exclude a *neoplastic* basis.

The symptoms are as follows. *Cough* is present in the majority of cases; either it is a new symptom, or there is a change in the character or severity of the patient's "smoker's cough." *Hemoptysis* is a serious symptom and sign, the history and significance of which should be carefully investigated. *Chest pain*, pleuritic in type, indicates pleural involvement or a pleuritis secondary to an obstructive pneumonitis. Severe constant chest pain is caused by invasion of the chest wall and ribs. *Febrile episodes* typical of pneumonitis are common and may be the first indication that such a lesion is present. *Pneumonias* in men, especially in heavy smokers over the age of 45, should be regarded with suspicion. *Unilateral wheezing*, *hoarseness*, the sudden appearance of *osteoarthropathies* and *neuropathies*, and *shortness of breath* may also focus the attention on this tumor. *Weight loss* is not reliable as a sign of lung cancer.

Investigative procedures should be repeated in suspicious cases which are difficult to diagnose.

Assessment and Management.—Once a diagnosis of bronchogenic carcinoma has been confirmed by histologic studies, the proper management and treatment are important. Of all cases of bronchogenic carcinoma on first observation, one third show evidence of metastases, one third prove to be unresectable on exploratory thoracotomy, and one third are resectable. Surgical removal of the affected lung is the best and only means of effective treatment.

It is possible by careful analysis of the findings to predict unresectability accurately, sparing many patients an exploratory operation. Three investigations are helpful. First, by x-ray, particularly tomography, extensive lymph node involvement may be definitely established. Spread into adjacent structures such as the esophagus contraindicates resection. Rib involvement may be demonstrated but does not necessarily preclude resection. Generally, paralysis of the diaphragm or of a vocal cord, indicating nerve involvement, signifies inoperability. Second, *bronchoscopy* may reveal signs of inoperability, such as extensive tracheal invasion, involvement of both mainstem bronchi, fixation of the carina or its distortion by involved subcarinal nodes. Compression stenosis of a lobar bronchus indicates extensive lymph node encroachment. Resection would be doubtful where this finding is present, particularly in respect to the right upper lobe bronchus. Third, the procedure of *scalene node* and *fat pad biopsy* will often reveal involvement by tumor, contraindicating thoracotomy. On the other hand, a negative biopsy is a favorable finding, necessitating a thoracotomy.

By using the above methods it is possible to increase the resection rate from 30-60%. Follow-up studies indicate that the prognosis in this highly selected group is much better than in the series reported to date which did not receive such preoperative investigation.

A patient should not be denied the benefit of a thoracotomy unless there is unequivocal pathologic evidence that the tumor is inoperable. Many cases in spite of assessment will present a doubtful chance of resectability, nevertheless these should be explored.

Those patients in whom resection has been proved impossible, by investigation or thoracotomy, should receive palliative therapy, which includes a course of radiation or chemical therapy with drugs such as *nitrogen mustard*. Both these methods of treatment may give temporary improvement, but they do not cure the underlying neoplastic process.

The Peripheral Round Nodule.—The asymptomatic peripheral round shadow, discovered accidentally by routine x-ray, presents a diagnostic problem. These nodules may be congenital, parasitic, inflammatory, or neo-

plastic in origin. Statistics show a malignant process in 30-40% of these cases. Investigation is limited to minimal essential points. The radiologic presence of calcium in such a lesion is no longer regarded as a positive sign of a benign process. Thoracotomy, biopsy of the mass by wedge resection, segmental resection, or lobectomy is indicated. The histologic identity of the mass is established by frozen section and dictates the course of treatment.

Surgical Treatment

The surgical treatment of bronchogenic carcinoma is total pneumonectomy with *en bloc* mediastinal lymph node dissection where possible. Lobectomy may be indicated when the carcinoma is peripherally situated. Preparation and management for operation are similar to that outlined in the section on tuberculosis, bearing in mind the general principles of all cancer surgery.

Survival Rate

As total pneumonectomy has only been performed for this disease in recent years, survival statistics have been reported for a correspondingly short period.

Many series have been reported where the 5-year survival rate following resection has ranged from 20-30%. One series reports a 5-year survival rate of 42.9% in patients with localized growths as compared with 6.2% for those with extension. The asymptomatic, localized, peripheral round lesions present a 5-year survival of 76%. In the symptomatic peripheral lesions, however, the survival rate after resection drops to 36%.

The time of survival of the untreated patient is seldom longer than 12-18 months. On the other hand, it has become apparent that a patient who survives pneumonectomy for 2 years has an excellent chance of living 5 years, i.e., the 2- and 5-year survival rates approximate closely, few deaths after 2 years being caused by persistence or recurrence of the tumor.

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Segmental Pulmonary Resection (Illustrates technique of segmental pulmonary resection for a variety of pathological conditions) (1950) (By Richard H. Overholt, M.D., Boston)	20 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn
Crushing Injuries of the Thorax The Treatment of Hemopneumothorax by Temporary Closed Suction Drainage and Pulmonary Decortication (1946) (By Howard K. Gray, M.D., and Robert W. Gentry, M.D., Rochester)		Silent Color	Howard K. Gray, M.D. Rochester, Minn
Lobectomy for Chronic Lung Abscess (1948) (By Alton Ochsner, M.D., New Orleans)	34 min	Silent Color	Alton Ochsner Medical Foundation 3503 Prytania St New Orleans 15, La
Treatment of Thoracic Injuries (Relates the various types of thoracic injuries to the disturbances in pulmonary function which they produce, and demonstrates the principles to be followed in maintaining and/or restoring adequate pulmonary ventilation) (1954) (By Rudolf J. Noer, M.D., Louisville)	31 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn.
Bronchoscopic Cinematography of Bronchial Tumors (1945) (By Paul H. Holinger, M.D., and Ralph G. Rigby, M.D., Chicago)	15 min	Silent Color	The Jacques Holinger Memorial Fund 700 N. Michigan Ave., Chicago 11, Ill
A Bronchoscopic Clinic in Kodachrome (1949) (By Paul H. Holinger, M.D., Kenneth C. Johnston, M.D., and Frank J. Novak III, M.D., Chicago)	31 min	Silent Color	The Jacques Holinger Memorial Fund 700 N. Michigan Ave., Chicago 11, Ill
The Bronchopulmonary Segments, Part I: Anatomy and Bronchoscopy (1955) (By Leo L. Leveridge, M.D., Brooklyn Medical Collaborators: Chevalier Jackson, M.D., John F. Huber, M.D., and Charles M. Norris, M.D., Philadelphia)	31 min	Sound Color	Film Library, Pfizer Laboratories, Div Chas. Pfizer & Co., Inc., 630 Flushing Ave., Brooklyn 6, N. Y.
Lung Cancer The Problem of Early Diagnosis (1954) (By American Cancer Society, New York and National Cancer Institute, Bethesda)	31 min	Sound Color	American Cancer Society 521 W. 57 St., New York 19, N. Y.
A Method of Thoracoplasty for Chronic Empyema (1955) (By Frederick G. Kergin, M.D., Toronto)	20 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn.
Thoracic Surgery, Decortication in the Treatment of Tuberculous Pleuritis PMF 5210 (1952)	25 min	Sound Color	Director, Armed Forces Institute of Pathology, Washington 25, D. C. Attn: Chief, Medical Illustration Service

Chapter 16

Cardiac Surgery

Arthur M. Vineberg, M.D., and David R. Murphy, MD

INTRODUCTION

Since the origin of the human race, the human heart has been the target of enemy weapons. Today it is the subject of the surgeon's scalpel. The possibility that a wounded heart could heal appears to have been discounted until Carrolanus described two patients in whom heart wounds did heal, with recovery. This fact was confirmed by Block in 1882 and by Del Vecchio in 1894, who successfully sutured wounds in the hearts of rabbits and dogs, respectively. This was immediately followed by a successful repair of a human right ventricle by Rehn in 1896.

During the past 15 years innumerable congenital and acquired lesions of the pericardium and great vessels of the heart have been corrected, with low mortality and excellent results. In the past three years development of the extracorporeal circulation, through the pump oxygenator, has enabled the surgeon to open individual heart chambers and repair congenital defects and correct acquired lesions. Recently, through induced cardiac arrest, human hearts have been stopped and successfully restarted. The arrested dry heart affords the opportunity to operate on intracardiac and mural defects with unhurried ease and accuracy.

PREOPERATIVE AND POSTOPERATIVE MANAGEMENT

Diagnostic Methods

In the evaluation of patients with congenital or acquired heart disease, specialized diagnostic methods may be of great assistance.

In fact, preoperative diagnosis is often impossible without some of these studies superimposed on a complete history and thorough physical examination.

X-ray and Fluoroscopy

Careful radiologic examination is of great importance. The cardiac chambers, great vessels, and any abnormal vascular contours can be evaluated and calcific deposits in the valves and pericardium detected.

Angiography

In general, the technique is to opacify the circulation by injecting a radiopaque medium and to follow its course by serial x-rays taken very rapidly. Recently, image-intensifying screens have been used to take motion pictures of the circulation.

Venous Angiocardiography.—In this test the dye is injected into a peripheral vein and its course followed through the heart and great vessels.

Selective Angiocardiography.—The lesion may be outlined more selectively by injecting the medium through a cardiac catheter placed near the suspected area.

Aortography.—A thin catheter is threaded through a systemic artery into the aortic arch and the dye injected. This will show the thoracic aorta and has been used to delineate the coronary arteries. (See Fig. 211.)

Arterial Oxygen Saturation

Arterial blood is analyzed to determine its oxygen content per 100 ml. This figure is

compared with the oxygen content of the same blood when fully saturated (oxygen capacity) to give the *oxygen saturation*. Normal arterial oxygen saturation is 94-99%. Arterial blood below 94% is termed *unsaturated*, although it may not be evident clinically as cyanosis

Cardiac Catheterization

Cardiac catheterization involves the passage of a catheter through a systemic vein into the vena cava and through the right atrium and ventricle into the pulmonary artery. Blood samples are taken and pressure recordings made as the catheter is withdrawn through the right heart chambers. A rise in oxygen saturation of the right heart blood indicates entrance of blood from the left heart. This *left-to-right shunt* may occur at the interatrial, interventricular, or arterial level. The pressure determinations may show abnormal gradients across the tricuspid or pulmonary valves, indicating stenosis or insufficiency, and they may also demonstrate pulmonary hypertension.

Physiologic data on lesions of the aortic and mitral valves may be obtained by left heart catheterization via the left atrium or left ventricle. The left atrium is entered by a needle introduced through the chest wall or through a bronchoscope. A catheter is then threaded through the needle into the chambers of the left heart and into the aorta. As the catheter is withdrawn, measurements are made of the pressures and of the gradients across the valves. Similar observations can be made by direct percutaneous needle puncture of the left ventricle.

Dye Curves

Dye curves may be of great aid in the diagnosis of intracardiac or aortic-pulmonary shunts. The technique involves the injection of a dye such as T-1824 (Evans blue) through a cardiac catheter into the right side of the heart and the subsequent plotting of dye concentration in serial samples removed from a systemic artery. The same information may be obtained by the use of a photoelectric cell attached to the ear lobe.

The dye curves are altered by a right-to-left or a left-to-right shunt. A right-to-left shunt shows an early appearance time and

often a double-peaked curve, one peak produced by the dye that has passed through the defect and one by that which has taken the normal route. The site of the right-to-left shunt can be determined by making dye injections at different points on the right side of the heart. Only those injections proximal to the shunt will give the characteristic curve. Left-to-right shunts have a normal appearance time, but the peak is lower than normal, and the disappearance time is prolonged due to the recirculation of dye through the shunt.

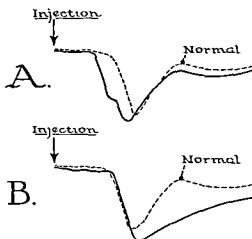


Fig 234.—A, Dye curve showing right-to-left shunt, demonstrating early appearance time as compared to normal.

B, Dye curve showing left-to-right shunt, demonstrating prolonged disappearance time.

(Courtesy The Montreal Children's Hospital)

Electrocardiogram

Multiple lead electrocardiographic studies at rest and after exercise are of great importance and help in detecting individual chamber enlargement, dilatation and/or hypertrophy, as well as the presence of myocardial ischemia or infarction.

Exercise Test

The Masters' two-step test and treadmill give objective evidence of exercise tolerance.

Preoperative Preparation

This must include not only the *careful* and *accurate* establishment of the diagnosis but also detailed investigations to detect other disease, which in itself may be incurable or

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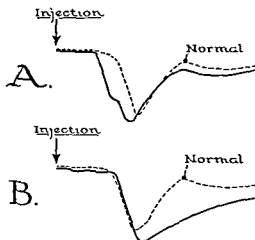


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Preoperative Preparation

This must include not only the *careful* and *accurate* establishment of the diagnosis but also detailed investigations to detect other disease, which in itself may be incurable or

alter the results of cardiac surgery. Long-standing heart disease may affect many organs which must be fully evaluated.

Preoperative Controls.—Certain studies aid greatly in postoperative management.

1 Daily records of pulse and blood pressure set a base line for maintenance during and after operation.

2. A record of daily fluid intake and output shows the effectiveness of control of heart failure and forms a basis for postoperative fluid administration. Changes in the patient's weight from day to day will confirm this.

3 Blood electrolytes should be estimated, particularly in patients with heart failure who have been treated by diuretics and low-salt diet.

4. A coagulogram may reveal altered prothrombin activity due to liver changes caused by venous congestion or hypotension.

5. Total and fractional blood volumes should be measured. Elevated blood volumes markedly increase the hazards of cardiac surgery. Alterations from the normal should therefore be corrected when possible before operation.

Control of Heart Failure.—Heart failure, whether of the right or left ventricle, must be controlled preoperatively. The *wet patient* is a poor operative risk. Satisfactory correction of heart failure is clinically manifested when the patient can sleep in the horizontal position without distress.

Search for Foci of Infection.—Foci of infection, particularly in the mouth and skin, must be noted and treated.

Carotid Artery Compression.—With the patient lying down, the carotids above the carotid sinus are compressed until he becomes dizzy, and this is repeated daily until tolerance is at least 5 minutes.

Psychologic Factors.—Any patient who submits to an operation is worried and anxious, even if this state is not noticeable. All personnel must work to reassure the patient. Careful sedation, avoiding hypotension and hypopnea, is helpful. It is of particular importance to avoid a nerve-racking wait in the operating room while the anesthetist prepares his drugs, and a terrifying jumble of impressions and medical words drifts into the patient's sedated brain. The patient with valvular

disease may go into failure; the patient with coronary artery disease may suffer infarction.

Postoperative Care

The care of the postoperative heart patient presents two facets: (1) the care of a thoracotomy, requiring rapid re-expansion of the lungs, and (2) the treatment of the cardiovascular system.

Nursing Care

This involves the following in addition to routine care:

1. Frequent regular recording of blood pressure and heart rate, both apical and radial.

2. Maintenance of blood pressure where necessary by regulation of rate of continuously administered intravenous vasopressors.

3. Massage of chest drainage tube or tubes every half hour to avoid blockage by clotting, and watching for excessive blood loss through the drainage tube.

4. Maintenance of Wangenstein drainage of stomach when used.

5. Administration of oxygen and assistance of patient with deep-breathing exercises, throat aspiration, and coughing.

6. Movement of patient from side to side; early movement and gentle massage of legs.

Medical Care

Postoperative heart patients should be returned to a recovery room where they are kept until the anesthetic staff and medical surgical team agree that they are fit for return to the ward. The quantity of fluid to be administered and the level at which blood pressure should be maintained are based upon preoperative blood pressure and fluid balance observations. Sedatives and cardiac drugs such as digitalis are given intramuscularly. For patients in whom maintenance of blood pressure is difficult, a blood volume study may reveal need for transfusion. All orders should be clearly written and the nurses' duties carefully explained.

In general, the first six hours following operations for acquired valvular disease or closed heart procedures for congenital defects present no more difficulties than pulmonary operations.

Patients who have undergone major surgery for coronary artery heart disease or who have had treatment for congenital cardiac lesions, using the pump oxygenator, require maintenance in the supine position, without turning, for much longer periods, as early movement frequently causes sudden hypotension and cardiac irregularities.

Consalescent Care

Despite surgical correction of a congenital or valvular defect, the heart does not become normal immediately. Weeks and sometimes months are needed for the heart to readjust itself. The same is true for coronary heart disease. Rest and supportive measures may be necessary for a considerable period of time.

MANAGEMENT IN THE OPERATING ROOM

It is wise to anesthetize the patient in a pre-op room rather than in the operating room. Precautions to maintain blood pressure during induction must be taken. Once the patient is anesthetized, at least three intravenous entries should be started to ensure rapid delivery of blood if necessary. In coronary artery patients the maintenance of blood pressure during the entire procedure and in the postoperative period is so essential that an intravenous cannula or catheter is tied into a vein in the leg or arm.

A *cardioscope* and *electrocardiogram* are connected to the patient so that the character of the heart contractions is visible at all times. In poor-risk patients who have had preoperative cerebral emboli, and in cases where the pump oxygenator is used, continuous recording of brain waves by the EEG machine indicates the efficiency of the circulation.

Cerebral anoxia is quickly reflected in the EEG and in most instances can be rapidly remedied. The efficiency of cardiac massage when cardiac arrest occurs, of pulmonary ventilation, bilateral carotid compression, and of the pump oxygenator is shown by the EEG tracing. A photocell oximeter attached to the patient's ear lobe acts as another monitor of oxygen saturation.

Pressure recording apparatus is used for measurement of intracardiac pressures. Continuous recording of intra-arterial pressure is helpful in aortic valve and open heart surgery. Multipanel electronic pressure recorders are useful for this purpose. Open heart surgery requires facilities for rapid determination of pH, CO_2 , and hemolysis.

Blood Loss.—Because of the narrow range of safety in fluid balance, estimation of blood loss is essential. A reasonably accurate method is the weighing of blood-soaked sponges (gravimetric method). Techniques for continuous measurement of blood volume are still experimental.

CARDIAC ARREST

Definition.—Cardiac arrest means cessation of effective heartbeat due to cardiac asystole or ventricular fibrillation during any operation. By common usage the term has been expanded to include postoperative death and cardiac arrest from purely medical causes.

Incidence.—Reed and Hunt report an incidence of cardiac arrest in the operating room of 1:2,354 operations. It occurs most frequently in children, and the cardiac surgeon operating on damaged hearts will naturally encounter it most often.

Predisposing Factors

I Myocardial hypoxia

A Inadequate oxygenation

1 Preoperative

- a Cyanotic heart disease
- b Failing heart
- c Pulmonary disease and edema
- d Oversedation

2 During operation

- a Obstructed airway—poor pulmonary ventilation
- b Overdose of anesthetic
- c CO_2 retention

B. Inadequate hemoglobin

1. Preoperative

- a Anemia, secondary or primary

2 During operation

- a Shock
- b Hemorrhage

C. Inadequate coronary artery blood flow

1. Preoperative

- a Systemic hypotension—shock, hemorrhage

- b Coronary artery disease

Treatment

Stage I

2. During operation
 - a. Systemic hypotension
 - b. Hemorrhage
 - c. Drugs
 - d. Low cardiac output
 - e. Cardiac displacement
 - f. Coronary artery disease

II Other Factors

- A. Myocardial irritability occurring in coronary artery insufficiency and during administration of certain drugs, e.g., chloroform
- B. Vasovagal reflex and hypoxia
- C. Marked electrolyte imbalance and CO₂ retention

All factors mentioned may play a part in postoperative cardiac arrest.

A knowledge and the avoidance of these factors which may predispose to cardiac arrest may help to prevent a catastrophe in the operating room.

Proper oxygenation of the tissues under anesthesia is the responsibility of the anesthesiologist who, along with the surgeon, must be constantly on the alert and must give conscientious attention to details of drugs, airways, and ventilation (see Chapter 8).

Maintenance of Blood Pressure.—The heart rate and blood pressure reflect very rapidly the presence of many factors said to predispose to cardiac arrest. General tissue anoxia, due to such factors as poor ventilation and drugs, is at first reflected by a rise in blood pressure with comparatively slow pulse rate, followed rapidly by a blood pressure decline which may lead to cardiac arrest if not corrected. Generally a diminution of blood pressure below a systolic level of 80-90 mm Hg decreases coronary artery flow with resultant myocardial ischemia and irritability. It is advisable to keep blood pressure levels within 20-30 mm. Hg of the average preoperative blood pressure level.

Recognition of Cardiac Arrest.—The two cardinal signs are the absence of a palpable pulse in major vessels and the disappearance of an audible blood pressure. *When these occur, open the chest.*

Don't waste time by listening for inaudible heart sounds or waiting for an ECG tracing. Don't inject drugs blindly into the heart or intravenously. Don't try to massage through the diaphragm. Don't worry about sepsis or skin preparation. Institute Stage I immediately. *You have only 3 minutes.*

Emergency Action

1. Adequate oxygenation of the blood
2. Restoration of circulating blood volume
3. Transport of oxygenated blood to vital centers.

Anesthetist

1. Notify the surgeon of cardiac arrest.
2. Note time.
3. Insert intratracheal tube and expand lung with oxygen. Use the breathing bag or mechanical respirator
4. Lower head of table
5. If blood loss has been excessive, start intravenous or intra-arterial transfusion
6. Check effectiveness of cardiac massage, blood pressure, and carotid pulse.

Surgeon

1. Check pulse in aorta or large vessels
2. If absent, make an anterior incision in 5th left intercostal space. Don't worry about bleeders.
3. Insert the right hand and squeeze the heart against the sternum. Release and compress rhythmically for several seconds
4. Pause to cut costal cartilages and insert rib spreader. Resume massage through the intact pericardium.
5. Pause to open pericardium longitudinally with each cardiac compression and fill between squeezes. Failure of heart to fill may be caused by (a) low blood volume (in which case, transfuse rapidly) and (b) Trendelenburg position insufficiently acute
6. Grasp heart between the palms of both hands and commence rhythmic compression from apex to base at 60-70 beats per minute

The emergency is over. With proper cardiac massage an adequate flow of blood can be maintained indefinitely.

Stage II

For Restoration of Heartbeat

1. Massage steadily and forcibly. Have anesthesiologist check pupils, blood pressure, and carotid pulse
2. Make certain that the ventricles are emptied with each cardiac compression and fill between squeezes. Failure of heart to fill may be caused by (a) low blood volume (in which case, transfuse rapidly) and (b) Trendelenburg position insufficiently acute

Increase angle to augment drainage of blood from feet and viscera.

3. Treat the flaccid heart muscle by injecting 3-5 ml. of 10% calcium gluconate into the left ventricle and by quickly massaging it through the coronary circulation.
4. Treat ventricular fibrillation, which can only be detected by opening the pericardium. This is one reason why cardiac massage performed through the abdomen may be ineffective in restoring heartbeat. Massage until the heart muscle is pink and the ventricles are well emptied; then apply the electric defibrillator electrodes.

2. Speed at which cardiac massage is started. After 3-3½ minutes irreversible cerebral changes occur. The return of a heartbeat with a normal blood pressure often occurs but the patient either dies in a few days or survives for many months or years as a decerebrate.

3. Effectiveness of massage. Unless the massage of the ventricles is sufficiently effective to provide a blood pressure of 60-80, the results will be poor.

In 1,200 cases collected by Stephenson, Reid, and Hinton a permanent survival rate of 28% was achieved. The prognosis was as good in

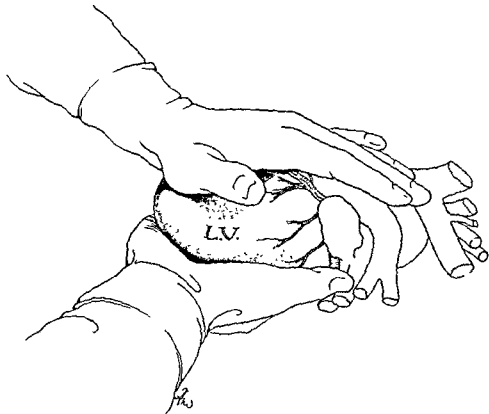


Fig. 235.—Diagram illustrating correct method of massaging the heart between the palms of the hands which enclose the two ventricles. The use of fingers and thumbs is ineffectual and leads to perforation of the ventricles. Drawing shows heart of patient postured in right lateral position and exposed through a left 5th interspace lateral thoracotomy, which is preferable. In emergency, an anterior left 5th interspace thoracotomy may be used.

Prognosis

This depends upon the following.

1. Cause of cardiac arrest. Functional cardiac arrest has a higher recovery rate than if the cause is organic

adults as in children. In the group that died, 87% did so within the first 27 hours, and only 3% after surviving for 6 days. In the entire group there were only eight so-called decerebrates who lived despite severe brain damage.

OPEN HEART SURGERY

The term *open heart surgery* is applied when operations are performed under direct vision, in a relatively bloodless field within the chambers of the heart or great vessels. To permit this type of operation, the venous inflow to the heart must be either completely obstructed or detoured about the heart by an extracorporeal heart-lung apparatus. Circulatory arrest by caval occlusion is tolerated for only a short time (about 3 minutes), but, by lowering body temperature the time limit can be extended sufficiently to permit certain intracardiac operations.

Hypothermia and Total Caval Obstruction

General body hypothermia reduces the metabolism of vital centers so that they can tolerate longer periods of circulatory arrest. The normothermic safety margin of 3 minutes is thus extended to 6-8 minutes at 30° C.

The advantage of hypothermia lies in its simplicity and in the small amount of special equipment necessary. The two main disadvantages are as follows: (1) the time limit of 6-8 minutes forces the surgeon to work under pressure and complex defects cannot be adequately repaired, and (2) the myocardial irritability and ventricular fibrillation become more frequent as the temperature drops. However, in the usual range of 28-30° C, ventricular fibrillation is much less frequent.

Hypothermia can be produced by *external* (surface) or *internal cooling*. Surface cooling is the usual technique and is accomplished by immersing the patient in an ice bath (Swan) or by refrigeration blankets (Bigelow). Internal hypothermia is less usual and is secured by cooling the blood (Brock), or by putting ice cubes in the thoracic cavity (Blades).

Indications.—The indications for the use of hypothermia to perform operations upon the open heart are still controversial. It is a satisfactory method for the repair of interatrial septal defects of the secundum type and for the opening of a pure stenotic pulmonary valve in infants and children. However, equally good results have been obtained by the use of the Gross well for atrial septal defects.

Hypothermia introduces a number of physiologic changes which may be harmful and which are at present not too well understood. Infants and children seem to tolerate the procedure better than adults. In some cases the working time allowed the surgeon is far too short to permit a satisfactory pro-

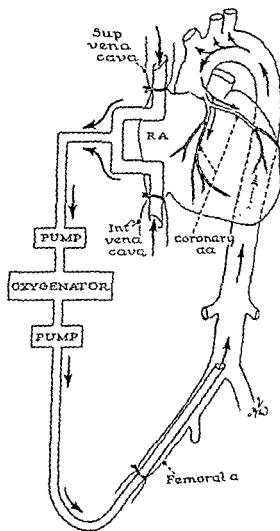


Fig. 236.—Diagram demonstrating extracorporeal circuit. The venous blood is removed from the venae cavae and pumped through an oxygenator, and the oxygenated blood is returned into the femoral artery. (The aorta has been foreshortened for diagrammatic purposes.)

cedure. However, in closed heart operations on poor-risk patients with either acquired or congenital heart disease, hypothermia may offer a considerable margin of safety because of the resultant lowered metabolic requirements.

Extracorporeal Heart-Lung Apparatus

The substitution of the human heart and lungs by a machine which perfuses the body with oxygenated blood to keep the patient alive while his diseased heart is repaired has long been the dream of those interested in heart surgery. The original apparatus was developed by Gibbon, who also performed the first operation on a human heart, using an apparatus of his own design.

There are several machines in use clinically today. They all have the same two basic components, i.e., (1) a pumping system to propel the blood to and from the apparatus, and (2) an oxygenator or artificial lung. It is with respect to the oxygenator, in particular, that the various machines differ. In the apparatus perfected by Lillehei and DeWall, the venous blood is oxygenated by bubbling oxygen through it. Other oxygenators expose, to a flow of oxygen, a large surface area on which a film of blood is produced. A third type of oxygenator in use today interposes a plastic membrane between blood and oxygen, a substitute alveolar membrane.

These machines have been used to repair a wide range of cardiac defects under direct vision. However, they are not entirely satisfactory and are still being modified.

SURGERY OF CONGENITAL MALFORMATIONS

CLASSIFICATION

There is still no better classification of congenital heart diseases than that of Abbott, which divides them into three classes.

Acyanotic Group.—This group includes patients in whom no abnormal communication exists between the two circulations but in whom the anomaly is liable to become the seat of strain, e.g., anomalies of the aortic arch and coarctation of the aorta.

Cyanose Tardive Group.—In this group patients have a predominant left-to-right shunt, which, however, may become reversed, producing cyanosis. This reversal occurs when the pressure on the pulmonary side of the defect exceeds that on the systemic side. In most patients with a patent ductus arteriosus, blood flows from systemic (high pressure) to pul-

monic (low pressure) circuits, so there is no cause for cyanosis. However, in the presence of heart failure or when the pulmonary artery pressure exceeds the systemic pressure, cyanosis can occur.

Cyanotic Group.—In cyanotic heart conditions, of which there are many types, the fundamental feature is the presence of venous blood in the aorta and its branches. This is due usually to some abnormal arrangement by which blood from the right ventricle gains access to the aorta (a right-to-left shunt).

Successful forms of treatment are available for patent ductus arteriosus, coarctation of the aorta, anomalies of the aortic arch, atrial septal defect, ventricular septal defect, tetralogy of Fallot, congenital aortic valve stenosis, sub-aortic stenosis, and pure pulmonary stenosis. Patients with total anomalous pulmonary venous return, tricuspid atresia, ostium atrio-ventriculare commune, and ostium primum interatrial defects are operable but the risk is greater. A satisfactory operation for transposition of the great vessels has yet to be devised, but the circulatory physiology of these patients can be improved by operation.

PATENT DUCTUS ARTERIOSUS

Embryology and Anatomy.—During fetal development the ductus arteriosus serves to conduct blood from the pulmonary artery to the aorta. This permits a large portion of blood issuing from the right ventricle to pass directly to the descending aorta, without traversing un-aerated lung tissue. Closure of the ductus arteriosus following birth normally occurs in two stages. Animal experiments indicate that the first stage or functional closure is generally complete within a few minutes after birth and is due to contraction of abundant muscular elements within its wall. This is followed by the second stage, a gradual fibrous obliteration of the ductus to form the ligamentum arteriosum. Investigation suggests that an adequate oxygen saturation of the systemic blood is necessary to initiate the first stage. The brief equalization of pressure between the systemic and pulmonary arterial systems following birth is considered by some to be important in allowing the muscular elements of this vessel to contract and obliterate

CARDIAC SURGERY

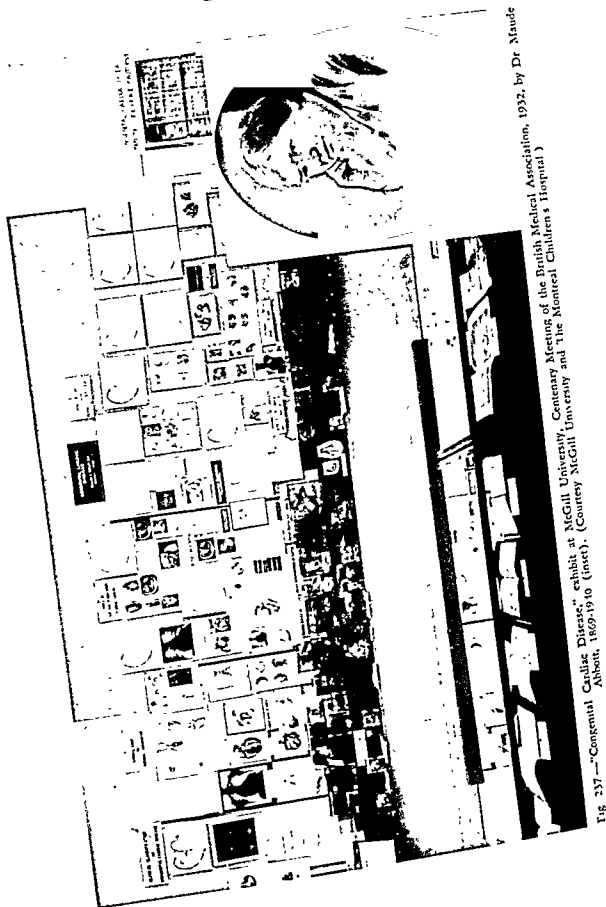


Fig 237

— "Congenital Cardiac Disease," exhibit at McGill University, Centenary Meeting of the British Medical Association, 1932, by Dr. Maude Abbott, 1869-1910 (inset). (Courtesy McGill University and The Montreal Children's Hospital)

the lumen. It has been suggested, therefore, that patency of the ductus arteriosus should not be considered as an anatomic error of development but rather as the result of an abnormal physiologic process. Christie's study of routine autopsies revealed that in 95% of specimens the ductus arteriosus was closed by the 12th week of life and in 99% by the end of the first year.

The ductus arteriosus is a funnel-shaped vessel originating from the aorta, just below the origin of the left subclavian artery, and joining the main pulmonary artery just to the left of its bifurcation. It is commonly approximately 1 cm in length but may be only represented by a fissure between the two vessels. Its diameter varies considerably.

Physiopathology.—Patency of this vessel after birth permits oxygenated blood to flow from the aorta to the pulmonary artery. The volume of blood shunted depends upon the size of the ductus and the pressure differential between the aorta and the pulmonary artery. Since the pressure within the aorta is normally higher than that within the pulmonary artery, the shunt of blood may be high and has been estimated as high as 80% of the left ventricular output. As the blood shunted has been oxygenated, its recirculation through the lungs serves no useful purpose but places a strain upon the left ventricle, which must pump considerably more blood than normal. Persistence of this shunt may result in an elevation of pulmonary artery pressure (pulmonary hypertension). The physiopathology then becomes more complex.

Diagnosis.—This should rarely present difficulty. Patients are usually of normal growth and stature, although underdevelopment may be observed. Frequently there are no complaints, and its presence becomes evident only during routine physical examination. Symptoms of palpitation, shortness of breath, and easy fatigability are sometimes elicited.

The characteristic physical finding is a murmur, best heard in the 2nd intercostal space, just to the left of the sternum. This is continuous throughout all phases of the cardiac cycle and has been termed "machinery-like." A palpable thrill is usually evident. An increased pulse pressure due to a low diastolic blood pressure is a common finding.

Although roentgenologic examination may reveal no abnormality, it is likely to demonstrate cardiac enlargement, a prominent main pulmonary artery, and increased pulmonary vascular markings, the latter being due to the increased flow of blood through the pulmonary vascular bed. The electrocardiogram may be normal but often reveals a left ventricular preponderance.

Complications.—The complications encountered are subacute bacterial endarteritis, aneurysm formation, cardiac failure, and pulmonary hypertension. Subacute bacterial endarteritis is most likely to affect the pulmonic end of the ductus and occurs most frequently after tooth extraction or secondary to other foci of infection. Degenerative disease may occur within the ductus wall with aneurysm formation. Left ventricular failure is encountered when the shunt of blood from aorta to pulmonary artery places too great a strain upon that organ. Pulmonary hypertension develops either as a result of a high flow of blood through the pulmonary vasculature or following pulmonary vascular changes (see section on pulmonary hypertension). Should pulmonary hypertension occur, right ventricular strain becomes evident.

Treatment.—The treatment is surgical obliteration, which can be accomplished by ligation in continuity or division of the ductus between clamps, and suture of the divided ends. The former technique is satisfactory for the majority of patients. If, however, the ductus is unusually wide and especially if it is thick-walled, division is advisable. The ductus is exposed through a left thoracotomy. The lung is deflected caudad, and a flap of mediastinal pleura raised off the aorta and lifted medially. The recurrent branch of the left vagus nerve, which courses below the ductus, serves as an identifying landmark and must not be traumatized. A pouch of pericardium overlying the lateral surface of the ductus is dissected off. The ductus is then ligated in continuity or divided between clamps and each of its ends oversewn. The operative mortality is less than 1%. Since the patient's cardiovascular physiology is normal following operation, resumption of full activities is permitted after one month.

Atypical Ductus Arteriosus

The majority of patients with a patent ductus arteriosus present typical clinical findings, and the diagnosis is not difficult. In some patients, however, the typical continuous murmur is not demonstrable, a systolic murmur alone being present. This occurs when the pressure within the pulmonary artery is sufficiently high that the diastolic pressure within it approximates the aortic diastolic pressure. Blood flows from aorta to pulmonary artery during systole with little or none during diastole.

Since the diagnosis of patent ductus arteriosus under such circumstances cannot be established by clinical examination alone, special investigation is necessary. At heart catheterization, the finding of an increased oxygen saturation within the pulmonary artery compared to the right ventricle is diagnostic.

The ductus arteriosus may also be visualized by aortography. With this technique, 35% Diodrast is injected rapidly toward the aorta by means of a catheter placed into the left carotid or brachial artery. In the presence of a ductus arteriosus the pulmonary artery is opacified simultaneously with the aorta. Catheterization, however, is used more often since the presence of complicating malformations and the associated altered hemodynamics can be better clarified by this means. It can be used successfully even in very small infants.

The circulatory physiology is seriously impaired. It is not surprising that patients in the infant age period are often seen and that they are usually retarded in growth and development. A history of repeated pulmonary infections is often given. A frequent finding is the presence of cardiac failure, which is often resistant to the usual medical management. Surgical intervention is usually lifesaving but may be contraindicated when pulmonary vascular changes secondary to pulmonary hypertension are of an advanced degree.

PULMONARY HYPERTENSION

In the newborn infant, the small pulmonary arteries normally are thick-walled muscular structures with a normal intima and a small

lumen, and the pulmonary artery pressure normally approaches that of the aorta. In the normal infant there is a gradual reduction in the thickness of the media of these vessels, and examination at 6 months reveals a thin-walled artery with a wide lumen. This is associated with a normal pulmonary artery pressure. It is well recognized that in a patient with a large left-to-right shunt, such as with a ductus arteriosus or a ventricular septal defect, the structures of the pulmonary arteries may change to resemble the infantile appearance with a thickened muscular coat. This pathologic state may progress to a stage of intimal fibrosis. The muscular hypertrophy is probably reversible with removal of the cause of the large pulmonary flow, while the intimal fibrosis is probably not.

Selection of patients for operation when pulmonary hypertension is present may be difficult. A patient with hypertension due to increased flow through the lungs will likely respond well to operation since pulmonary vascular changes are likely to be minimal or absent. This physiologic state is suggested by examination of chest by x-rays, which indicate evidence of increased pulmonary flow, i.e., increased vascular markings. Catheterization will confirm the presence of increased vascular flow through the lungs. Obliteration of left-to-right shunt will thus reduce the pulmonary flow, and the patient will be expected to respond well.

Conversely, obliteration of the ductus arteriosus may be contraindicated in the presence of advanced pulmonary vascular changes. An advanced degree of pulmonary arteriosclerosis increases the peripheral resistance of the vascular bed in the lungs to such a degree that pulmonary flow may be reduced below normal. Thus, x-rays of the chest are then likely to reveal that the pulmonary vascular markings are diminished. In this patient there may be clinical evidence of reversal of flow in the ductus (flow from pulmonary artery to aorta). The observation of cyanosis of the left upper extremity and lower extremities can only be explained by the escape of pulmonary venous blood from the pulmonary artery into the aorta and so into the left subclavian artery and descending aorta. A period of extreme

pulmonary hypertension in such a patient can thus be relieved through the patent ductus arteriosus. If, in this patient, the ductus is ligated, episodes of excessive pulmonary hypertension can no longer be relieved, and death results.

it is due to a progression of the obliterative process of the ligamentum arteriosum on to the aortic wall. The fact that in some cases the stenosis of the aorta is not at the point of attachment of the ligamentum arteriosum is evidence against this hypothesis. Coarctation

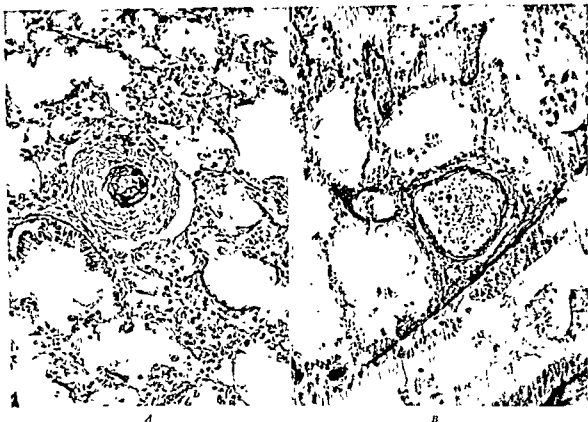


Fig 242—Photomicrographs of normal lung taken from a 4-day-old infant and a 7-year-old child.

A, A small pulmonary artery in the newborn infant is seen to have a narrow lumen and a thick wall due to hypertrophy of its muscle layer

B, The lumen of the artery is wide, and the amount of muscle present is relatively small (Courtesy The Montreal Children's Hospital)

COARCTATION OF THE AORTA

Embryology and Anatomy.—Characteristically this anomaly consists of an eccentric constriction of the descending thoracic aorta just below the origin of the left subclavian artery. This is usually uncomplicated by other anomalies, but its combination with a bicuspid aortic valve, subaortic or aortic stenosis, or patent ductus arteriosus is possible. The etiology of this vascular defect is not understood. The suggestion has been given that

of the abdominal aorta and of the aortic arch has been described.

Types of coarctation of the aorta are classified by Johnson as follows:

- I Ductus arteriosus closed
 - a With collateral circulation
 - b Without or with inadequate collateral circulation (incompatible with life)
- II Ductus arteriosus open
 - 1 Proximal to coarctation
 - a With collateral circulation
 - b Without or with inadequate collateral circulation (incompatible with life)

2. Distal to coarctation
- With collateral circulation
 - (1) Pressure maintained by collateral circulation adequate to prevent flow from pulmonary artery to aorta
 - (2) Pressure maintained by collateral circulation inadequate to prevent flow from pulmonary artery to aorta
 - Without collateral circulation

circulation, stimulated by the presence of the coarctation, is largely through vessels issuing from the arch of the aorta and its branches to join intercostal arteries below the coarctation, where the blood flow is opposite to normal. It has been postulated that the hypertension cannot be entirely explained by the ob-

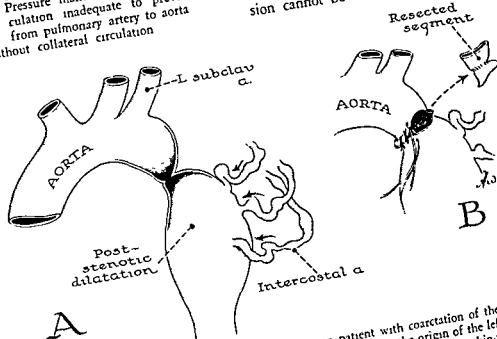
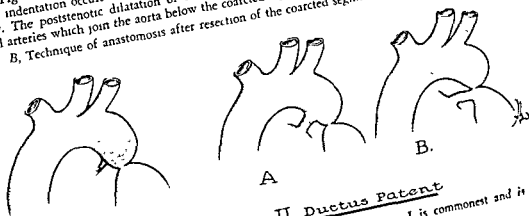


Fig. 243—A, Diagram showing typical findings in patient with coarctation of the aorta. The major indentation occurs on the lateral wall of the aorta just distal to the origin of the left subclavian artery. The poststenotic dilatation of the aorta is shown, and the large, tortuous thin-walled intercostal arteries which join the aorta below the coarcted segment are visualized.
B, Technique of anastomosis after resection of the coarcted segment



I. Ductus Closed

Fig. 241.—Diagram of three types of coarctation of the aorta. Type I is commonest and is not associated with a patent ductus arteriosus.

Physiopathology.—Type I coarctation of the aorta, the commonest variety encountered, results in elevated systolic and diastolic pressures proximally. Intra-arterial blood pressure recordings within the femoral artery have revealed that it is lower than normal and associated with a narrow pulse pressure. Collateral

II. Ductus Patent

struction in the aorta, but is partly due to the release of renin from the kidney.

Diagnosis.—The diagnosis of coarctation of the aorta is not difficult. The symptoms and signs depend upon the degree of constriction present and the presence or absence of complicating anomalies. Most patients with

coarctation fall into Type I and have no complaints, the diagnosis being made at routine examination. Males, who are the more frequently affected, are usually well developed and muscular. Complaints of headache, dyspnea on exertion, and palpitation may be given. Intermittent claudication is rarely noticed. The diagnosis can be made on physical examination, which reveals (1) hypertension in the upper extremities, (2) diminished or absent femoral artery pulsations and low blood pressure or imperceptible blood pressure by cuff

indentation of the aorta, best seen in the P.A. projection, is often demonstrable. Examination of the esophagus filled with barium reveals the so-called reversed 3 sign.

Complications.—Complications encountered include (1) cerebral hemorrhage due to rupture of a congenital aneurysm (present in approximately 20% of the patients) or rupture of an artery secondary to hypertension, (2) left ventricular failure, (3) aneurysm formation, or (4) subacute bacterial endarteritis. Statistics indicate that prior to the age

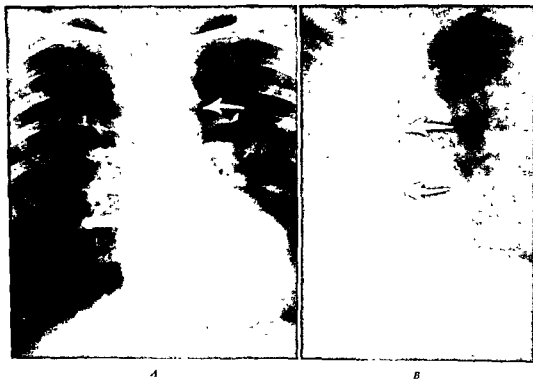


Fig. 245—X-rays of chest in patient with coarctation of aorta.

A, Notching of undersurfaces of ribs (not clearly seen after reproduction) and an indentation of descending thoracic aorta (arrow).

B, X-ray taken with the esophagus filled with barium demonstrates the reversed 3 sign (arrows) caused by impression of the esophagus by prestenotic aortic dilatation above and the poststenotic dilatation of the aorta below.

(Courtesy The Montreal Children's Hospital.)

in the lower extremities, (3) evidence of left ventricular strain, and (4) the presence of strongly pulsatile collateral vessels about the shoulder girdle. Chest x-rays may reveal notching of the undersurface of the first 7 ribs by enlarged, tortuous, intercostal arteries. This sign is not consistent, however, and is rarely seen prior to the age of 9 years. The actual

of 40 years, 75% of the patient will be in trouble from one of the above causes.

Treatment.—It is recognized that surgery is not indicated for every patient with coarctation of the aorta. Early operation is advised if the individual child or adult has symptoms referable to the malformations, such as marked hypertension, or if he has electrocardiographic

evidence of left ventricular strain. The optimum age for operation is 8-12 years. As with ductus arteriosus, a successful operation results in a normal circulation. Resection of the coarcted segment of aorta and end-to-end anastomosis is the preferred operation but, if this is not possible, as when a long segment of aorta is affected, the resected portion can be replaced by an arterial homograft or by a plastic prosthesis.

The results of operation are good. Operative mortality is low. The blood pressure falls gradually during the first 2-3 postoperative weeks. Resumption of full activity without restriction is permitted 6 weeks after operation.

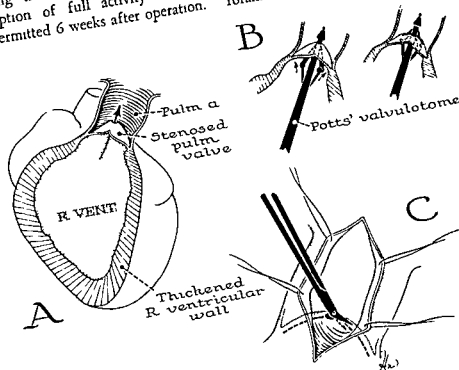


Fig 246.—A, Diagram of abnormality presented in pure pulmonary valvular stenosis. B, Technique of valvulotomy, using Brock procedure. C, Valvulotomy performed under direct vision, using hypothermia.

In patients who have a patent ductus arteriosus with coarctation of the aorta, operation may be indicated at an early age, due to the presence of a large left-to-right shunt through the ductus arteriosus. The decision to resect the coarctation of the aorta at the same time should be made only if collateral circulation is sufficient to permit clamping the aorta for the time necessary to resect and anastomose

PURE PULMONARY STENOSIS

Anatomy.—Congenital stenosis of the pulmonary valve without other complicating abnormalities is the commonest form of this anomaly. The valve cusps are fused, and there is a small central orifice which permits blood to escape from the right ventricle to the pulmonary artery. A poststenotic dilatation of the main pulmonary artery is almost invariably present.

Stenosis of the outflow tract of the right ventricle, i.e., an infundibular stenosis, may be encountered, and a combined valvular and infundibular stenosis is possible. A patent foramen ovale is not uncommon. A patent

ventricular septal defect in association with an infundibular stenosis has been described.

Physiopathology.—Pulmonary blood flow is diminished due to the obstructing abnormality between the right ventricle and pulmonary artery. Hypertrophy and dilatation of the right ventricle due to an elevated right ventricular pressure is found. A right-to-left shunt with cyanosis is possible in the presence of



Fig 247—X ray of chest in patient with pure pulmonary valvular stenosis revealing a prominent main pulmonary artery (arrow) due to poststenotic dilatation and diminished vascular markings in the lung fields (Courtesy The Montreal Children's Hospital)

a patent foramen ovale or other septal defect when the right atrial pressure exceeds the left.

Diagnosis.—Most patients are acyanotic but, in the presence of an atrial septal defect with a right-to-left shunt, cyanosis may be present. Dyspnea during exercise is the principal complaint.

On physical examination the second pulmonic sound is characteristically diminished or absent, and there is a grade 4, harsh systolic murmur, best heard in the pulmonic area. X-rays of the chest reveal that the pulmonary vascular markings are diminished, the outline of the main pulmonary artery is prominent, and the heart is enlarged due to hypertrophy and dilatation of the right ventricle. The electrocardiogram demonstrates right ventricular hypertrophy.

Valuable information is obtained by cardiac catheterization. The presence of a high right ventricular pressure and a low pulmonary artery pressure without evidence of left-to-right shunt confirms the diagnosis. By obtaining a pressure tracing during withdrawal of the catheter from the pulmonary artery to the right ventricle, it is possible to clarify the nature of the obstructing lesion. A pure

valvular stenosis is indicated by a low pressure in the pulmonary artery and the abrupt appearance of an elevated pressure when the catheter reaches the right ventricle.

In the presence of a valvular and an infundibular lesion, the transition from low to high pressure will be more gradual as the catheter is pulled back from the pulmonary artery into the chamber between the pulmonary valve and infundibular bar and then into the right ventricle.

Treatment.—Selection of patients for operation is based on the presence or absence of symptoms and on the evaluation of the physiopathology as clarified by laboratory investigation.

Pulmonary valvulotomy is performed for pulmonary valvular stenosis, whereas excision of the stenotic area is required to cure infundibular stenosis.

Pulmonary valvulotomy may be accomplished blindly through a small incision in the right ventricular wall, through which a valvulotome is introduced into the right ventricle. The cutting blades of the valvulotome are then opened and the instrument forced through the valve into the pulmonary artery. By this means a bicuspid pulmonary valve is created. This operation is associated with a low operative mortality and patients are symptomatically greatly improved. Although postoperative catheter studies often reveal a satisfactory drop of right ventricular pressure, this is not always consistent.

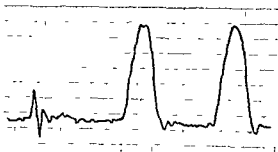


Fig 248—Blood pressure tracing recorded during heart catheterization and while the tip of the catheter is withdrawn from the pulmonary artery into the right ventricle. A low pulmonary artery pressure with an abrupt rise as the tip of the catheter slips past the pulmonary valve demonstrates the presence of a pure pulmonary valve stenosis. (Courtesy The Montreal Children's Hospital)

Pulmonary valvulotomy, however, is best performed under direct vision, which permits a more extensive operation upon the valve and a greater assurance of a complete valvulotomy. This is accomplished with the patient under hypothermia and with arrest of circulation achieved by temporary occlusion of both venae cavae. The pulmonary artery is then incised and the valve cut under direct vision. Postoperative catheter studies have indicated that this results in a more consistent reduction of the right ventricular pressure. Similar direct valvulotomy can be performed while the heart is excluded from the general circulation, using an artificial heart-lung apparatus.

ANOMALIES OF THE AORTIC ARCH AND ITS PRIMARY BRANCHES

Abnormalities of these vessels can produce complications due to compression or distortion of the esophagus and trachea.

Right Aortic Arch

A right aortic arch is not an uncommon finding in association with other severe cardiac malformations. It may appear, however, as a single entity. The arch is directed to the right of the esophagus and trachea and may descend on the right or traverse behind the esophagus and trachea to descend to the left of the midline. This course alone, although it may

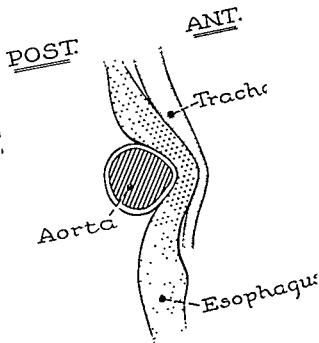


Fig. 249—X-ray of the chest (with explanatory diagram) in a patient with a "vascular ring" caused by the aorta as it traverses the mediastinum from right to left. There is also compression of the trachea. (Courtesy The Montreal Children's Hospital.)

The results of operation using hypothermia, with inflow caval occlusion and division of the valve under direct vision, are good. The mortality rate is low and the circulatory hemodynamics returns to normal in most patients.

distort the esophagus, is not likely to produce symptoms, since the capacity of the mediastinum is such that obstruction of the esophagus and trachea does not occur. The presence of a ductus arteriosus or ligamentum arteriosum connecting the main pulmonary

artery to the descending aorta completes a ring of vessels about the trachea and esophagus. The ring thus formed is made up of the ascending aorta in the midline, the arch of the aorta to the right, the terminal portion of the arch and first portion of the descending aorta behind the trachea and esophagus, and the ligamentum arteriosum or patent ductus arteriosus to the left. It is possible for this configuration of vessels to compress the trachea and esophagus sufficiently to cause complaints.

Diagnosis.—The principal complaint may be dysphagia due to distortion of the esophagus. The history of repeated episodes of bronchopneumonia, particularly when associated with a persistent brassy cough, is suggestive of the presence of the malformation and is secondary to narrowing of the trachea by the vascular ring.

Examination of plain films of the chest may reveal compression of the trachea. Confirmation of the presence of a tracheal or esophageal compression is obtained by barium swallow.

An oblique indentation of the posterior aspect of the esophagus, caused by the arch traversing from right to left, and an indentation of the side of the esophagus, caused by a persistent ligamentum arteriosum, are demonstrated during fluoroscopic examination of the barium-filled esophagus. The combination of tracheogram and esophagogram to ascertain the precise nature of the ring is rarely indicated.

Treatment.—Operation may be indicated at an early age in order to obviate complications resulting from this abnormality. At operation a wide dissection of the mediastinum is performed to clarify the type of vascular anomaly. A ligamentum arteriosum, which completes the ring, is divided. Further dissection and displacement of vascular structures relieve any persistent compression of the trachea and esophagus.

Double Aortic Arch

In this malformation the ascending aorta splits into two main trunks which encircle the esophagus and trachea, joining to form the descending aorta. This abnormality is due to the persistence of both right and left

branchial arteries. In most instances the anterior or left arch is smaller than the posterior one.

Although this anomalous arrangement of vessels may produce no complaints and no complications, compression of the esophagus and trachea is possible. The complaints are similar to those previously described and the diagnosis is suggested by the type of investigation given above. Surgical therapy is undertaken in the presence of dysphagia or respiratory complications. At operation the smaller limb, usually the anterior, is selected for division, which is made in such a manner that circulation to the head and neck is not compromised. As with the surgical treatment of anomalies associated with the right aortic arch, the vessels are displaced and sutured in such a manner that further compression will not occur.

Anomalous Origin of Cephalic Arteries

Bayford (1789) described a malformation consisting of anomalous origin of the right subclavian artery, which arose from the left side of the aortic arch and, as a result of its course to reach the right upper extremity, traversed behind the esophagus, compressing it forward in so doing. This caused severe dysphagia, and so the descriptive term *dysphagia lusoria* was given by this author. It is recognized that such compression may be caused by an abnormal course of other cephalic vessels which may indeed traverse between esophagus and trachea rather than behind them.

The presence of this abnormality is suggested by fluoroscopic examination of the barium-filled esophagus. Operation is indicated in the presence of dysphagia. The subclavian artery or other vessels are exposed and divided between ligatures, and all strands of tissue which may cause a persistence of esophageal distortion are similarly freed.

ATRIAL SEPTAL DEFECT

Differentiation should be made between the two types of atrial septal defect, i.e., *ostium secundum* and *ostium primum*. In *ostium secundum*, due to inadequate development of the secondary septum, a large defect remains (commonly including the foramen ovale).

Such defects are situated in the upper and posterior parts of the interatrial septum and have normal septal tissue between the defect and the atrioventricular canal. *Ostium primum* defects, on the other hand, are rare and are situated in the lower part of the interatrial septum. They are due to persistence of the ostium primum because the septum primum failed to descend and unite with the cushion between the atrioventricular orifices. Their close association with the mitral and tricuspid valves accounts for the fact that the latter are commonly deformed.

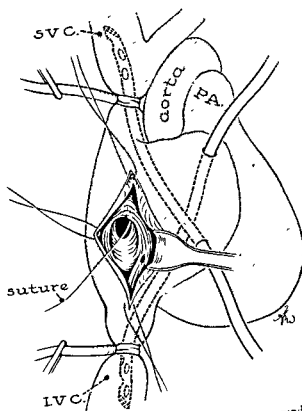


Fig. 250.—Diagram demonstrating a secundum type of interatrial septal defect. The venous return to the heart has been diverted to a pump oxygenator. Suture closure of the defect is being accomplished by an approach through the right atrium.

Diagnosis.—In most patients blood flows across the defect from the left atrium to the right atrium; consequently there is absence of cyanosis. In early life there may be no related complaints or disability, but many patients will exhibit a gracile habitus and shortness of breath on exertion. On physical examination a precordial thrill may be palpated and a grade 3 systolic murmur heard at the

left 2nd intercostal space just to the left of the sternal border. These findings are suggestive of the diagnosis of an atrial septal defect. The electrocardiogram in a high proportion of patients exhibits a so-called partial right bundle branch block, which is important evidence in favor of this diagnosis. Fluoroscopic evidence in favor of this diagnosis is that fluoroscopy reveals considerable enlargement of the right atrium and ventricle and markedly increased pulmonary vascular markings. The aorta is not prominent. Confirmation of the diagnosis is obtained by heart catheterization.

It is important to differentiate this defect from the anomalies of the pulmonary veins which, instead of draining into the left atrium, enter the right atrium or venae cavae. This may be possible by the use of angiocardiology and by the study of dye dilution curves.

Treatment.—Successful closure of atrial septal defects of the secundum type has been accomplished by several different techniques. First attempts at obliteration of the defect were made by invaginating portions of the atrial wall into the defect and suturing them in place. Swan experimentally used the tip of the atrial appendage inverted into the defect. Subsequently, Bailey modified this technique by inverting the lateral wall of the atrium to the rim of the defect, using as a guide the finger introduced into the atrium via the right atrial appendage. Modifications of this technique have included the use of pericardium and plastic materials attached to the atrial wall. Subsequently, Gross described the use of a rubber well sutured to the side of the atrium. This permitted the surgeon to introduce sutures to repair the defect by placing instruments into the atrium via a well containing blood. More recently it has been shown by Sondergaard that it is possible to dissect the plane between the right atrium and the right pulmonary veins in such a manner that a circular suture can be passed through the lower portion of the atrial septum which, when tightened, closes the atrial septum. It should be emphasized that all these techniques are blind or closed heart methods, the manipulation being carried out by touch. Other methods have permitted suturing the defects under direct vision with the interior of the right atrium widely exposed. This has

been successfully accomplished during hypothermia with the venous return to the heart obstructed temporarily. It has also been possible during temporary by-pass of the heart and lungs, using an artificial heart-lung machine. The chamber is explored through an incision placed in the atrium. The defect or defects are then closed by means of a running suture or by stitching a plaque of Ivalon sponge to the edges of the defect.

It cannot be overemphasized that selection of patients for operation is important. The operative mortality of patients with a primum type of defect is high. The results of operation with the secundum type, however, are eminently good. As with patients having a patent ductus arteriosus, evaluation of the pulmonary hypertension is important since, if it is of systemic levels with established pulmonary vascular changes, closure of an atrial septal defect is usually contraindicated.

VENTRICULAR SEPTAL DEFECTS

Anatomy.—In this malformation there is a defect in the septum between the right and left ventricles. The various anomalies have been classified according to whether they occur in the inflow or outflow tracts of the right ventricle. The outflow portion of the right ventricle may be defined as that part lying between the pulmonary valve above and the nearest portion of the tricuspid valve below. Those related to the outflow tract seem more common, and defects just inferior to the crista supraventricularis are most often encountered. Multiple defects have been described.

Physiopathology.—The presence of a ventricular septal defect permits a shunt of blood from left to right ventricle. Should the defect be small, the volume of blood shunted may not cause significant alteration of the circulatory dynamics. However, a large shunt of blood results in a greatly increased pulmonary vascular flow. There is a strain upon the left ventricle since it must pump a greatly increased volume of blood. The presence of this large left-to-right shunt is likely to result in pulmonary hypertension which, as with the patent ductus arteriosus, may place a strain on the right heart and may become irreversible.

Diagnosis.—The patient with this lesion is acyanotic since the shunt of blood is from left to right. Growth and development may be normal, but in the presence of a shunt of an appreciable level the child may appear thin and frail, and his growth is likely to be retarded.

Characteristically, on physical examination the heart is enlarged, and a loud systolic thrill and murmur are present. The latter is of a harsh, rasping quality of maximal intensity in the 3rd and 4th intercostal spaces. X-rays of the chest reveal cardiac enlargement, a prominent main pulmonary artery, and increased pulmonary vascular markings. Cardiac

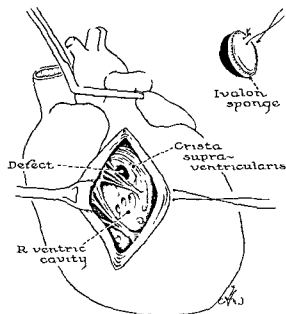


Fig 251.—A ventricular septal defect caudad to the crista supraventricularis. Exposure is obtained during by-pass of the heart by means of heart-lung machine. A patch of Ivalon has been prepared for suturing to the edges of the defect.

catheterization is employed in order to confirm the diagnosis and to provide information regarding the abnormal physiologic state.

Treatment.—Closure of the ventricular septal defect has been made possible with the development of artificial heart-lung machines. Such apparatus permits exposure of the defect and its closure under direct vision. The heart is widely exposed through a bilateral thoracotomy incision. With the patient's blood

diverted by means of the artificial heart-lung apparatus, an incision is made in the wall of the right ventricle. The nature and site of the defect are determined, and closure is accomplished by direct suture or by application of an Ivalon pledget to fill the defect. The risk from operation seems related to the size of the lesion and to the degree of associated physiopathology. Operation on patients with pulmonary hypertension of a high degree may be technically successful, but the incidence of complete heart block following operation is higher than in those with a lesser degree of pulmonary hypertension

stenosis of the pulmonary valve or the orifice of the pulmonary conus, high interventricular septal defect, and hypertrophy of the right ventricle.

Physiopathology.—From a knowledge of the anatomic arrangement it is understood that blood entering the right atrium flows to the right ventricle, where it cannot readily go to the pulmonary artery because of the pulmonary stenosis and, since the aorta arises from both right and left ventricles, venous blood escapes directly into the aorta along with blood from the left ventricle. As a consequence the pulmonary flow is reduced.

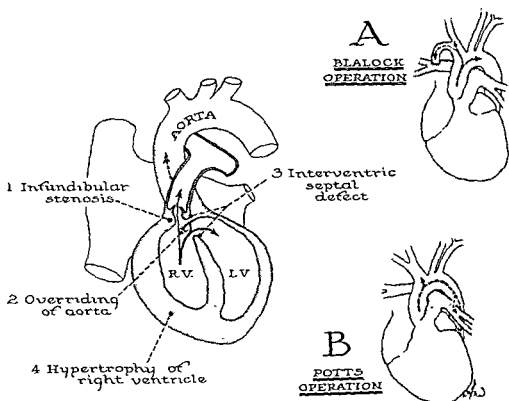


Fig. 252.—Diagram demonstrating types of deformity in tetralogy of Fallot. The outflow tract of the right ventricle is narrowed (infundibular stenosis). The aorta is overriding, and an interventricular septal defect is present.

A, Technique of Blalock operation.

B, Technique of Potts operation.

The operative mortality rate of patients under 2 years of age is high. A successful operation is curative, and a normal cardiovascular system is the result.

TETRALOGY OF FALLOT

Anatomy.—The tetrad of Fallot consists of dextroposition (overriding) of the aorta,

The systemic blood with its admixture of venous blood from the right ventricle accounts for the degree of cyanosis present. The hematopoietic tissue responds in an attempt to compensate and an excessive number of red blood cells are produced so that blood samples show a high red blood count, hemoglobin, and hematocrit reading.

Diagnosis.—In patients with this anomaly, cyanosis is generally observed from birth or within a few months. Retardation of growth, shortness of breath, and easy fatigability are common findings. When tired, such patients characteristically tend to squat rather than to sit or lie down.

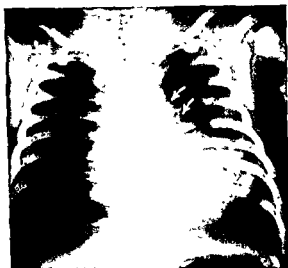


Fig 253—X-ray of a patient with tetralogy of Fallot. The typical boot shape of the cardiac contour is revealed. The outline produced by the main pulmonary artery (arrows) is concave, and the pulmonary vascular markings are reduced. (Courtesy The Montreal Children's Hospital.)

On physical examination, in addition to cyanosis, clubbing of the fingers and toes is present. A thrill may be palpable over the precordium. Auscultation reveals the presence of a harsh systolic murmur heard over the base of the heart. Laboratory examination demonstrates polycythemia and an elevated blood hemoglobin. Characteristically, roentgenologic examination demonstrates that the heart is of normal diameter but boot-shaped in contour. The outline of the pulmonary artery is generally concave. Pulmonary vascular markings are, as one might expect, diminished. Right ventricular hypertrophy is the typical electrocardiographic finding.

A correct diagnosis can frequently be made after clinical examination alone, but in some instances special procedures must be undertaken. Angiocardiography will demonstrate that dye injected into a systemic vein will be seen to fill the aorta and pulmonary artery

simultaneously. This suggests that the aorta overrides the right ventricle.

At heart catheterization, pressure recordings reveal a normal or low intrapulmonary artery pressure and a high right ventricular pressure indicative of an obstruction between the right ventricle and pulmonary artery. In addition, the pulmonary artery blood flow is diminished, and the systemic arterial blood is unsaturated. Table 19 gives the report of a 5-year-old child, height 44½", weight 50 lb., body surface area 0.83 M².

Complications.—(1) Cerebral accident secondary to thrombosis within a cerebral artery and (2) anoxic spells which, in some patients with severe degrees of this malformation, result in episodes of unconsciousness.



Fig 254—Venous angiocardiogram in transposition of great vessels. This demonstrates the opaque medium filling the superior vena cava, right atrium, and right ventricle and passing from the right ventricle into the transposed aortic root, then outlining the aortic arch. (Courtesy The Montreal Children's Hospital.)

Treatment.—Two fundamentally different forms of treatment are available. In the first, a partial recirculation of systemic mixed venous and arterial blood from the aorta, through the lungs, results in an elevation of the systemic blood-oxygen saturation. This form of therapy does not alter the structural abnormality. The second approach is aimed at surgical correction of the malformation.

Treatment by Systemic Pulmonary Artery Anastomosis.—The purpose of this type of operation is to create an anastomosis between

CARDIAC SURGERY

TABLE 19
CARDIAC CATHETERIZATION REPORT

SITE	O ₂ CONTENT VOL% %	HEMOGLOBIN O ₂ SATURATION	PRESSURE SYSTOLE MM. HG	PRESSURE DIASTOLE MM. HG	MEAN
Left subclavian vein	11.70	61.6			30
High superior vena cava	11.72	61.7		2.5	26.0
Low superior vena cava	11.69	61.5	4.0	3.0	9.0
High right atrium	12.21	64.3	86.0	3.0	
Mid right atrium	12.29	64.7	24.0		
Mid right ventricle	12.90	68.0		6.0	9.0
High right ventricle (subvalvular)	13.78	72.7	16.0	7.0	61.0
Left pulmonary artery	13.04	68.7	15.0	52.0	
*Main pulmonary artery	13.10	69.0	88.0		
*Femoral artery	15.76	82.1			
*Oxygen consumption	= 126 ml/min				
Oxygen capacity	= 18.98 ml/100 ml blood				

Systemic flow = $\frac{126 \times 100}{15.76 - 12.29} = 4,632.3 \text{ ml/min} = 5,578.8 \text{ ml/M}^2/\text{min}$

Effective pulmonary flow = $\frac{126 \times 100}{18.54 - 13.04} = 2,018 \text{ ml/min} = 2,430 \text{ ml/M}^2/\text{min}$

Total pulmonary flow = $\frac{126 \times 100}{18.54 - 13.04} = 2,290 \text{ ml/min} = 2,759 \text{ ml/M}^2/\text{min}$

Right-to-left shunt
Left-to-right shunt

Comment: Pressures in the pulmonary artery were normal. Significant stenosis was demonstrated, both at the pulmonic valve and about 1 cm below the valve in the right ventricular outflow tract. Systolic pressures in the right ventricle and the femoral artery were almost identical. Bi-directional shunting was demonstrated at the high ventricular level, the right-to-left predominating.

Conclusions: Tetralogy of Fallot with pulmonic valvular and infundibular stenosis and predominant right to-left shunt

*Simultaneous observations

the aorta or one of its branches and a pulmonary artery. This permits a portion of the mixed venous and arterial blood within the aorta to flow to the lungs. This shunted blood returns to the left atrium fully oxygenated. The effect of this alteration in blood flow is that the average level of oxygenated blood in the systemic system is increased, resulting in a diminution or complete disappearance of cyanosis, a reduction of polycythemia and, consequent danger of cerebral thrombosis, and, of greater importance, an increase in the exercise tolerance of the patient.

The operation may be accomplished by joining the end of a systemic artery (preferably a subclavian artery) to the side of the ipsilateral pulmonary artery—the *Blalock operation*. A similar result may be achieved by anastomosis of the side of the descending thoracic aorta to the side of the ipsilateral pulmonary artery—the *Potts operation*.

Corrective Operations.—The *Brock operation*, described by Sir Russell Brock, consists of the removal of the obstruction to pulmonary blood flow, whether due to pulmonary valve or infundibular stenosis. Pulmonary valvulotomy is performed by inserting a valvulotome into the right ventricle through a small incision. This instrument is then directed through the pulmonary valve which it splits during its passage into the pulmonary artery. Resection of the infundibulum (infundibulotomy) is performed by biting out segments of the obstructing muscular structure within the right ventricle. The use of an artificial heart-lung apparatus has made a more complete corrective operation possible. With the heart-lung apparatus and the heart widely exposed, a long right ventricular incision is made. Under direct vision, pulmonary valvulotomy or infundibulotomy is accomplished, and the interventricular septal defect and over-

riding of the aorta are treated. If the outflow tract of the right ventricle is then not sufficiently wide, it can be increased by sewing a piece of Ivalon sponge between the cut edges of the right ventricle.

The results of this type of operation have become impressively good. Although the operative mortality is currently higher than that incurred with the Blalock or Potts operation, the indications are that the long-term results may be better.

OTHER CYANOTIC MALFORMATIONS WITH DIMINISHED PULMONARY FLOW

This category includes patients with tricuspid atresia, pseudotruncus arteriosus, and a common ventricle with pulmonary stenosis. The essential features are an obstruction of the flow of blood between the right atrium and the lungs and a septal defect, either atrial or ventricular or both, which permits venous blood to escape into the aorta.

In carefully selected patients, a systemic to pulmonary artery anastomosis is beneficial. The risk of operation in all is considerably higher than with tetralogy of Fallot, and the results in general are not as good. The exception to this is the trilogy of Fallot, pulmonary valve stenosis with an atrial septal defect which can be treated by valvotomy and closure of the septal defect. This operation is curative and may be performed during inflow occlusion under hypothermia or with a heart-lung machine.

TRANSPOSITION OF THE GREAT VESSELS

In this condition, the pulmonary artery originates from the left ventricle and the aorta from the right ventricle. An associated ventricular septal defect, patent foramen ovale, or patent ductus arteriosus is commonly observed. Other coexisting malformations, such as a right aortic arch, pulmonary valvular stenosis, coarctation of the aorta, and a stenosis of other valves, have been observed. The principal defect, the transposition of the aorta and the pulmonary artery, may be complicated by a variety of other anomalies of the heart and great vessels. The principal malformation has been ascribed to the failure of the aortic-pulmonary septum to spiral.

Physiopathology.—Although the abnormality permits survival of the fetus without compromising the function of the heart or oxygenation of the tissues, the situation is greatly altered following birth. In the newborn infant, with transposition of the great vessels, blood which enters the right atrium and right ventricle is expressed to the aorta, whereas pulmonary venous blood returning to the left ventricle is pumped out to the pulmonary artery. It is clear that survival would not be possible unless mixing of venous and arterial blood were possible. The presence of an atrial or ventricular septal defect may permit sufficient mixing of venous and arterial blood for the infant to survive. However, in most patients the strain placed on each ventricle is great, and they become dilated and hypertrophied in early life. The patients often die of cardiac failure within the first year of life.

The presence of venous blood in the systemic circulation accounts for the degree of cyanosis. If a septal defect is absent, cyanosis will be intense and survival beyond a few days or weeks will not be expected. In the presence of a large septal defect with sufficient mixing of blood, cyanosis may not be as obvious and indeed may be present only when the child cries lustily. It is characteristic, however, that cyanosis progressively becomes more intense.

Diagnosis.—Cyanosis is usually obvious from birth and may be very intense. The infant characteristically fails to thrive and exhibits marked dyspnea. Auscultatory findings are not characteristic and vary according to the type of secondary malformation present. X-ray and fluoroscopic examination is usually significant since it reveals a marked enlargement of both ventricles. This enlargement is rapidly progressive, as revealed when x-rays taken soon after birth are compared with ones taken 2-3 months later. There is evidence of pulmonary vascular congestion, and with fluoroscopy careful evaluation can often indicate the abnormal origin of the great vessels. Electrocardiograms commonly indicate a combined ventricular enlargement. Diagnosis may be confirmed by means of heart catheterization or by the use of angiocardiology.

Complications.—Death may result from cardiac failure or during an anoxemic spell.

Treatment.—This malformation has been a perplexing one in regard to the development of an operation which could be expected to improve the patient. Attempts have been made to increase mixing of venous and arterial blood by the creation of an atrial septal defect and by a systemic to pulmonary artery anastomosis. Others have tried to transpose the aorta to the left ventricle and the pulmonary artery to the right. These procedures have met with little success.

It has recently been recorded that the circulation can be improved by partially transposing the venous return to the heart. In the Baffes operation, the principal procedure consists of transplanting the right pulmonary veins to the right atrium and directing the inferior vena cava blood to the left atrium. By this means the right pulmonary venous blood returns to the right atrium and is then delivered into the right ventricle and so to the aorta. The inferior vena cava blood is redirected to the left atrium, the left ventricle, and consequently to the pulmonary artery. The operative procedure is accomplished as follows: The right pulmonary vein is divided and its distal end sutured to the side of the right atrium. The inferior vena cava is connected with the stump of the divided right pulmonary vein by means of an aortic graft. Subsequent to this the junction between the vena cava and the right atrium is ligated. This ensures that all of the blood returning to the heart via the inferior vena cava is directed through the graft and through the stump of the right pulmonary vein to the left atrium.

The operative mortality for this operation is high, being in the vicinity of 50%. There has been a sufficiently high increase in exercise tolerance and improvement in general condition of the patients who have survived that the operation should be seriously considered for a patient with transposition of the great vessels. It is impressive indeed to observe a small infant with this sort of serious heart ailment undergo a major technical operation and be so obviously improved following the operation.

ACQUIRED HEART DISEASE

TRAUMA

The earliest form of heart surgery was concerned with the treatment of wounds. Although these were almost invariably fatal, they formed the basis for the development of cardiac surgery.

Since all forms of heart trauma may be complicated by hemopericardium, the recognition of acute cardiac compression is essential.

Cardiac Compression

Cardiac compression (tamponade) interferes with cardiac action by preventing adequate ventricular filling. In the acute form, death occurs if the tamponade is not relieved. The heart will tolerate a considerable degree of chronic compression, but inevitably cardiac failure ensues.

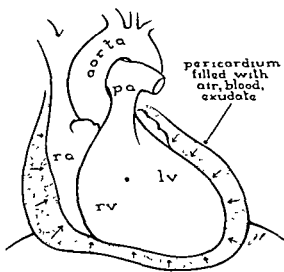
Acute compression, whether due to exudate from pericarditis or from bleeding into the pericardium, presents the characteristic signs of the *Beck triad*: (1) a small quiet heart, (2) rising venous pressure, (3) falling arterial pressure. Unless the pressure is relieved, the patient will die. The treatment of choice is pericardial aspiration. Operation is rarely necessary.

Trauma to the Heart and Great Vessels

Traumatic lesions may be divided into (1) contusions, (2) penetrating wounds, and (3) foreign bodies within the heart or its chambers.

Contusions.—Nonpenetrating trauma to the chest or abdomen may result in cardiac contusion. The steering-wheel injury is a frequent example. The diagnosis rests, to a large extent, upon the surgeon's awareness of the possibility of such injuries which vary greatly in their symptomatology and complications.

The patient may be asymptomatic, or he may develop angina, myocarditis with or without failure, coronary thrombosis with or without infarction, delayed rupture, or thromboembolism. Frequently arrhythmias or conduction disturbances are manifested. Such cases are best treated medically, including pericardiocentesis where there is evidence of hemopericardium and tamponade. Surgery has proved effective in the treatment of structural



A



B

Fig. 255—A, Diagram demonstrating cardiac tamponade due to pericardial fluid which prevents adequate ventricular filling (From Vineberg, A., Gialloredo, O., and Laberge, J., in Thorek, Max Surgical Errors and Safeguards, ed. 6, Philadelphia, J. B. Lippincott Co.)

B, X-ray showing pericardial fluid causing cardiac tamponade. Note widening at the waist of the heart which is usually associated with large amounts of pericardial fluid (Courtesy Montreal Heart Institute)

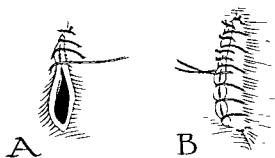


Fig. 256—Technique of closing laceration of ventricle

defects such as rupture of the cardiac septa or the aortic valve. The aorta may be ruptured and a false aortic aneurysm develops.

Penetrating Wounds.—Penetrating wounds, most often caused by bullets or stabbing, result in massive hemorrhage or acute compression. The entrance wound need not even be in the thorax, and tamponade may mimic hemorrhagic shock.

Ravitch and Blalock have advocated conservative treatment for cardiac wounds and point out that if cardiac compression is re-

lieved by pericardial aspiration, the bleeding usually ceases spontaneously. If bleeding continues, thoracotomy and repair of the wound are indicated.

Foreign Bodies.—Foreign bodies in the heart, rarely seen in civilian life, give rise to complications in 50% of cases. These may be emboli, cardiac aneurysm, myocardial rupture, abscess, ulcerative endocarditis, or psychologic disturbance from worry. Foreign bodies should be removed only if definitely indicated.

PERICARDITIS

Anatomy.—The pericardium is composed of two layers: (1) the *parietal pericardium* which consists of an outer *fibrous layer* lined by a *serous membrane*; (2) the *visceral pericardium* which is the reflection of the inner serous layer of the parietal pericardium over the base of the great vessels and over the heart (*epicardium*). The serous layer thus forms a continuous membrane which is the *pericardial sac*.

Classification of Pericarditis

Acute	Chronic
Serofibrinous	Chronic (pericarditis)
Hemorrhagic	Adhesive (pericarditis)
Purulent	Constrictive (pericarditis)
	Adhesive mediastinopericarditis

Acute Pericarditis

The rapid accumulation of fluid in the pericardium produces the signs of cardiac compression. The *Beck triad* plus tachycardia and orthopnea are present. When the fluid is nonpurulent there may be an intermittent low-grade fever, when there is pericardial pus, the constitutional signs of infection are present.

X-ray examination may show a typical pattern and is useful in the detection of pockets of pus. The ECG may show the typical pattern.

Differential Diagnosis.—Occasionally acute pericarditis that has progressed to the stage of cardiac failure is difficult to differentiate from *cor bovinum* which also presents a markedly widened heart shadow.

Treatment.—Pericardiocentesis is usually performed under local anesthesia with the patient sitting. A needle is inserted between the xiphoid and left costal margin and directed upward through the diaphragm into the pericardium. The removal of serous fluid is therapeutic. If pus is withdrawn, pericardial drainage (pericardiostomy) can be done under

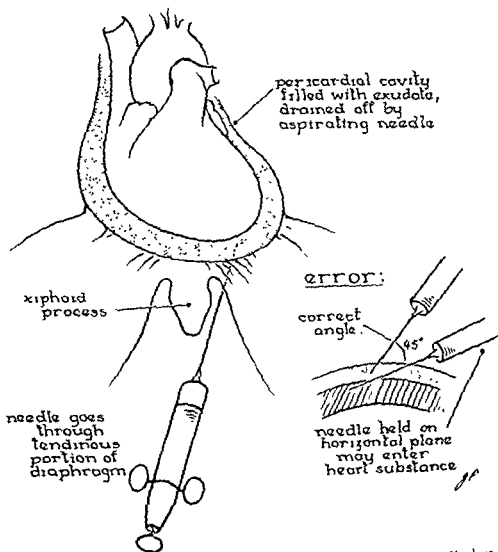


Fig 257.—Diagram demonstrating a method of pericardiocentesis (From Vineberg, A., Gialloreti, O., and Laberge, J., in Thorek, Max: *Surgical Errors and Safeguards*, ed 6, Philadelphia, J. B. Lippincott Co.)

local anesthesia. One of two approaches may be used. (1) resection of the 7th left costal cartilage or (2) below the left costal margin, by reflecting the attachment of the abdominal muscles.

Chronic Pericarditis *Constrictive Pericarditis*

Etiology.—Tuberculous pericarditis is the commonest cause. Suppurative and serofibrinous pericarditis account for a small percentage of cases, and recently it has been reported that organizing hemopericardium may result in constriction. Postoperative drainage of the pericardium is thus extremely important.

Pathology.—During acute pericarditis, fibrin is deposited on the serous surfaces of the pericardial sac. Organization results in contracting scar tissue which may become calcified.

Hemodynamics.—Because the cardiac chambers are constricted, ventricular filling is decreased. Venous congestion and its sequelae occur. This picture is similar to that of congestive failure, although the myocardium is intact. However, changes in the aortic-coronary pressure gradient and low cardiac output may produce true myocardial failure as well. These latter cases have a poorer prognosis following surgery.

Clinical Picture.—The patient rarely gives a history of tuberculosis. Fatigability, abdominal enlargement, dependent edema, and dyspnea are the usual symptoms. Mild peripheral cyanosis and venous engorgement, hepatomegaly, ascites, and hydrothorax are usually present. Blood pressure is low with a relatively high diastolic pressure, and venous pressure is markedly elevated. X-ray may show a normal heart size. Absent pulsation of the cardiac chambers, on fluoroscopy, and the

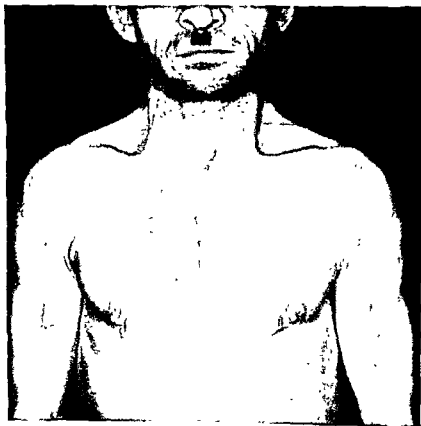


Fig. 258—Infrared photograph demonstrating the venous distention of the upper extremities, neck, and thorax to the level of the 2nd interspace, caused by pericardial occlusion of the superior vena cava due to constrictive pericarditis (Courtesy Montreal Heart Institute)

presence of calcification are important signs. Calcification is the only pathognomic sign of pericarditis, but constriction is not necessarily present. The Beck triad again is evident.

Treatment.—The aim is to remove the constricting scar which prevents the heart from filling. This is termed *cardiac decortication* or *pericardiectomy*. The approach to the pericardium can be made either through a median sternotomy, as recommended by Holman and Willett, or by a transsternal incision opening both pleurae, as favored by Johnson and Kirby. Other incisions made adequate decortication difficult. There are usually two distinct layers of scar tissue, of which the inner layer is tightly adherent to the heart and must be removed by sharp dissection. The principle of first freeing the outflow tract is followed

First the left ventricle, then the right side and finally the venae cavae are decorticated. Care must be taken to avoid injury to the coronary vessels and perforation of the heart. At the conclusion of operation the superior vena cava pressure should be less than 10 cm. of water. Antituberculosis therapy is given postoperatively.

Results.—Holman and Willett report 265 cases with 22 operative and 48 postoperative deaths. Of the 265, 118 patients were cured and 44 improved. Bigelow reports 13 cases with 2 deaths, and the cure of all survivors.

VALVULAR DISEASE

Etiology.—Rheumatic fever is by far the commonest cause of acquired valvular disease. Frequently, however, no history of rheumatic



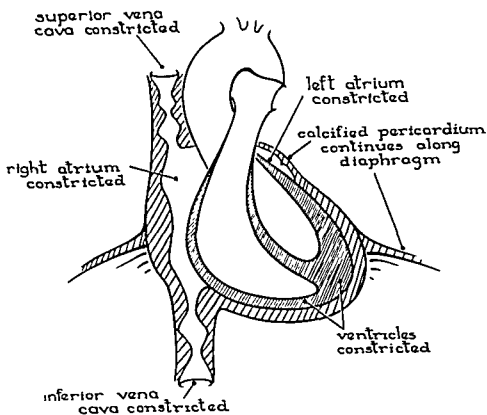
A

Fig. 259.—A, Lateral x-ray of heart showing calcification of pericardium in patient with advanced constrictive pericarditis, subsequently successfully treated by decortication.

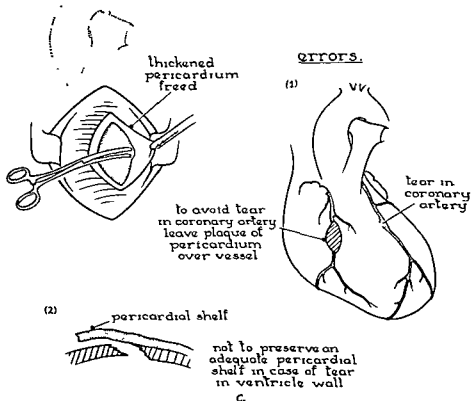
B, Calcific constricting pericardium.

C, Method of decortication (pericardiectomy).

(B and C from Vineberg, A., Gialloreti, O., and Laberge, J., in Thorek, Max: *Surgical Errors and Safeguards*, ed. 6, Philadelphia, J. B. Lippincott Co.)



B



C.

Fig 259 (cont'd) — (See opposite page for legend)

fever can be obtained and sometimes the only past illness is scarlet fever or tonsillitis.

The effects upon the heart valves may be divided into three main groups.

1. *Valvular stenosis.* The valve is narrowed, impeding the forward flow of blood.

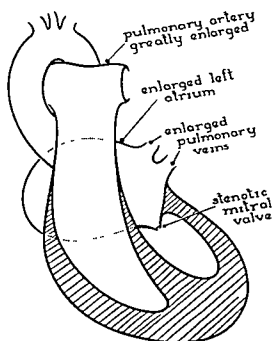
2. *Valvular insufficiency.* The valve permits a retrograde flow of blood after it is closed.

3. *Combined valvular stenosis and insufficiency.*

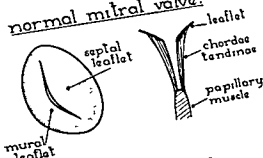
Clawson et al. found at autopsy that the valves are involved in the following frequency: mitral 73%, aortic 62%, tricuspid 10%, and pulmonic 3%. The mitral valve alone was

leaflet. At their periphery the leaflets are attached to the annulus which surrounds the whole valve. The papillary muscles, through the chordae tendineae, are inserted into the free edge of the leaflets.

Ventricular systole distends the leaflets like two parachutes. Their free edges approximate, preventing the return of blood into the left atrium. The papillary muscles and the chordae tendineae prevent the leaflets from blowing back into the atrium, and the aortic leaflet guides the blood into the aorta. Valve adjustments are made by contraction of the papillary muscles and by the mitral annulus. The mitral



normal mitral valve:



stenotic mitral valve:

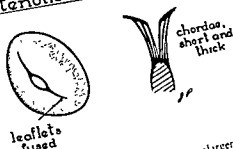


Fig 260—Mitral stenosis. Note fusion of leaflets along commissures, resulting in enlargement of left atrium (From Vineberg, A., Gialloreti, O., and Laberge, J., in Thorek, Max. *Surgical Errors and Safeguards*, ed 6, Philadelphia, J B Lippincott Co.)

damaged in 34% and the aortic valve alone in 24%. Tricuspid involvement never occurred as a solitary lesion, and combined aortic and mitral disease occurred in 38%.

Valvular Stenoses

Mitral Stenosis

Anatomy.—The mitral valve, between the left atrium and left ventricle, is composed of two leaflets, the larger, anterior septal or aortic leaflet, and the lesser, posterior or mural

valve is thus similar to the human lips. when open it is conical, when closed, slitlike in shape.

Pathology.—The sequence of events which leads to mitral stenosis is not too clearly understood. It is postulated that during acute rheumatic fever, vegetations involve all points of contact of the leaflet surfaces.

Magarey feels that these irregularities cause blood eddies which may initiate further deposits of fibrin, thus explaining the gradual thickening of the valve leaflets and narrow

ing of the aperture that occurs over the intervening years.

The suggestion that mitral stenosis may develop without an initiating attack of rheumatic fever received support from the fact that 7% of normal valve leaflets have fibrin deposits. Further, 45% of patients operated upon for mitral stenosis give no history of rheumatic disease and 5% a history of scarlet fever.

Prior to leaflet edge fusion the commissures are only present when the valve leaflets are approximated during systole. Disease fuses the leaflets, forming true lateral and medial commissures. The mitral orifice may be reduced to 3-5 mm in diameter from its normal size of 1.5-2 cm. Fusion and shortening of the chordae tendineae may form subvalvular stenosis.

Changes in the papillary muscle, chordae tendineae, and valve leaflets which prevent accurate valve closure may produce an associated mitral insufficiency.

Physiopathology.—A narrowed mitral valve interferes with the free passage of blood from the left atrium to the left ventricle. The pressure in the left atrium increases, as does that in the pulmonary vessels and right heart. The strain thrown upon the left atrium causes hypertrophy, progressing to dilatation in most cases. The left ventricle receives less blood and becomes smaller, and the systemic blood pressure tends to be low. The back pressure in the pulmonary arteriolar bed may be reflected as pulmonary edema or hemoptysis. This pressure usually causes enlargement of the pulmonary artery. The right ventricle, pumping against increased resistance hypertrophies, may fail. Right ventricular failure may lead to functional tricuspid valve insufficiency and its sequelae.

Atrial fibrillation increases the pooling of blood in the atria and favors the formation of thrombi in right and left atria and particularly in the left atrial appendage. These may be dislodged to embolize to the brain, other viscera, and extremities. Thrombi leaving the right atrium cause pulmonary infarction. It must be remembered, however, that in advanced mitral stenosis, pulmonary emboli originate more often from the stagnant peripheral veins.

Clinical Picture.—Only about 50% of the patients will give a history of rheumatic fever. The cardinal symptom of mitral stenosis is dyspnea. This changes, as the disease progresses, from mild dyspnea on exertion to severe orthopnea. Hemoptysis, varying from blood-tinged sputum to pulmonary apoplexy, and pulmonary edema are common. Frequently there are repeated episodes of bronchitis and bronchopneumonia. There may be precordial soreness or pain and in the older patients, pain similar to angina pectoris.

Systemic embolism is seen in 9-14% of the cases, of which 60% are to the brain. The right ventricle may fail, and edema and swelling of the abdomen may become the predominant signs.

Physical examination reveals a rumbling diastolic murmur at the apex, a loud M_1 , and an opening snap. P_2 may be accentuated. Peripheral cyanosis and mitral facies, small radial pulse, and mitral thrill are usually present. Examination for evidence of ventricular failure should be made. Atrial fibrillation is common.

The pulmonary circulation time is usually prolonged, but its determination adds little to the diagnosis.

X-ray usually reveals the typical mitral pattern. Fluoroscopy shows more clearly the enlarged left atrium, pulmonary artery, and right ventricle. Pulmonary congestion, a hypodynamic left ventricle, and calcification of the valve may also be seen. A large or giant left atrium, particularly with left ventricular enlargement, indicates mitral insufficiency rather than stenosis.

The electrocardiogram will show right axis deviation in about 80% of the patients with pure mitral stenosis. A prominent P mitral and a right ventricular strain pattern, when present, point to the diagnosis.

Differential Diagnosis.—Classical mitral stenosis offers little difficulty in diagnosis. The diagnostic problem is more that of the detection of associated mitral insufficiency, the presence of other valve lesions, and the evaluation of the state of the ventricular myocardium. Certain conditions, however, must be excluded when mitral stenosis is not entirely typical.

Mitral insufficiency may be pure or associated with stenosis. When both lesions are present it is often difficult to determine which one predominates. Left heart catheterization has not been of sufficient diagnostic help nor has visualization of retrograde flow of radio-paque media. New techniques in dye-dilution studies, at present under trial, may hold the answer to this problem.

Essential pulmonary hypertension characteristically has right heart strain, an enlarged pulmonary artery, but a small left atrium. Right heart catheterization and study of pulmonary capillary pressures will help to differentiate this condition. Pulmonary capillary pressures are minimal in essential pulmonary hypertension and elevated in mitral disease.

Tumors of the left atrium when pedunculated may cause intermittent obstruction of the valve and moderate atrial enlargement. Often symptoms are improved when the patient lies down, as the tumor falls out of the valve orifice.

Cor triatrium. Occlusion of 50% or more of the pulmonary veins will give all the symptoms and signs of mitral stenosis except for enlargement of the left atrium.

Surgical Procedures.—The scope of medical management of mitral stenosis is constantly being redefined as surgical techniques improve. There are, no doubt, a number of patients with narrowed mitral valves who may live for many years with adequate medical treatment, and some may do so with little or no restriction of normal activity. In the majority of cases, the strain imposed upon the heart is cumulative, and eventually, as work tolerance is decreased, the patient may become totally incapacitated. Surgical intervention, to be of the greatest value, must be undertaken before irreversible pulmonary and cardiac changes have developed.

MITRAL COMMISSUROTOMY Indications

- 1 Exertional dyspnea
- 2 Hemoptysis
- 3 Progressive fatigue
- 4 Peripheral embolization
- 5 Auricular fibrillation
- 6 Repeated attacks of bronchopneumonia
- 7 Pregnancy, when previous pregnancy associated with a break in cardiac compensation

- 8 Cardiac enlargement, evidence of right heart strain
- 9 Associated stenosis of aortic, tricuspid, and rarely, pulmonic valves
- 10 Associated valve insufficiency of.
Aortic, slight
Pulmonic, slight
Tricuspid, marked, functional

Contraindications

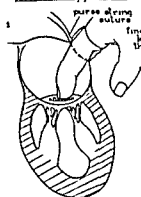
- 1 Left ventricular failure
- 2 Rheumatic disease activity
- 3 Uncontrolled right ventricular failure
- 4 Recent peripheral embolization (await stabilization and full recovery)
- 5 Uncontrolled auricular irregularities (await proper digitalization)
- 6 Blood volume elevated 15% or more above normal to be corrected
- 7 Associated marked valve insufficiency of:
Mitral
Aortic
Tricuspid

PRINCIPLES.—The reconstitution, so far as possible, of valve function is carried out by (1) separating the fused aortic and mural leaflets in the lateral and medial commissures, (2) sorting out fused chordae tendineae, and (3) dividing, if necessary, fused papillary muscles.

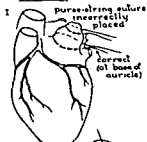
TECHNIQUE.—The thorax is opened through a left anterolateral or posterolateral intercostal incision and the pericardium opened parallel to the phrenic nerve. A purse-string suture is placed around the base of the left atrial appendage, which is then incised and the right index finger inserted into the atrium. After careful exploration and assessment of the valve mechanism, the mitral orifice is enlarged by fracturing the fused lateral commissure, followed by the medial commissure. This is *digital commissurotomy* or *finger fracture*. In the majority of cases it is necessary to use a knife to start or complete the fracture.

Chordae tendineae are separated when fused and papillary muscles cut to correct subvalvular stenosis. When the valve orifice has been increased in diameter to 2-2½ fingerbreadths, the leaflets usually function well, unless they are heavily scarred or calcified. During actual fracture it is customary to have the anesthetist occlude both carotids in an effort to decrease the possibility of cerebral embolism.

left sided approach



errors



II if left auricle too small may tear under pulmonary artery as towards pulmonary veins as towards circumflex artery



to avoid tearing

1 enlarge opened auricle



2 place no cotton sutures in dangerous areas



III clot not allowed to 'blow out'



clot breaks when left auricle clamped, free piece continues on to left ventricle

errors

mitral commissurotomy



1 cut in mitral leaflet producing insufficiency

2 subvalvular stenosis overlooked



3 commissures insufficiently opened

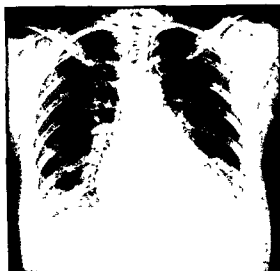
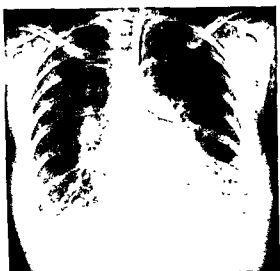


Fig. 261—A, Mitral commissurotomy through the left atrium (From Vineberg, A, Gialloredo, O, and Loberge, J, in Thorek, Max. Surgical Errors and Safeguards, ed. 6, Philadelphia, J. B. Lippincott Co)

B and C, Case of Grade IV mitral stenosis before (B) and after (C) mitral commissurotomy. Note decrease in size of pulmonary artery and clearing of lung field

The approach through the left atrial appendage is the easiest and most commonly used. However, Bailey advocates entering the thorax through the 4th right anterior intercostal space and the left atrium in the interatrial groove. He claims that the right-sided atrial groove permits exploration of the tricuspid valve which is sometimes involved, the treatment, if necessary, of aortic stenosis, and, finally, better access to the medial commissure. Further, in certain cases due to scar, calcification, or previous operations, the left atrial appendage is too small to enter. The disadvantages of this right-sided approach are that it is more difficult and dangerous technically, and in addition the surgeon is unable to remove thrombi in the left atrial appendage.

COMPLICATIONS.—

Operative.—

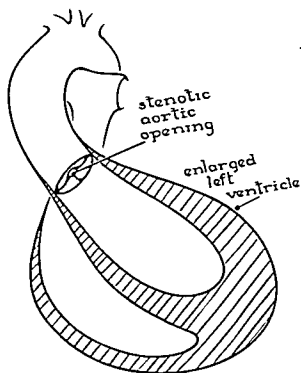
Peripheral embolization Thrombi may be dislodged from the atrial appendage or aortic wall. These should be removed before inserting the finger. Emboli also originate from calcified particles released at the time of commissurotomy.

Production of mitral insufficiency. This may occur when the correct line of fracture is missed and a leaflet is damaged, or when chordae tendineae or papillary muscles are accidentally cut.

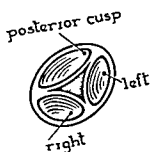
Tear of the atrial or left ventricular wall. This results in severe blood loss.

Damage to circumflex branch of left coronary artery.

Cardiac arrest.



normal aortic valve:



aortic stenosis



calcified aortic stenosis

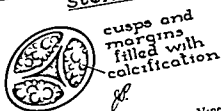


Fig 262A-D.—A. Aortic stenosis (From Vineberg, A., Gialloreti, O., and Laberge, J., in Thorax, Max Surgical Errors and Safeguards, ed 6, Philadelphia, J B Lippincott Co)

Postoperative.—

1. Pulmonary edema
2. Pericardial and pleural fluid
3. Pneumonia, empyema, and septicemia
4. Peripheral embolization
5. Secondary hemorrhage
6. Electrolyte imbalance
7. Cardiac irregularity
8. Postcommisurotomy syndrome
9. True flare-up of rheumatic fever
10. Late recurrence of mitral stenosis

RESULTS—The results of mitral commissurotomy are influenced by the following:

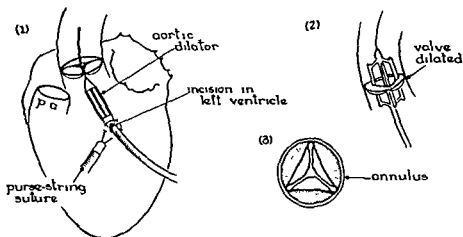
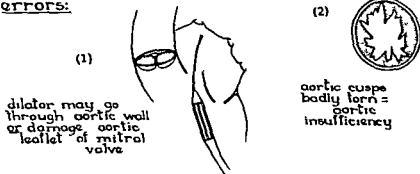
1. *Extent of mitral commissurotomy*: the character of the valve at the time of operation and the skill and experience of the surgeon

2. *Presence of other valvular disease*, aortic or tricuspid

3. *Degree of irreversible damage* to the myocardium and pulmonary arterioles

DISCUSSION.—In a minority of cases, the chordae and papillary muscles are so altered that it is anatomically impossible to obtain by any means, short of valve substitution, a satisfactory functioning mitral valve. Bailey reports that in his first 1,200 cases there was, in addition to fusion of the commissures, a 20% incidence of a significant degree of sub-valvular obstruction, due to a complex inter-fusion of the chordopapillary supporting tissues

He reports that 65-75% of 1,000 patients treated by mitral commissurotomy and followed up to 8 years have experienced long-term benefit. There were 7.7% operative deaths and a 5.8% late mortality. Of the original 1,000, 71.1% (89.8% of the sur-

errors:

B.

Fig 262 (cont'd)—Aortic stenosis B, Indirect (transventricular) approach for aortic commissurotomy (From Vineberg, A., Gialloretto, O., and Laberge, J., in Thorek, Max Surgical Errors and Safeguards, ed 6, Philadelphia, J B Lippincott Co)

vivors) were improved, 6.2% were the same, and 4% were worse. Roughly, 10-15% of the patients are not helped by operation. Many poor results were due to inadequate commissurotomy and failure to detect and correct associated lesions of the other valves.

Aortic Stenosis

Definition.—The term aortic stenosis has been used clinically to imply narrowing the outflow tract of the left ventricle. In many instances this is caused by disease of the aortic valve, but occasionally it may be due to

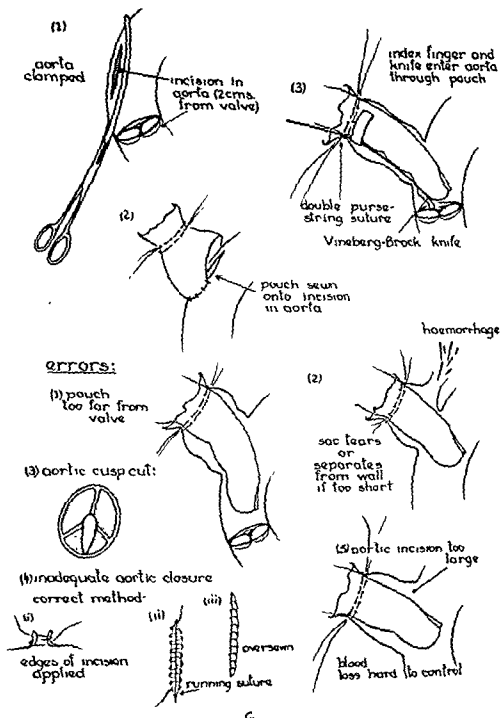


Fig 262 (cont'd) —Aortic stenosis C. Indirect (supra-aortic) approach for aortic commissurotomy. (From Vineberg, A., Gialloreti, O., and Laberge, J., in Thorek, Max: *Surgical Errors and Safeguards*, ed. 6, Philadelphia, J. B. Lippincott Co.)

to a congenital condition involving the sub-valvular region.

Incidence.—Acquired aortic stenosis comprises 40% of all deformities of the valves and occurs in a ratio of 4:1 in the male. The average age of death is 45 to 50 years, being approximately 15 years later than that of mitral stenosis.

Etiology.—Two different causes are given for acquired aortic valvular stenosis: rheumatic valvulitis, considered to be the more frequent cause, and aortic valvular sclerosis, said to be the degenerative disease (Mönckeberg).

Anatomy.—The aortic valve is formed by three semilunar-shaped cusps, attached peripherally to the aortic ring. During ventricular systole the ejected blood flattens the cusps against the aortic wall, during diastole they fall back to meet in the center, preventing the return of blood to the ventricle. There are three commissures: the anterior, which is in

line with the posterior commissure of the pulmonary valve, and a right and a left posterolateral. Each cusp, when distended, forms a sinus (Valsalva) between itself and the aortic wall. The two main coronary arteries arise from the two anterior sinuses. No artery leaves from the posterior sinus.

Pathology.—The state of the valve in acquired disease may be classified as follows:

Fusion of commissures with intact cusps. The valve leaflets are still intact and pliable, and the obstructive effect is caused mainly by the fusion of the commissures. This group gives the best results following surgery.

Fusion of commissures with damaged cusps. In addition to interfusion, the cusps are thickened, scarred, wholly or partially calcified, and may be retracted to cause an associated insufficiency. Such valves, despite adequate surgical separation of the fused commissures, may give poor functional results.

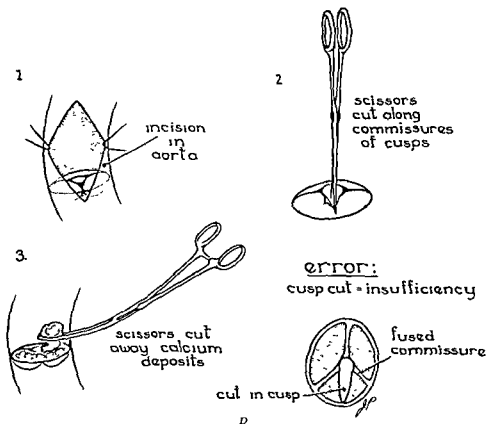


Fig. 262 (cont'd) —Aortic stenosis. D, Direct approach for aortic commissurotomy (From Vineberg, A., Gialloreti, O., and Laberge, J., in Thorek, Max: *Surgical Errors and Safeguards*, ed 6, Philadelphia, J. B. Lippincott Co.)

Damaged cusps without fusion of commissures. This condition is believed to be caused mainly by Mönckeberg's sclerosis of the valve, i.e., a degenerative rather than a rheumatic disease. The cusps are thickened, rigid, and often completely calcified, but they remain unfused. The effect of aortic stenosis is produced by their immobility.

Physiopathology.—The left ventricle becomes concentrically hypertrophied, and its intraluminal pressure rises to overcome the obstruction of the outflow tract. Blood pressure and cardiac output are low, and there may be inadequate coronary blood flow. The resultant myocardial ischemia may produce angina pectoris, myocardial degeneration, or sudden ventricular fibrillation. The continued strain on the left ventricle and its precarious blood supply eventually lead to ventricular failure. The jetlike blood stream ejected from the stenotic valve causes a postvalvular dilatation of the aorta.

Clinical Picture.—The patient may remain asymptomatic for many years. When the valve opening has been reduced to approximately 25% of its normal size, compensatory mechanisms begin to fail, and dyspnea on effort, angina pectoris, vertigo, and syncope occur with increasing severity. When the orifice narrows to 10% of its normal size, the clinical course is usually rapidly downhill. Death may occur from congestive failure, sudden ventricular fibrillation, or bacterial endocarditis.

Physical Signs.—A systolic thrill may be felt in the aortic valve area and up into the right carotid. The typical, short, harsh systolic murmur, heard best in the 2nd right interspace near the sternum, may be transmitted to the carotid arteries and to the apex. The pulse is small and sustained, and the systolic and pulse pressures are usually low. A_2 is split or even absent. Mitral stenosis is also found in approximately 38% of cases, and by decreasing the strain on the left ventricle it may mask the aortic valve disease.

Laboratory Findings.—In early cases, x-ray reveals globular hypertrophy of the left ventricle and usually a poststenotic aortic dilatation. In the advanced case, left ventricular dilatation, pulmonary engorgement, and other signs of heart failure may be seen.

The electrocardiogram shows left axis deviation or left ventricular strain. When marked mitral stenosis is present, a mid or right axis deviation may be seen.

The pressure gradient across the aortic valve may be measured (see cardiac catheterization). A gradient of 40 mm. Hg or more indicates marked stenosis, and surgery is warranted. Concomitant mitral stenosis and a failing myocardium, however, can produce a small pressure gradient, despite severe aortic stenosis.

Surgical Treatment.—The objective is to open the fused commissures and to re-establish cusp function. When the cusps themselves are badly scarred and immobile, little relief from surgical measures can be expected short of valve replacement.

In those patients with rheumatic aortic valvulitis, the commissures are generally solidly fused, and if the valve cusps are not too thick and rigid, opening of the commissures favors cusp action and at least removes the obstruction to forward blood flow. When there is marked calcification of the valve cusps, opening of the commissures may result in little relief of aortic stenosis since the rigid cusps fail to yield during systole, in the same way as do the nonfused arteriosclerotic cusps.

SURGICAL METHODS OF AORTIC COMMISSURE TOMY —

The Transventricular Route.—A valvulotome and dilator are passed through a small incision in the left ventricle and into the aortic valve. The main disadvantages of this method are that the valve cannot be palpated, that the procedure is entirely blind, and that ventricular fibrillation frequently occurs when an already irritable myocardium is manipulated. Uncontrollable hemorrhage from the site of valvulotomy is not uncommon.

Supra-aortic Approach.—An opening is made in the anterolateral surface of the aorta just distal to the valve to which a pouch is sewn. Through this pouch the index finger explores the valve and in many instances opens it. In other instances the commissures have to be cut with a knife or forced open with a dilating instrument.

Valvuloplasty Under Direct Vision.—This technique necessitates arrested circulation, either under hypothermia or preferably with

a pump-oxygenator. The valve cusps may be accurately separated and trimmed of excessive calcified tissue (as for pulmonic stenosis, see Fig. 246).

RESULTS.—Results of aortic commissurotomy vary according to the severity of the disease and the type of commissurotomy performed. Mortality is about 28% for the transventricular approach and 14% for the transaortic approach. Clinical improvement occurred in over 80% of the survivors

Incidence.—Significant tricuspid stenosis occurs in roughly 6% of cases of mitral stenosis submitted to surgery, and a minor degree of stenosis occurs in another 14%.

Physiopathology.—The pathology is similar to that of mitral stenosis. The right atrial pressure is elevated and causes venous engorgement, hepatomegaly, ascites, and peripheral edema. It must be remembered that this lesion is almost always associated with mitral stenosis and that the obstruction of tricuspid stenosis

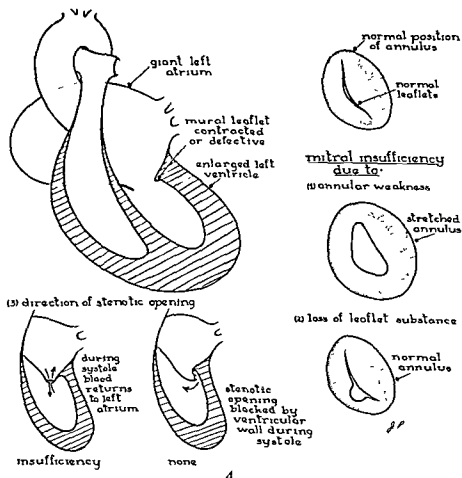


Fig. 263A-G—A, Mitral insufficiency (From Vineberg, A, Gialloreti, O, and Laberge, J, in Thorek, Max Surgical Errors and Safeguards, ed 6, Philadelphia, J. B. Lippincott Co.)

Tricuspid Stenosis

Etiology.—Rheumatic fever is the commonest cause. Isolated tricuspid stenosis is so rare that, if found, other less common causes such as atrial tumor or malignant carcinoid should be suspected.

may mask many of the pulmonary symptoms that characterize mitral disease.

Symptoms.—The symptoms are similar to those for tricuspid insufficiency except for the character of the murmur. The jugular pulse has characteristic giant A waves. Heart catheterization may confirm the diagnosis.

Treatment.—Treatment is similar to that for mitral stenosis, i.e., separation of the fused commissures. Because of its association with other valve lesions, it has been suggested that whenever a mitral commissurotomy is performed, the tricuspid valve should be explored.

Valvular Insufficiency

Unlike valvular stenosis, valvular insufficiency is very difficult to correct surgically. At present there is great doubt as to the value of the numerous surgical procedures devised to treat valvular incompetence.

scarred and retracted that they do not meet properly at the valve closure.

Destroyed Chordopapillary Mechanism.—Rupture of the chordae produces an unstable leaflet that cannot control reflux. Extreme scarring and contraction of chordae may prevent the leaflet from approximating with its mate.

Valvular Stenosis.—Fusion of leaflets may produce a rigid narrow valve, which, in addition to obstructing flow, permits regurgitation.

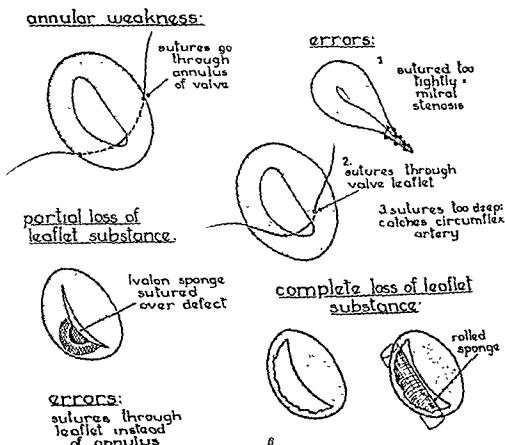


Fig. 263 (cont'd) —Mitral insufficiency B, Operative treatment, using the extracorporeal circulation (From Vineberg, A., Gialloreti, O., and Laberge, J., in Thorek, Max: *Surgical Errors and Safeguards*, ed. 6, Philadelphia, J. B. Lippincott Co.)

Pathology.—The following types of lesions may occur alone or in combination.

Stretched Valve Annulus.—Disease of the annulus permits stretching of this structure, causing separation of the leaflets which may be normal. This type is also seen in functional insufficiency.

Loss of Valve Substance.—The annulus may be normal, but the leaflets and cusps are so

Mitral Insufficiency (Regurgitation)

A significant degree of insufficiency has been found in 11% of cases explored for mitral stenosis. The presence of insufficiency materially alters the prognosis for patients undergoing mitral commissurotomy. It is frequently impossible to determine preoperatively whether stenosis or incompetence predominates or which of the mechanisms mentioned above



C.



D.



E.



F.

Fig 263 (cont d) —Mitral insufficiency

C and D, Posteroanterior x-rays before and after open heart repair of mitral valve by annuloplasty. Note marked diminution in size of left atrium. Extracorporeal circulation was used in this case.

E and F, Same case, lateral views. Observe how the posterior displacement of the esophagus is diminished following mitral annuloplasty.

has caused the insufficiency. Since correction of mitral stenosis may improve certain types of mitral insufficiency, exploratory operation may be justified in doubtful cases

Clinical Picture.—The picture is similar to that for mitral stenosis but with perhaps fewer respiratory symptoms initially. When deterioration does occur, the downhill course is rapid. A loud systolic murmur radiating from apex to axilla is characteristic. Electrocardiography reveals left ventricular preponderance. Fluoroscopy shows an enlarged left ventricle and a very large or giant left atrium which may have a systolic expansion.

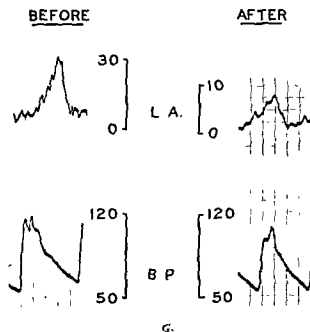


Fig. 263 (cont'd) —Mitral insufficiency

G. Intra-atrial pressure in patient shown in C-F, before and after mitral annuloplasty. Note the reduction in average systolic pressure in the left atrium

Treatment.—Numerous operations have been devised and most of them discarded. Three techniques under evaluation at the present time seem promising. Unfortunately they all have two points in common, i.e., a high mortality and equivocal results

Circumferential Tape Suture—The annulus may be constricted by suturing an umbilical tape circumferentially around the left arterio-venous groove. This is tightened until the leaflets meet

Cross Polar Ligation—The annulus is constricted by inserting two heavy sutures through

it at its medial end. This is perhaps the best of the closed methods.

Open Reconstruction.—By the use of extra corporeal circulation, the valve is visualized; the leaflet defects are filled with Ivalon sponge and the annulus narrowed. This technique, when perfected may be the treatment of choice

Aortic Insufficiency

Etiology.—Rheumatic fever is said to cause 67% and syphilitic aortitis 19% of cases presenting aortic insufficiency.

Clinical Pathophysiology.—Blood leaks back into the left ventricle during diastole which, with the blood entering from the left atrium, places an extra load upon the left ventricular fibers. There is a rapid ejection of blood into the aorta during systole with a diastolic fall of aortic pressure. The regurgitation of blood into the left ventricle increases the end diastolic ventricular pressure, making it more difficult for blood to enter the left ventricle from the left atrium. The resulting left atrial hypertension causes pulmonary hypertension

Clinical Picture.—At first the patient may be asymptomatic, but as left ventricular strain develops there may be paroxysmal attacks of dyspnea, acute pulmonary edema, hemoptysis, and anginal pain. An early blowing diastolic murmur, heard best at the 2nd right intercostal space close to the sternum, is transmitted down the left sternal border; an Austin Flint murmur may be heard at the apex as a mid-late diastolic murmur; a water-hammer pulse, an elevated pulse pressure, and low to no diastolic pressure with capillary pulse are characteristic. In advanced cases the patient's head is often observed to nod with each cardiac beat

X-ray shows a characteristic bootlike shadow due to enlargement of the left ventricle. Brachial artery tracings are likewise characteristic.

Treatment.—The Hufnagel prosthetic ball valve, placed in the descending aorta beyond the left subclavian artery, is the best treatment now available. The valve relieves the left ventricle of part of its load but fails to protect the coronary or cerebral vessels from the effects of aortic insufficiency. Deaths have been

reported from coronary insufficiency, erosion of the prosthesis through the aorta, and peripheral emboli. This form of treatment is still highly experimental

Multiple Valve Lesions

Rheumatic fever may involve all four heart valves, either singly or in combination. Autopsy studies show that disease (stenosis, insufficiency, or both) may involve the valves with the following frequency: aortic and mitral 19.2%; aortic, mitral, and tricuspid 11%; mitral and tricuspid 2.9%; pulmonary and tricuspid 1%, all four valves 1%.

Mitral and Aortic Stenosis

Mitral stenosis has been reported by Bailey to occur in 38% of patients with aortic stenosis, or 15% of all acquired valvular disease.

Physiopathology.—The obstruction of the mitral valve decreases the flow of blood into the left ventricle and masks the signs of aortic stenosis. The elevated left ventricular diastolic pressure augments the effect and may even intensify the picture of mitral stenosis. The pressure gradient across the aortic valve may be low because of the protective effect of the mitral stenosis, and the unwary clinician may underestimate the severity of the aortic valve lesion.

Diagnosis.—The characteristic signs and symptoms of each lesion are present in a greater or lesser degree. The ECG may show preponderance of either ventricle but more often of the right. Left heart catheterization and measurement of the pressure gradients across the valves will confirm the diagnosis.

Surgical Treatment.—At present, closed techniques seem to give the best results. The following approaches may be used:

1 *Vertical sternal splitting incision* gives adequate access to the aortic valve but makes mitral valvotomy difficult.

2 *A left anterolateral thoracotomy* is excellent for the mitral valve and permits transventricular aortic valvotomy.

3 *A right anterolateral thoracotomy*, while good for the transaortic valvotomy, necessitates approaching the mitral valve through the inter-

atrial sulcus. The tricuspid valve is easily explored if indicated.

4. *Bilateral anterior thoracotomy with division of sternum* gives adequate exposure for all valves and permits any technique of valvotomy desired.

Whatever the approach, the aortic valve is first opened, following the principle of first relieving distal obstruction. The mitral commissurotomy may then be performed when the heart has stabilized.

The results are surprisingly good following commissurotomy of both valves. Mortality is about 18%, and 31% of the patients are markedly improved and 51% improved. Objective evidence of the adequacy of valvotomy may be obtained by taking cross-valve pressure recordings several months postoperatively.

Mitral and Tricuspid Stenosis

Incidence.—Tricuspid stenosis is said to occur in about 20% of cases of mitral stenosis, although in only 6% is the stenosis severe. In general, results of commissurotomy for combined mitral and tricuspid stenosis are most satisfactory.

CORONARY ARTERY HEART DISEASE

Definition.—This is an acquired disease caused by arteriosclerotic narrowing or occlusion of the coronary arteries. A distinction should be made between the terms *coronary artery disease*, in which only the coronary arteries are diseased, and *coronary artery heart disease*, in which both coronary arteries and the myocardium are involved.

Incidence.—Heart disease is the leading killer in the United States. In 1953, 65% of the 566,420 persons dying from heart disease had arteriosclerosis as the basic cause. At autopsy, 1 of every 4 hearts showed evidence of coronary artery disease and 1 in 10, of infarction. Males predominate 4:1.

Anatomy

The main coronary arteries arise from the aorta in the sinuses of Valsalva and branch over the right and left heart to form a corona. The major branches are on the surface and can readily be seen pulsating. The branches of the coronary arteries penetrate the myocardium and form four arteriolar zones. In

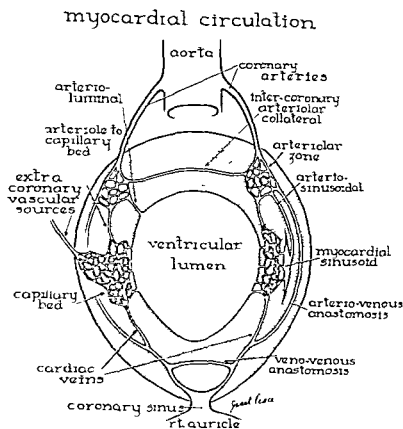


Fig 264.—Diagrammatic representation of coronary circulation showing its complexities

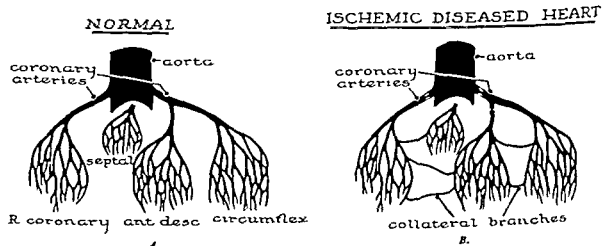


Fig 265.—A, Arteriolar zones in the normal heart. Note absence of collaterals
B, Arteriolar zones in the diseased heart. Note presence of collaterals

normal hearts these arteriolar networks do not usually communicate. In addition there is some evidence to suggest that an individual spiral muscle is supplied by its own arterial branch.

The majority of arterioles end in capillaries which surround and supply the muscle fibers. Some, the arteriololuminal, by-pass the capillary system and communicate directly with the cardiac chambers. Others, the arteriosinusoidal, join the myocardial sinusoids lying between the muscle bundles. These large endothelial-lined spaces communicate with the arteriolar system and with the capillaries surrounding the muscle fibers.

Only 60% of the arterial blood entering the myocardium is recovered from the coronary sinus. The remainder is believed to reach the heart chambers by anterior cardiac veins and thebesian canals. The thebesian canals are venouslike spaces joining the capillary systems and ventricular lumina.

Extracardiac Sources of Blood.—There are capillary communications between the vessels supplying the pericardium and the coronary circulation. These are small and functionally insignificant. The coronary arterial flow is influenced by differences in pressure in the aorta and in the myocardial arteriolar network. A lowered aortic diastolic pressure markedly decreases the rate of coronary flow. Further the differences in pressure between one arteriolar zone and another seem to favor the formation of intercoronary anastomoses.

Pathology

Arteriosclerosis when it occurs is usually in the first 3-5 cm of the coronary vessels and is generally confined to the epicardial portions. The vast myocardial arteriolar network is, for the most part, disease free. Normally, there is no communication between the four arteriolar zones. However, when ischemia develops, intercoronary anastomoses open up to form collaterals.

The fate of an ischemic zone depends on (1) the size of the vessel involved, (2) the rate of narrowing of the supplying coronary artery, (3) the rate of development of col-

laterals, and (4) the demand of the ischemic area for oxygenated blood.

Acute Myocardial Infarction.—Sudden occlusion or narrowing of a large or even small vessel may result in death from cardiac arrest. Survival of the patient is almost always accompanied by infarction of the ischemic myocardial area. There appear to be two main types of acute myocardial infarction: one which involves the whole thickness of the ventricular muscle, and the other, a laminated area of necrosis. In the latter type there may be a healthy zone of muscle on the surface. In general, an interval of six weeks is required for removal of necrotic muscle and at least six months for the establishment of a collateral circulation.

Chronic Myocardial Infarction.—Slow progressive occlusion of a coronary artery may not result in any myocardial change, providing the pressures in the adjacent arteriolar zones favor the development of arteriolar anastomoses.

Unfortunately, atherosclerosis generally involves more than one major coronary artery, and the good neighbor arteriolar zone is little better off than the affected area. In such cases repeated small losses of muscle fibers may result in the almost complete fibrous replacement of the ventricular myocardium.

Myocardial fiber loss, when gradual, may at first pass unnoticed. Gradually the myocardial reserve is reduced, and eventually the left ventricle dilates and fails. Patients with this condition are beyond help. It is too late once the myocardium has been reduced to scar tissue to obtain benefit from introducing fresh extracardiac sources of blood.

Localized areas of fibrosis may dilate to form ventricular aneurysms.

Angina Pectoris

Angina pectoris is a typical substernal or precordial pain radiating to the left shoulder or arm, initiated by exertion and relieved by rest or nitroglycerin. It was first described by Heberden. The mechanism in the production of anginal pain is not clear. There is general agreement that this symptom reflects myocardial ischemia, usually caused by nar-

rowed coronary arteries. Sustained chest pain, anginal in character, is generally caused by myocardial infarction.

Coronary artery disease may be present and be asymptomatic. Likewise coronary artery heart disease may develop to a far-advanced stage with marked myocardial ischemia and destruction, without the patient's experiencing anginal pain.

or the treadmill exercise may leave the diagnosis in doubt. Unfortunately most observers do not live long enough to learn from pathologic studies the true effect of a given treatment in the alleviation of a chronic disease.

Surgery of Coronary Artery Heart Disease

During the past 50 years many attempts have been made by surgeons to help their

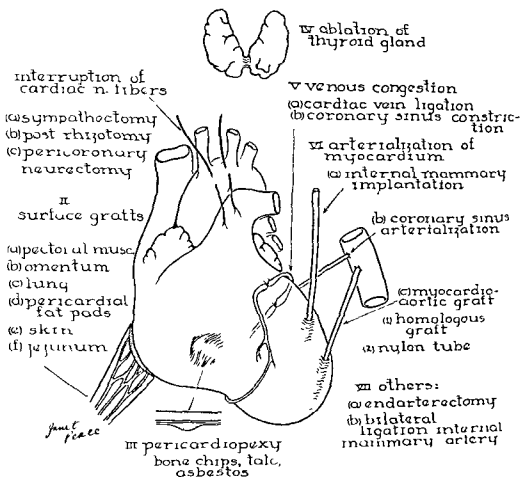


Fig 266—Illustration of various operative procedures for the treatment of coronary artery heart disease

Chest pains that are not initiated by physical activity or emotion, are of long duration, and do not disappear quickly with rest or nitroglycerin are not caused by myocardial ischemia and should be regarded with suspicion. A search for hiatus hernia, biliary tract disease, peptic ulcer, cervical or upper thoracic discs, and neurosis may disclose the source of the patient's pain. The electrocardiogram is of value when it is positive. Failure to obtain ECG evidence of coronary artery disease with the Master's two-step test

medical colleagues in the treatment of coronary artery heart disease. The following surgical procedures have been attempted:

- 1 Neurosurgery to interrupt afferent pain paths
- 2 Reduction of thyroid function
- 3 Development of collaterals and/or anastomoses
 - a Ligation of cardiac vein
 - b Ligation of coronary sinus
 - c Bilateral internal mammary artery ligation
- 4 Direct attack on diseased coronary arteries
 - a Resection and graft substitution of occluded coronary artery
 - b Endarterectomy

- 5 Myocardial vascularization by extracardiac blood
- Surface grafts applied to the heart: pectoral muscle, omentum, lung, pericardial fat pad, skin, jejunum
 - Cardiopericardiopexy (pericardial poudrage), bone chips, asbestos, talc
 - Ventricular arterIALIZATION by vascular implants
 - Internal mammary artery implant
 - Splenic artery implant
 - Carotid artery implant, or
 - Graft from aorta to myocardium

procedure frequently relieves anginal pain but fails to improve myocardial ischemia.

The concept of reducing the demands of the heart by causing hypothyroidism is badly founded. The progress of the disease is not arrested and may be increased. The failure of this method to increase the blood supply to the heart explains why there is no improvement in the patient's exercise tolerance.

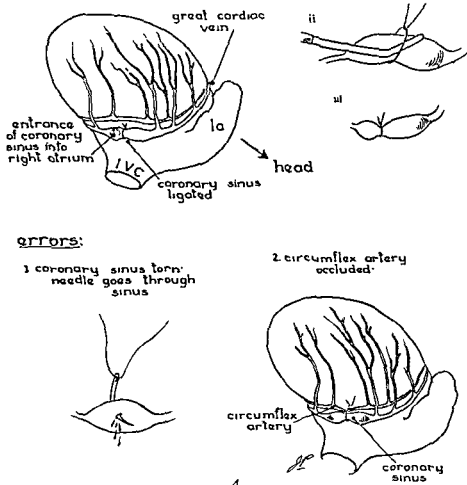


Fig. 267A C —Beck I operation for coronary artery insufficiency

A, Partial ligation of coronary sinus (From Vineberg, A, Gialloreti, O, and Laberge, J, in Thorek, Max Surgical Errors and Safeguards, ed 6, Philadelphia, J B Lippincott Co)

There are four objectives for surgery in the treatment of coronary artery heart disease (1) to relieve angina pectoris, (2) to increase exercise tolerance and ability to work, (3) to prevent further loss of myocardial fibers, and (4) to prolong life expectancy.

Neurosurgery is carried out at times to block afferent pathways of cardiac pain. This

The encouragement of the development of collaterals should not be considered a basic treatment but part of a procedure which distributes arterial blood to the heart without introducing extracardiac blood.

Coronary artery resection and endarterectomy are procedures that are still highly experimental.

Surgical revascularization methods attempt to by-pass points of coronary artery obstruction by introducing an extracardiac source of blood to the intact ventricular arteriolar zones. In practice, the development of channels which carry a volume of blood sufficiently large to relieve myocardial ischemia is difficult. The following three methods are in current use.

tion of coronary sinus to promote intercoronary anastomoses, (b) removal of epicardium and sprinkling with irritating asbestos powder over the left ventricle, (c) application of pericardial fat pad grafts to the surface of the left ventricle. This operation has been shown experimentally to distribute blood evenly throughout the myocardium.

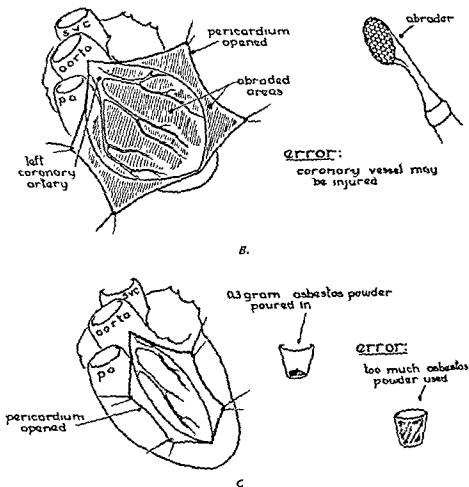


Fig. 267 (cont'd).—Beck I operation for coronary artery insufficiency.

B, Pericardium and epicardium scraped

C, Pericardio-cardioplexy.

(From Vineberg, A., Gialloreti, O., and Laberge, J., in Thorek, Max: *Surgical Errors and Safeguards*, ed. 6, Philadelphia, J. B. Lippincott Co.)

1. **Cardiopericardioplexy.**—In this operation the left 6th intercostal cartilage is removed, the pericardium is opened, and talc powder is introduced into the pericardial sac. This procedure forms granulomatous adhesions between the serous layers.

2. **Beck I Operation.**—The Beck I operation is performed as follows: (a) partial liga-

3. **The Vineberg Internal Mammary Artery Implantation.**—(a) The left internal mammary artery is detached from the chest wall. (b) The internal mammary artery is pulled into a left ventricular tunnel after the 6th intercostal artery has been severed, thus leaving an open bleeding vessel in the myocardium. (c) Supplementary fat pads are

sewn to the surface of the left ventricle after the removal of the epicardium by sharp dissection.

With regard to the Vineberg operation it has been proved experimentally that the internal mammary artery remains open after implantation into the ischemic myocardium because it bleeds into the myocardial sinusoids. Within 12 days it branches to form true

arterioles, which later join with the arterioles within the heart muscle. The amount of arteriolar blood delivered is sufficient to prevent death or infarction in the experimental animal subjected to coronary artery occlusion. Further, the artery has been shown to remain open with little or no intimal proliferation in both animal and man when examined up to 3½ years after implantation.

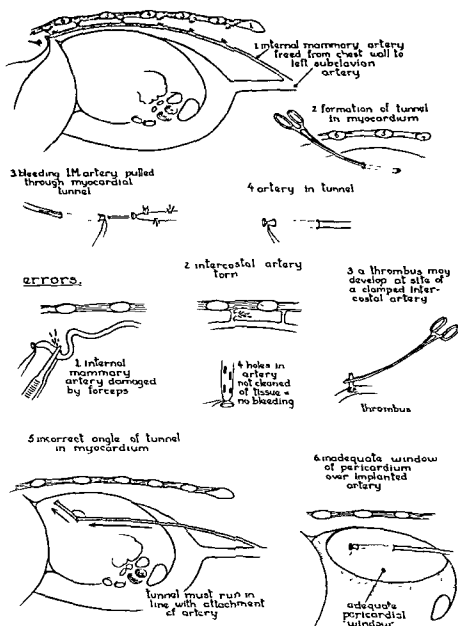


Fig. 268—The Vineberg internal mammary artery implant operation for the treatment of coronary artery insufficiency (From Vineberg, A, Gialloreti, O, and Laberge, J, in Thorek, Max Surgical Errors and Safeguards, ed 6, Philadelphia, J B Lippincott Co.)

Recently, using the principle of internal mammary artery implantation, a few patients with coronary artery heart disease have been treated by inserting a nylon tube graft from the aorta into the left ventricular wall. Italian workers, utilizing the same principle, have placed a polyethylene T tube into the left ventricle, joining the left ventricular lumen to the myocardial sinusoidal system.

Selection of Patients for Remedial Surgery.—This should be largely dependent on the stage of development of the patient's disease, and the following criteria are suggested

CONTRAINDICATIONS FOR SURGERY.—

1. Asymptomatic coronary artery disease. This view, as mentioned, is not shared by all.
2. Recent infarction or evidence of discrete activity. A period of 6 months should elapse after infarction before surgery is considered.
3. Marked enlargement of the left ventricle. The enlarged left ventricle may represent dilatation due to failure, hypertrophy due to hypertension, or a combination of both, with marked scar replacement. Such ventricles are beyond help; so much damage has been done to the myocardial fibers that they are either

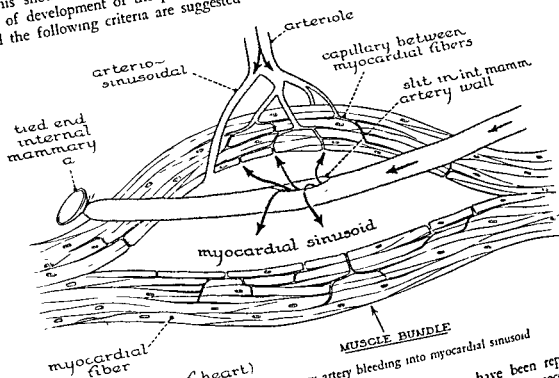


Fig 269.—Implanted internal mammary artery bleeding into myocardial sinusoid

INDICATIONS FOR SURGERY.—

1. There must be proved coronary artery heart disease with typical angina pectoris. Patients who have angina at rest are acceptable for surgery, provided there is an exciting cause.
2. Failure of angina to improve on medical treatment over a period of 1-2 years.
3. Two coronary occlusions with myocardial infarctions. Patients who have had two or more coronary artery occlusions with infarction, even though asymptomatic, have such a poor outlook for survival that some cardiologists are of the opinion that these should be accepted for surgery.

beyond restitution or have been replaced by scar tissue. No revascularization procedure can benefit such a heart any more than a femoral artery graft can revive a gangrenous leg.

4. Rapid progressive deterioration. With this group of patients many surgeons prefer to wait for some degree of stabilization. Not infrequently the accentuation of anginal pain may be a precursor of coronary artery thrombosis and infarction. Such patients may die in the anesthetic room, on the operating table, or shortly thereafter, from an exacerbation of their disease.

5. Malignant hypertension, severe diabetes, and other incurable diseases.

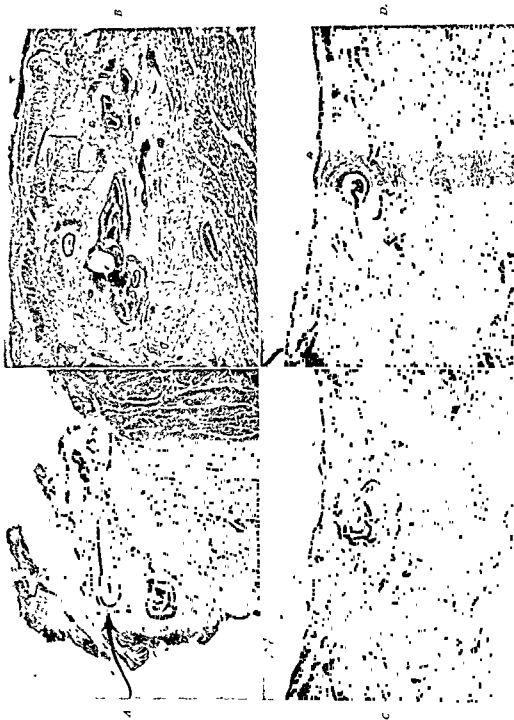


Fig 270.—Photomicrographs of human internal mammary artery 18 months after implantation. Patient was completely relieved of symptoms following operation and died of cancer of the pancreas. Internal mammary artery in myocardial tunnel: A, At beginning of tunnel (flattened due to preservation of specimen). Note patent lumen and absence of intimal proliferation. B, One third of the way along tunnel. Observe extensive branching of artery which has been filled with India ink injected antegrade into left circumflex coronary artery. C, Two thirds of the way along tunnel. Artery still branching. D, Close to exit of tunnel. Artery still patent and branching. Note extensive fibrosis throughout the myocardium.

6 The angina decubitus group, i.e., patients who have angina at rest without exciting cause. Those who experience night pain or pain at rest while watching television or after eating do not necessarily have angina decubitus. Patients who have true angina decubitus should not undergo revascularization surgery because the mortality risk is high, and the end results are poor in those who survive operation.

RESULTS OF TREATMENT.—The recent report of Richards et al. on a 25-year follow-up of cases of myocardial infarction can be used as a guide in the selection of patients for revascularization surgery. To quote these authors: "The best index to long term prognosis following myocardial infarction is the degree of recovery of the patient following the acute period of infarction." In their patients who had complete medical recovery following infarction, 82% were alive at the end of 5 years, whereas the longevity for the entire group of 162 cases was 49% 5 years after myocardial infarction.

Although patients with angina pectoris without infarction appear to have a better future, White states that angina pectoris implies a continuous mortality which is essentially constant and is usually 7% above that expected. The asymptomatic patient therefore has a good prognosis, his myocardium being well supplied with blood by undiseased neighboring arteries, which are known to send collaterals to the ischemic zone so that there is no need for extracardiac sources of blood. Any type of revascularization surgery upon such patients can do no more than Nature has already done.

The treatment of coronary artery disease is probably one of the most difficult to evaluate because of the numerous and unpredictable variations which occur during its natural course. This is true for both medical and surgical therapy. The comparison of individual cases is difficult, but not so with those cases which fit into a certain broad classification. Thus, in evaluating the results of surgery for coronary artery heart disease, there has to be a certain basic classification of cases for comparative purposes.

There are two main groups of patients in whom entirely different results may be expected from revascularization surgery. (1) pa-

tients with angina decubitus, i.e., those who suffer angina at rest without exciting cause, and (2) patients with no angina at rest. Until objective radiologic and other evidence of improved myocardial circulation after surgery is available, the observer must, of necessity, rely upon the following criteria: (1) survival, (2) disappearance of anginal pain for more than 6 months; increase in work and exercise tolerance, (3) improvement in the electrocardiogram, and (4) failure to develop fresh infarction.

Survival.—This pertains to both postoperative and long-term survival. A postoperative death is one that occurs from any cause within 30 days of operation (A.C.S. definition).

Anginal Pain.—Pain is subjective and thus very difficult to estimate both before and after operation. For this reason our patients are seen by two cardiologists and a psychiatrist. Certainly the disappearance of anginal pain a few days or even a few weeks after any surgical revascularization procedure may be on a psychologic basis. Experience indicates that evaluation of an operative procedure should not be made under 6 months. It is again stressed that a careful record be made of the patient's ability to work and exercise without pain before operation. The Master's two-step test and the treadmill give a dependable estimate of the patient's ability to exercise.

Electrocardiogram.—The results obtained by ECG studies leave much to be desired. Too much reliance should not be placed on the ECG in either direction in estimating postoperative improvement.

Failure to Develop Fresh Infarction.—Revascularization surgery does not alter the predetermined course of coronary artery disease except to prevent myocardial fiber loss.

Evidence is now accumulating to indicate that coronary artery occlusion may occur without evidence of myocardial infarction following a satisfactory revascularization. This appears to have been true in at least five postoperative internal mammary artery implantation cases.

There are two operations in use in the treatment of human coronary artery heart disease which have a most extensive experimental background and a long period of clinical trial

These are (1) Beck I operation; (2) internal mammary artery implantation of Vineberg.

The Beck I operation has a mortality rate of 63%, and 80% are markedly improved. Unfortunately there are no published figures indicating the type of patient operated upon, thus making a division into the cases with far-advanced angina decubitus and those without anginal pain at rest impossible at the present time. The over-all low mortality and good postoperative results are most impressive.

Vineberg and Walker have recently reported their results in 88 patients with coronary artery heart disease treated by internal mammary artery implantation. The over-all mortality rate in the combined series for those patients who had no angina at rest was 58% and for those patients who suffered from angina decubitus, 40%. There were 20 patients with angina decubitus in this group of whom 60% survived the operation. Only 35% showed no pain or less pain, and 30% of the completely disabled group returned to work.

In the group of 68 patients who had no angina at rest, 72% were totally disabled prior to operation. The group was followed from 6 months to 6 years, 78% had no pain or less pain, with 79% having returned to work.

THORACIC ANEURYSMS

Definition.—An aneurysm is a bulging due to a local defect of the arterial wall. If the arterial weakness involves the whole circumference, the aneurysm is fusiform, if only part of the circumference is involved, it is saccular.

Etiology.—The causes are (1) arteriosclerosis, (2) syphilis, (3) rheumatic fever, (4) poststenotic dilatation, (5) trauma, (6) mycotic infection, (7) dissecting process, (8) congenital defect.

Aneurysms may appear anywhere in the aorta or great vessels, from the origin of the coronary arteries onward. Most aneurysms of the ascending aorta are syphilitic. Aneurysms may also be congenital, and these are occasionally multiple. In coarctation of the aorta there may be poststenotic dilatation, which sometimes becomes aneurysmal. In addition there may be smaller aneurysms of the inter-

costal vessels due to the high flow through them in this disease. Aneurysms may follow trauma, such as is seen when a penetrating foreign body may graze and so weaken a portion of the arterial wall. Violent nonpenetrating trauma may produce a tear in the vessel wall which will later become aneurysmal. Most thoracic aneurysms, however, result from arteriosclerosis. Whatever the cause, aneurysms dilate relentlessly until they rupture. (See also Chapter 33.)

Clinical Picture.—An enlarging intrathoracic mass may produce symptoms from pressure. Pressure on the tracheobronchial tree may cause atelectasis and a tracheal tug, that on the esophagus may produce dysphagia, and that on the adjacent nerves, a Horner's syndrome and vocal cord paralysis. The aneurysm may erode bone and produce severe pain. Thrombosis and vessel distortion may cause differences in peripheral pulses.

X-ray and fluoroscopy suggest the diagnosis by revealing a mass in relation to the aorta which may pulsate. In certain cases bronchoscopy may show tracheal or bronchial compression. Visualization of the aneurysm is possible by retrograde aortography or angiocardioradiography.

Treatment.—The treatment of aneurysms of the thoracic aorta is surgical excision. Two types of procedures may be employed, depending on the type of aneurysm. Saccular aneurysms may be excised distal to a clamp placed across the neck, the aorta being repaired without encroachment on its lumen. More extensive aneurysms require resection of a segment of aorta and replacement by aortic homograft or plastic prosthesis. The latter procedure requires temporary interruption of aortic flow. The absence of collateral channels prohibits prolonged aortic occlusion because of the danger of damage to brain and spinal cord. Ischemic damage to the kidneys occurs but is not as significant as the cord damage. In either case the damage increases in proportion to the length of aorta that must be excluded by clamps.

To avoid these complications, a temporary path for blood flow must be created with vessel grafts or plastic tubes, or metabolism of the vital centers may be reduced by hypo-

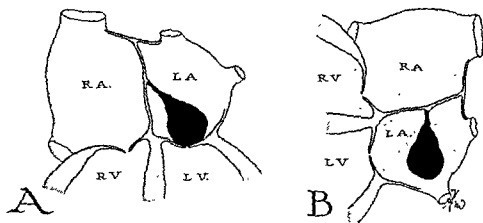


Fig 271 —Heart tumor, left atrium

A, Patient in upright position has symptoms of mitral stenosis, caused by tumor blocking mitral valve

B, Patient in supine position is, paradoxically, relieved of dyspnea as tumor falls away from mitral valve

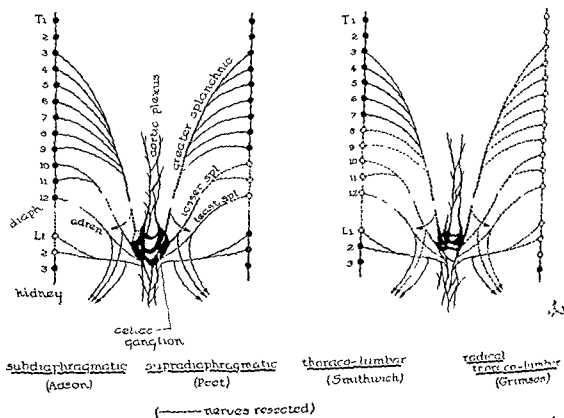


Fig 272 —Composite diagram showing four types of sympathectomy used in the treatment of essential hypertension

thermia. Extracorporeal circulation is necessary for resection of the ascending aorta.

Dissecting Aneurysm

This condition results from the passage of blood through a tear in the intima and media of the aorta. Usually it arises in the ascending aorta and is due to arteriosclerosis. The blood pressure forces the blood between the layers of the aortic wall, so that the dissection may proceed down to the bifurcation. The intramural passage may rupture outward and exsanguinate the patient, or it may rupture back into the aortic lumen, a condition compatible with life but unfortunately less common than the first alternative.

Treatment.—However, this communication between the dissecting aneurysm and the aortic lumen may be produced surgically. All layers of the aorta involved by the dissecting process are divided between clamps. The inner and outer walls of the proximal segment are sutured together for part of the circumference and a wedge portion removed from the inner tube. The inner and outer walls of the distal portion are sutured together and then anastomosed to the proximal segment (See Plates 52 and 53 and Chapter 33).

TUMORS OF THE HEART

Pathology.—Primary tumors are rare, atrial myxoma being the commonest. Fibroma of the ventricular wall and sarcomas filling the cardiac chambers are less common. Rhabdomyoma may be multiple and occasionally is associated with tuberous sclerosis.

Secondary tumors are not infrequent, occurring in some 10% of malignancy cases at post mortem, although they are symptomatic in less than 1%. Leukemic infiltration does occur but is uncommon.

Clinical Picture.—The symptomatology is bizarre, and the diagnosis is rarely made preoperatively. As many of the tumors arise in the atrium and are pedunculated, atypical signs of mitral or tricuspid stenosis may be seen. These signs may alter on change of body posture. Most laboratory studies afford little help, although angiocardigraphy may show a filling defect.

Treatment.—Although a few atrial myxomas have been enucleated by closed techniques, it seems preferable to do an open cardiomy.

ESSENTIAL HYPERTENSION

The surgical treatment of essential hypertension by sympathectomy has greatly lessened in significance in the last few years. The various procedures used were the subdiaphragmatic, supradiaphragmatic, thoracolumbar, and radical thoracolumbar sympathectomies which are shown in Fig. 272. Bilateral adrenalectomy has also been performed but is such a crippling operation and the results so uncertain that it again is seldom used. This decline in surgical intervention has resulted from advances in the medical management and the further development of medication to control the hypertension.

Certain special conditions such as coarctation of the aorta, pheochromocytoma of the adrenal and unilateral renal disease may be found on the investigation of the hypertension. In such cases, surgery is specifically indicated to obviate the underlying causation.

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Film References

- Title
- Wounds of the Heart (Illustrates a composite picture showing the surgical treatment of wounds of the heart and the physiology of tamponade) (1951) (By R. Arnold Griswold, M.D., Louisville)
- Resuscitation for Cardiac Arrest (1956) (By Claude S. Beck, M.D., Cleveland)

Running Time	Sound or Silent	Procured From
21 min	Silent Color	Department of Visual Education Louisville General Hospital 323 E. Chestnut St. Louisville 2, Ky
20 min	Sound Color	E. R. Squibb & Sons 715 Fifth Ave. New York 22, N. Y.

Film References—Cont'd

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Surgical Closure of Atrial Septal Defect Under Hypothermia (Demonstrates method of open cardiectomy under general body hypothermia for repair of atrial defect in a 10-year-old girl with temporary caval inflow occlusion) (1956)	11 min	Sound Color	Denton A Cooley, M.D. Baylor University College of Medicine, Houston 5, Texas
Tetralogy of Fallot (Describes condition, embryology, and developmental pathology of lesion, intimate details of surgery of Blalock-Potts-Smith operations and final results) (1956) (By John C. Jones, M.D., and Bertrand W Meyer, M.D., Los Angeles)	33 min	Sound Color	E. R. Squibb & Sons 745 Fifth Ave., New York 22, N. Y.
Action of the Human Heart Valves (Action of human heart valves is stimulated in post-mortem heart valves as well as surgical correction of latter) (1956) (By Karl P Klassen, M.D., and Charles V Meckstroth, M.D., Columbus)	20 min	Sound Color	American College of Surgeons Motion Picture Library 40 E. Erie St., Chicago 11, Ill
Methods of Surgical Treatment of Pulmonic Stenosis (Including Hypothermia and Extracorporeal Circulation) (Demonstrates the accepted methods, including both closed and	17 min	Sound Color	Denton A Cooley, M.D., Baylor University College of Medicine, Houston 5, Texas

Chapter 17

Esophagus

Darrell D. Munro, MD, Edward J. Tabah, MD, and
H. Fred Moseley, DM

INTRODUCTION

Many factors make surgery of the esophagus technically difficult. Some of these are related to anatomic peculiarities. Others have to do with the ordinary problems of intrathoracic surgery, such as control of the open thorax and the maintenance of lung expansion sufficient to ensure adequate oxygenation of the blood. Because of great advances in the field of anesthesiology, the thorax may now be opened with as little risk as the abdomen. However, it was as recently as 1938 that Phemister and Adams were the first to perform a successful one-stage resection of the esophagus and the cardiac portion of the stomach.

APPLIED ANATOMY AND PHYSIOLOGY

The esophagus is a muscular canal which extends from the pharynx to the stomach. Its wall is composed of three layers: an inner mucous membrane, a submucous layer, and an outer muscular layer. Unlike other parts of the gastrointestinal canal, the esophagus has no serosal covering, and this fact has been mainly responsible for the failure of early attempts at resection and esophageal anastomosis. The mucous membrane is of the squamous cell type except for the most distal portion where the cells become columnar in character.

The circular and longitudinal muscle layers of the cervical and upper thoracic portions are composed entirely of voluntary muscle fibers. Below this level there is an increasing proportion of smooth muscle with a corresponding decrease of striated muscle, and, in the lower third, the musculature is entirely involuntary in type.

The main function of the esophagus is to convey fluid and food from the pharynx to the stomach. Food is propelled down the esophagus by peristaltic waves, but fluids descend mainly by gravity. Before food or fluid can enter the stomach, the very terminal portion of the esophagus must relax. Whether a true sphincteric mechanism exists here or not is still controversial. This area is designated the *cardiac sphincter*. The term *cardia* was originally introduced by Galen, who realized that disease in this area could simulate heart disease very closely. The mechanism that opens the sphincter is obscure, but it is dependent on the nerve supply of the esophagus which is derived from the vagi and the sympathetic trunks.

Normally, the esophageal lumen is narrowed at three points: (1) at the level of the cricoid cartilage, (2) where it is crossed by the left bronchus, and (3) where it pierces the diaphragm.

The esophagus is divided into cervical, thoracic, and abdominal portions. The cervical portion begins at the lower border of the cricoid cartilage, opposite the 6th cervical vertebra. Anteriorly, lie the trachea and the thyroid gland, while posteriorly are the vertebral column and the longus colli muscles. The common carotid arteries and part of the lateral thyroid lobes lie on either side of it. Between it and the trachea, the right and left recurrent nerves ascend. The thoracic duct lies to its left side.

portions, and a lower thoracic segment from the root of the lungs to the diaphragm. After entering the thorax, the esophagus rests upon the vertebral column, the longus colli muscles, the right intercostal arteries, the thoracic duct, and the hemiazygos and accessory hemiazygos veins. In front are the trachea, the left main bronchus, pericardium, and diaphragm. On the left side, in the supra-aortic part, lie the left subclavian artery, thoracic duct, and the left pleura, with the left recurrent laryngeal nerve between the trachea and the

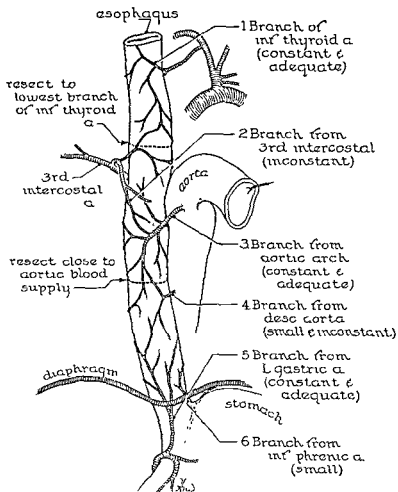


Fig. 273—Segmental arterial blood supply of the esophagus

The thoracic portion begins at the thoracic inlet and ends where the esophagus penetrates the diaphragm. For surgical reasons, it is divided into two segments—a mid-thoracic segment, comprising supra-aortic and infra-aortic

esophagus. In the infra-aortic portion, the thoracic duct lies posteriorly, with the descending aorta and pleura on its left. On the right side are the pleura and the azygos vein, the latter being partly covered by the esophagus.

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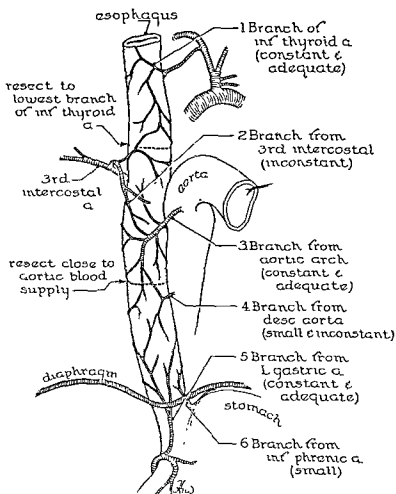


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esophagus. In the infra-aortic portion, the thoracic duct lies posteriorly, with the descending aorta and pleura on its left. On the right side are the pleura and the azygos vein, the latter being partly covered by the esophagus.

The vagus nerves descend in contact with the esophagus below the roots of the lungs, the right lying behind and the left in front.

The relationship of the thoracic duct to this structure is of great importance since the structure may be easily damaged during esophageal manipulation. In the infra-aortic segment, the thoracic duct lies posterior to and on the right of the esophagus. As it ascends it passes slightly to the left, lying closer to the esophagus. In the supra-aortic segment, the duct continues forward and, about midway in the superior mediastinum, ascends anteriorly and to the left until it enters the neck and terminates in the left subclavian vein.

The abdominal portion of the esophagus lies in the esophageal groove on the posterior surface of the left lobe of the liver. It is covered by peritoneum in front and on its left side.

Blood Supply.—The blood supply of the esophagus, which is much richer than was previously supposed, is segmental in distribution. The vessels, for the most part, run in a longitudinal direction. From above downward, the most important individual arteries are the following:

- 1 Inferior thyroid, supplying the cervical and supra-aortic segments
- 2 Branches from the inferior surface of the aortic arch
- 3 Branches of the bronchial arteries
- 4 Branches of the thoracic portion of the descending aorta
- 5 Inferior phrenic artery
- 6 Left gastric artery
- 7 Superior suprarenal arteries

The most critical level for resection is in the upper thoracic portion in the region of the tracheal bifurcation and arch of the aorta where the main blood supply is from the bronchial arteries and where the other collateral channels are minimal. Here, resection with anastomosis is precarious if the bronchial arteries are sacrificed, and therefore it should be performed at a higher level, where descending branches from the inferior thyroid arteries will assure blood supply adequate to prevent necrosis at the suture line.

The veins are arranged in two main groups. The external or periesophageal group follows

the course of their associated arteries. The internal group forms 10-15 parallel trunks which run longitudinally and are evenly distributed throughout the submucosa. The venous drainage of the upper esophagus is into the inferior thyroid and other systemic veins at this level, that of the lower two-thirds of the thoracic esophagus is into the azygos and hemiazygos veins, while that of the abdominal portion is downward into the left gastric (coronary) vein which belongs to the portal system. Free communication between the systemic and portal venous systems is thus established. In portal hypertension there is a rerouting of blood upward through the longitudinal submucous venous plexus into the systemic system. As a result, the submucous veins become abnormally distended and dilated to form *esophageal varices*, often the source of massive gastrointestinal hemorrhage.

Lymphatics.—The esophagus has a rich network of intramural lymphatic vessels arranged in two main groups which drain the mucosa and submucosa and the muscular wall, respectively, with free anastomosis between them. The intramural lymphatics drain to the external surface and from there into three main groups of lymph nodes.

1 *Cervical portion.* Anterior superior mediastinal, the lower deep jugular, and the inferior scalene lymph nodes.

2 *Upper thoracic portion.* Anterior mediastinal and retrotracheal nodes.

3 *Lower esophagus.* Nodes of the lesser curvature of the stomach, particularly those along the left gastric artery.

The intramural lymphatics and the collecting vessels may ascend or descend to empty into distant nodes or may drain directly into adjacent nodes.

In esophageal cancer spread may occur directly into the closest lymph node or to those situated at a considerable distance above or below the lesion. Furthermore, the intramural lymphatic vessels may spread neoplastic cells longitudinally, 8-10 cm. above or below the visible and palpable limits of the primary cancer.

Nerve Supply.—The upper portion of the esophagus is supplied with motor fibers by the

recurrent laryngeal nerves which lie adjacent to it. Below this level, the vagal trunks are motor to the remaining musculature by way of the esophageal plexus, which also derives fibers from the thoracic sympathetic and splanchnic nerves. The sympathetic and parasympathetic fibers form a myenteric and submucous plexus within the wall. From experimental studies it has been shown that there is reciprocal innervation between the lower part of the thoracic esophagus and the terminal abdominal portion. The vagi are responsible for increased tone and motility of the

e.g., acute retropharyngeal abscess; and in the larynx, e.g., acute laryngitis, may make swallowing almost impossible. Neoplastic lesions of the pharynx and extrinsic larynx may exhibit dysphagia as the first symptom.

Neurologic disorders affecting the musculature of the pharynx constitute another group of causes of dysphagia. Among such disorders are included bulbar palsy, myasthenia gravis, postdiphtheritic paralysis, and functional disturbances as found in hysteric patients. In infants the mechanical defects of harelip and cleft palate should also be remembered.

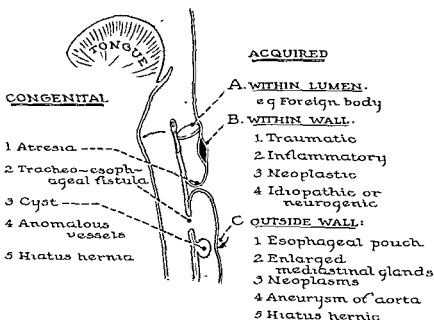


Fig. 274—Classification of lesions causing dysphagia

esophagus and for producing relaxation of the cardiac sphincter, while the sympathetic system has the opposite action.

SYMPTOMS OF ESOPHAGEAL DISEASE

Dysphagia may be defined as the difficulty in swallowing either fluids or solid food. There are many inflammatory and neoplastic lesions of the mouth, pharynx, larynx, and esophagus which are manifested by this symptom. The student should remember that acute pain resulting from inflammatory lesions in the mouth, e.g., acute parenchymatous glossitis, acute stomatitis, and quinsy; in the pharynx,

Esophageal lesions causing dysphagia may be classified as follows.

- A. Congenital causes** atresias, tracheo esophageal fistulas, cysts, anomalous vessels, and hiatus hernia
- B. Acquired causes**
- 1 Within the lumen: e.g., foreign bodies such as coins, tooth plates, etc.
 - 2 Within the wall
 - a Traumatic, e.g., strictures caused by swallowing corrosives
 - b Inflammatory, e.g., peptic ulcer or esophagitis at cardiac end
 - c Neoplastic, e.g., carcinoma
 - d Idiopathic or neurogenic, e.g., cardiospasm

- 3 Outside the wall
 - a Esophageal pouches of pulsion or traction types which fill with food and compress the lumen from without.
 - b Enlarged mediastinal glands of inflammatory or neoplastic origin
 - c Neoplasms infiltrating or compressing from without
 - d Aneurysms of the aorta
 - e Hiatus hernia

Pain may be present only on swallowing or the patient may complain of attacks of substernal distress, either sharp and severe or dull and aching in character.

Regurgitation of undigested food occurs fairly commonly, particularly in the presence of a pharyngoesophageal diverticulum or of cardiospasm.

Hemorrhage into the esophageal lumen may produce blood-tinged vomitus and is caused by ulceration, foreign body erosion, or bleeding varices (see Chapter 19).

EMBRYOLOGY AND CONGENITAL ANOMALIES

(See Chapter 30)

FOREIGN BODIES

Infants and children are most prone to the hazard of foreign bodies in the esophagus, but such are not infrequently seen in adults.

The majority of such accidents is the result of carelessness. In adults, this complication results from the failure to detect bones or other hard substances in swallowed foods, from poorly masticated meat, or from the loss of poorly fitting or damaged dentures. Psychotic patients may intentionally swallow foreign bodies. In children, coins, nails, tacks, screws, safety pins, buttons, meat, and fish bones are the most common foreign bodies concerned.

Such an object may lodge almost anywhere in the esophagus but is usually arrested at the level of the cricoid cartilage. If it passes this point it will usually proceed to the cardiac sphincter or into the stomach. In some cases it may lodge at the other sites of natural narrowing, i.e., at the level of the aortic arch and of the left main-stem bronchus.

The presence of esophageal disease may block the passage of substances which normally descend without difficulty.

Symptoms.—The symptoms vary from vague discomfort or a sticking sensation to severe pain and dysphagia. In an adult it is generally possible to obtain a history of a swallowed foreign body, but in a child this information may be completely lacking. Pain is usually localized, by the patient, on the body surface corresponding to the level of lodgment in the neck or chest. Dysphagia may

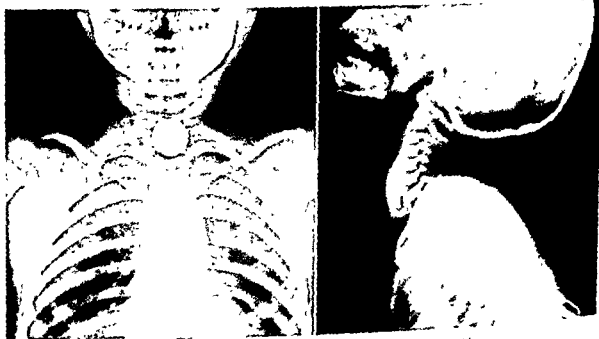


Fig. 273.—Foreign body (coin) impacted at level of cricoid.

result, and, if severe, is accompanied by an accumulation of saliva in the hypopharynx which finally overflows into the trachea, producing choking and coughing. Eventually, if the foreign body remains in situ, perforation may occur, producing acute mediastinitis, mediastinal abscess, or an erosion of a major blood vessel with fatal hemorrhage. In other cases a fistula may be produced between the trachea or bronchus and the esophagus and, in long-standing cases, stricture formation follows.

Diagnosis.—This is made from the history and symptoms. Examination of the hypopharynx may, on rare occasions, reveal the presence of the foreign body. Careful x-ray studies are useful, especially when the foreign bodies are radiopaque, or, if radiolucent, a barium swallow will outline the object or reveal the presence of obstruction. If perforation is suspected, Lipiodol instead of barium should be used.

Treatment.—The primary treatment is endoscopic examination and extraction. Operative intervention is necessary when endoscopic removal has failed and when complications such as perforation with an associated mediastinitis or abscess formation, stricture, or hemorrhage has occurred.

STRICTURE

Benign strictures may occur from a variety of causes. The majority result from the accidental swallowing of caustic chemicals, which produce acute inflammation and secondary scarring. The ingestion of such material occurs mainly in children and is commonly caustic soda or lye (see Chapter 30). The patient may complain of substernal burning, epigastric pain, excessive salivation, and vomiting but is usually able to swallow normally for a few days after the accident. However, after 1-2 weeks there is slight difficulty in swallowing solids, which becomes progressively more severe until the patient is finally unable to take fluids. Prolonged dysphagia results in progressive weight loss, weakness, anemia, vitamin deficiency, and general malnutrition. Very severe burns may produce a secondary mediastinitis with its accompanying symptoms.

Benign strictures may also occur following the impaction of a foreign body, gunshot wound of the esophagus, benign tumors, peptic

esophagitis, or following operations for tracheo-esophageal fistula or esophageal diverticulum.

Treatment.—A person who is suspected of having swallowed a caustic agent should be hospitalized as soon as possible and an attempt made to establish the nature of the material swallowed. Immediate neutralizing substances should be used, such as vinegar after alkali, bicarbonate of soda after acids, and milk or egg white after phenol. Examination of the oral cavity, the pharynx, and larynx may reveal the presence of a burn. In such cases laryngeal edema may ensue and a tracheostomy be required if there is the slightest suggestion of resulting respiratory difficulty.

If the patient is able to take nourishment by mouth, a soft liquid diet is given. If, however, swallowing becomes impossible, a gastrostomy must be performed for feeding purposes. Esophageal dilations, using olive-tipped dilators or mercury-filled bougies specifically designed for this purpose, are commenced as soon as possible in order to re-establish and maintain adequate passage.

A chronic or well-established stricture requires careful roentgenographic and esophagoscopy studies to determine its exact site and extent. In these cases, following preliminary gastrostomy, dilations are performed, using flexible-tipped dilators or olive-tipped bougies passed over a previously swallowed thread.

In other cases of well-established tight strictures, heavy silk thread is swallowed and brought out through the gastrostomy. One can thus carry out rapid and frequent dilations with bougies of increasing size either in a retrograde or peroral manner, using the silk thread as a guide.

Strictures that are caused by benign tumors, foreign bodies, and peptic esophagitis are discussed more fully in the section dealing with these conditions. Localized strictures, such as those following diverticulectomy, gunshot wounds, or stabbing, may be amenable to resection and end-to-end anastomosis. In more extensive strictures, more radical operative procedures may be necessary, such as excision with esophagogastrostomy or esophagojejunostomy. In selected cases an isolated segment of upper jejunum may be anastomosed between the resected esophagus and the stomach.

3 Outside the wall

- a Esophageal pouches of pulsion or traction types which fill with food and compress the lumen from without.
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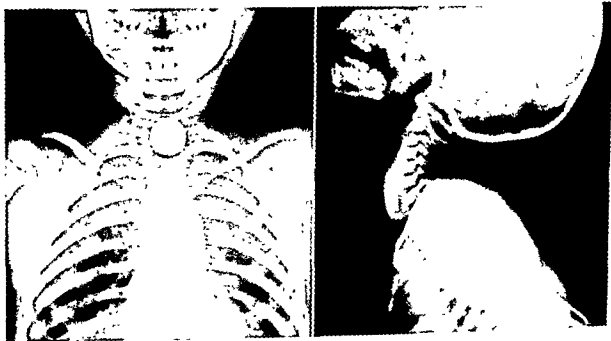


Fig. 275.—Foreign body (coin) impacted at level of cricoid

result, and, if severe, is accompanied by an accumulation of saliva in the hypopharynx which finally overflows into the trachea, producing choking and coughing. Eventually, if the foreign body remains in situ, perforation may occur, producing acute mediastinitis, mediastinal abscess, or an erosion of a major blood vessel with fatal hemorrhage. In other cases a fistula may be produced between the trachea or bronchus and the esophagus and, in long standing cases, stricture formation follows.

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ESOPHAGEAL DIVERTICULA

Diverticula are classified according to their anatomic position and their method or origin. The following types of diverticula are recognized:

- 1 Pharyngoesophageal (pulsion type)
- 2 Epibronchial (traction type)
- 3 Epiphrenic (pulsion type) (see Plate 15)

Pharyngoesophageal Diverticulum

This is a pulsion diverticulum that occurs characteristically on the posterior wall at the pharyngoesophageal junction. At this point, the inferior constrictor or cricopharyngeus muscle is divided into two muscle bundles, i.e., an upper oblique and a lower transverse. There is usually a slight gap, a weak area, frequently called the pharyngeal dimple, between these two sets of muscle fibers. During the act of swallowing, food is projected from the back

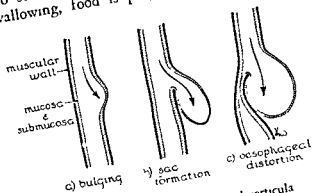


Fig 276—Stages of esophageal diverticula

of the tongue and impinges on this weak part of the pharyngeal wall. Furthermore, a peristaltic wave, beginning in the nasopharynx and passing downward to the pharyngoesophageal junction, is initiated by the pharyngeal constrictor muscles. The upper oblique fibers of the inferior constrictor are propulsive, while the lower transverse fibers are said to be sphincteric in action. If the coordination involving the relaxation of the propulsive muscle fails to occur while the peristaltic wave, which is active during this initial peristaltic wave, undue pressure will be exerted by the bolus of food at the pharyngeal dimple. A small bulge develops at this point, and the mucous membrane is eventually herniated as a pouch between the two parts of the inferior con-

strictor muscle. The ultimate development of the diverticulum is merely a matter of repetition of this mechanism. A true sac, consisting of mucosa and submucosa, is formed and eventually is of sufficient size to retain food and saliva and give rise to symptoms.

Symptoms.—The symptoms depend upon the stage of sac development and are directly referable to the act of swallowing. They may be absent or range from minimal discomfort to progressively increasing dysphagia and regurgitation. Regurgitation of previously swallowed materials into the pharynx is probably the most frequent complaint and occurs in the majority of patients with well-established pharyngoesophageal diverticula. Another frequent complaint is the occurrence of gurgling noises during deglutition of liquids. This symptom may become so noticeable as to embarrass the patient. At other times, patients complain of obstruction, which is usually described as a sticking or a delay of food passage in the throat, lasting only a few minutes and relieved by taking fluid or by pressure against the side of the neck. In the very large diverticulum, the sac extends into the superior mediastinum and distorts the esophageal wall so that the normal esophageal opening is now directly into the sac. Here, complete esophageal obstruction occurs associated with progressive weakness, malnutrition, and weight loss.

Diagnosis.—In addition to a suggestive history, x-rays with barium swallow will demonstrate a sac which communicates with the esophagus. Esophagoscopy, which may be employed for confirmatory purposes, carries with it the danger of perforation through the diverticulum.

Treatment.—When symptoms are sufficiently severe, surgical resection of the sac must be performed. The incision employed is usually oblique, following the anterior border of the left sternomastoid muscle. A collar incision may also be used. Formerly the two-stage operation was widely practiced to minimize the development of acute mediastinitis. In the first stage the diverticulum was mobilized and the fundus suspended to the adjacent tissues at a point above its origin. Ten to 14 days later, after fibrosis had sealed off this area, the

sac was excised and the defect in the esophageal wall closed.

Today chemotherapy diminishes the risk of mediastinal infection, and the one-stage resection is practiced.

Complications.—Serious complications may occur following such operations.

1. Injury to the recurrent laryngeal nerve may occur. This nerve must be carefully identified and protected during the operation.

2. Injury to the thoracic duct is possible. This complication is rare. Severe, if unrecognized, will result in a chylous fistula with its serious sequelae.

3. The occurrence of a fistula at the site of resection is usually temporary and closes spontaneously with conservative management. However, large or persistent fistulas may require secondary surgery for closure. Rarely, the fistula is caused by an unrecognized obstructing esophageal web below the site of the diverticulum. After excision of the pouch, routine exploration of the esophageal lumen is necessary. A web, if present, should be excised.

4. Stricture formation may result from inadvertent inclusion of a portion of the esophageal wall with the sac. Strictures may also occur following healing of a fistula and may require subsequent dilatations or secondary operation.

5. Local infection with mediastinitis and abscess formation is sometimes associated with large diverticula with peridiverticulitis. In such cases it may be preferable to employ the two-stage method of removing the diverticulum.

Epibronchial Diverticulum

This is a traction diverticulum and is commonest in the mid-portion of the esophagus at the level of the main bronchi. Inflammatory processes in adjacent lymph nodes produce adhesions to the esophagus which gradually contract. As a result, the wall of the esophagus is pulled out in tentlike fashion.

Since their walls contain all layers of the esophagus, the mucosa, submucosa, and muscularis, they are true diverticula. They rarely exceed 2 cm in diameter and have a broad neck and a contracting muscular wall which prevents retention of food.

These lesions are common, occurring in approximately 35% of all autopsies. The majority are asymptomatic and without clinical significance. Occasionally patients may complain of mild substernal burning and pain.

Traction diverticula are easily demonstrated by barium swallow.

Treatment.—Treatment is unnecessary in most cases, but if severe symptoms are present, excision employing a transthoracic approach is indicated.

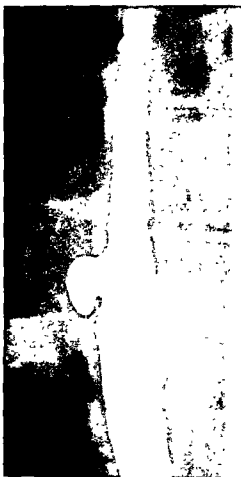


Fig 277.—Traction diverticulum at typical site, bifurcation of trachea.

Epiphrenic Diverticulum

These pulsion type diverticula occur most frequently at a point in the esophagus several inches above the diaphragm. Corresponding to the pharyngoesophageal diverticula, those which develop in this region consist of a pouch of

mucosa appearing between the fibers of an attenuated muscular layer.

Etiology.—The etiology of epiphrenic diverticula is obscure. Some believe they are congenital in origin, while others feel that they are produced as a result of distal esophageal obstruction with herniation of the mucosa through a weakened muscular area.

Symptoms.—In the majority of patients, the symptoms at first are vague and indefinite. In some, pain and burning referred to the substernal area and the lower back may be present. Varying degrees of dysphagia may result from the distended diverticulum and the pressure and distortion it produces. In the rare complicated case, complete mechanical obstruction may result with subsequent weight loss, malnutrition, and weakness. Diagnosis is established during barium swallow.



Fig 278—Pulsion diverticulum in lower esophagus

Treatment.—Mild symptoms can be relieved by conservative therapy, prescribing soft bland foods, a full glass of water after each meal to wash out the sac, and antispasmodics. Severe persistent symptoms necessitate resection of the sac with repair of the defect in the esophageal wall. A transthoracic approach from the side to which the diverticulum presents is employed.

RUPTURE OF THE ESOPHAGUS

Rupture of the esophagus may occur as a result of trauma, inflammation, or eroding neoplasm. It may also occur spontaneously in a perfectly normal esophagus.

Traumatic Perforation

Most traumatic perforations result from endoscopic procedures or follow the impaction of foreign bodies. Osteoarthritic spurs in the cervical vertebrae may bruise and perforate the esophageal wall during endoscopy. Perforation may also follow procedures such as biopsy or instrumental dilatation. The esophagus, being a deeply seated organ in the cervical and thoracic regions, is rarely involved by external violence.

Signs and Symptoms.—Those resulting from rupture of the esophagus are not always immediate and depend on the size and location of the perforation. Perforation should always

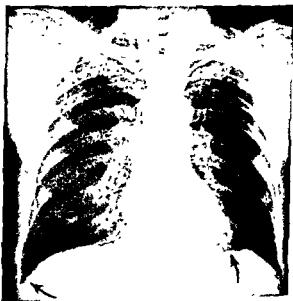


Fig 279—Traumatic perforation, lower esophagus, 3 hours following esophagoscopy. Note free air beneath diaphragm.

be suspected when pain or discomfort follows endoscopy or the ingestion of a foreign body or caustic material.

In perforation of the cervical esophagus, local tenderness beneath the sternomastoid muscle and crepitation of the soft tissues of the neck are early signs. Dysphagia, hoarseness, pain, and tracheal stridor usually appear 3-5 days later. Usually present are a rising temperature, increased pulse and respiratory rates, a sore throat, and leukocytosis. X-rays of the neck and superior mediastinum may reveal some widening of the prevertebral soft tissue shadow or a retropharyngeal gas pocket with fluid

level. Extension downward to the superior mediastinum will result in a superior mediastinal abscess clearly visualized on x-ray. The fascial planes of the neck are responsible for the route of spread for infection produced by a perforation (see Chapter 12).

Perforations of the thoracic esophagus are generally associated with mediastinitis, pleuritis, pleural effusion, or peritonitis. Perforations into the pleural cavity are followed rapidly by accumulation of fluid and air in the pleural space. A tension pneumothorax may develop accompanied by severe dyspnea and cyanosis. Physical findings may be minimal, and a chest x-ray may reveal mediastinal emphysema, mediastinitis, free air in the pleural space or beneath the diaphragm, or a frank pleuritis and hydropneumothorax.

Rupture of the lower esophagus is usually associated with severe substernal and epigastric pain, and the signs and symptoms of peritonitis are present. Perforations immediately above or below the hiatus produce this picture.

Treatment.—Successful treatment of this condition depends largely upon early diagnosis and active operative treatment. Antibiotic therapy combined with suitable repair and drainage procedures is mandatory. Tracheostomy may be required in some cases.

Conservative management of perforations involving the cervical esophagus is generally successful, especially in patients having small perforations involving the mucous membrane only. Intensive antibiotic therapy combined with bed rest, nasogastric suction, and no oral feedings will control the spread of infection and result in complete recovery in a large percentage of cases. In more complete perforations where the infection has had a chance to become well established, an acute retropharyngeal abscess may result in extension downward into the superior mediastinum. Such cases urgently require cervical mediastinotomy for drainage and decompression of the tense abscess.

If the abscess is confined to the neck it may be approached by an incision behind the sternomastoid muscle. An emergency tracheostomy may be necessary because of acute respiratory difficulty associated with edema and swelling about the larynx. The usual supportive measures are also prescribed.

Patients with lacerations of the thoracic esophagus rarely survive with conservative management, particularly where the perforation has extended into the pleural cavity. Once the pleural space is contaminated, immediate open operation is the only means of preventing almost certain death. A thoracotomy, with repair of the torn esophagus and suitable drainage of the pleural cavity, must be performed.

Spontaneous Rupture

Spontaneous rupture of the esophagus is an extremely rare condition which occurs four times more frequently in males than in females. In certain cases the cause is some pre-existing weakness of the esophageal wall, which gives way when intraesophageal pressure is suddenly increased. This usually occurs in the lower third on the left posterolateral wall, and is followed by rupture into the left pleural space. It may, however, occur through the left anterolateral wall. The mortality rate which in the past was 100% has been reduced in recent years to approximately 30% by early diagnosis and prompt surgical intervention.

Diagnosis.—The patient is commonly a previously healthy male, robust, a heavy eater, and possibly an alcoholic, who complains of excruciating pain immediately following a bout of vomiting, which may have been either spontaneous or induced. The pain is epigastric, radiating to the left side of the chest and to the back. Dyspnea, cyanosis, and shock develop rapidly. Hematemesis is rarely copious. Vomiting generally ceases after the onset of pain and is followed by intense thirst. Some patients present a history of previous ulcer, esophagitis, or esophageal stricture. Physical examination reveals severe shock and a unilateral (usually left) or bilateral hydropneumothorax. Subcutaneous emphysema is present at the base of the neck in 60% of cases. Perforated peptic ulcer, acute pancreatitis, mesenteric thrombosis, dissecting aneurysm of the aorta, coronary thrombosis, pulmonary embolism, and spontaneous pneumothorax must be excluded.

The presence of hydropneumothorax associated with shock and vomiting is almost pa-

thognomonic At an early stage, x-rays reveal mediastinal emphysema, which ascends rapidly into the cervical region. They may also demonstrate fluid levels in the mediastinum and in the pneumothorax. Extravasation of radiopaque oil introduced into the esophagus further confirms the rupture. Aspiration of the pleural cavity yields fluid characteristic of gastric or esophageal contents.



Fig. 280.—Hydropneumothorax caused by spontaneous perforation of lower esophagus

Treatment.—Control of shock, decompression, and drainage of the tension pneumothorax and replacement of oral feedings by parenteral fluids are immediate requirements. Chemotherapy is instituted. Thoracotomy is performed with closure of the esophageal tear. The mediastinum and pleura are cleansed and drained. A nasal catheter is introduced to prevent postoperative distention of the stomach.

ACHALASIA

(Synonyms: Idiopathic Dilatation, Cardiospasm)

Early theories postulated that in this condition proximal esophageal dilatation was produced by failure of the cardiac sphincter to open reflexly during the act of swallowing. The term *achalasia*, meaning lack of relaxation, was then adopted. Absence of hypertrophied muscle fibers in the narrowed portion of the esophagus led others to assume that there had occurred progressive degeneration of Auerbach's

plexus in this region. Whatever the cause, cardiospasm accounts for approximately 20% of all patients complaining of dysphagia. It is commoner in the female than in the male. Although cardiospasm has been recorded in infancy, most cases appearing for treatment are in the third or fourth decade of life. In some cases there seems to be a definite psychogenic factor.

Pathologic Features.—The narrowed segment is a thin, pale area, 3-6 cm. in length just proximal to the stomach; the muscular coats are to some degree replaced by fibrous tissue, and degeneration of myenteric ganglia is evident. Proximal to this the esophageal musculature may be very thick and hypertrophied, and in long-standing cases the wall stretches and become tortuous.

The radiologic deformity is at first a funnel-shaped dilatation which, in the later stages, becomes flask-shaped and ultimately assumes a sigmoid form.

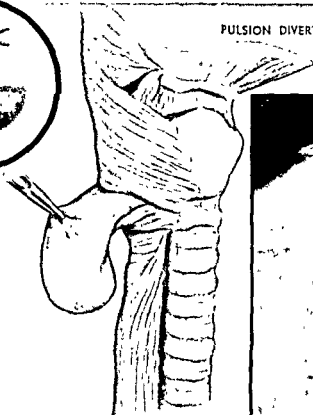
Symptoms.—Patients with cardiospasm can feel the esophagus contract when they are excited. The first swallows of food pass easily, but there soon follows a sensation of fullness behind the lower portion of the sternum. Symptoms of dysphagia are aggravated by rapid eating, roughage, and hot or cold fluids. As the disease progresses, regurgitation may and weight is lost. Nocturnal regurgitation may result in aspiration pneumonia or pulmonary abscess.

Diagnosis.—X-ray examination and esophagoscopy are confirmatory. The latter is particularly helpful in distinguishing between cardiospasm and esophageal carcinoma. In advanced cases, aspiration by catheter will recover putrefying food products.

Treatment.—Treatment is divided into medical and surgical phases. Medically, a bland diet, antispasmodics, and psychotherapy are worthy of trial. If the disease progresses, dilatation by means of a mercury-loaded bougie or a hydrostatic bag will bring relief to some patients. If repeated dilatations are necessary, the patient can often be taught to pass it on himself. There is a definite hazard of accidental perforation when the esophagus is edematous, friable, and tortuous.

A special balloon catheter has been designed which is introduced through the cardiac sphincter.

PULSION DIVERTICULUM



TRACTION DIVERTICULUM

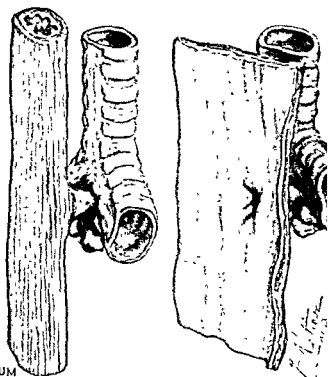


Plate 15.—Types of Esophageal Diverticula.



Fig 281—X-ray appearance in advanced cardiospasm, showing markedly dilated esophagus. Inset shows area of spasm at cardioesophageal junction

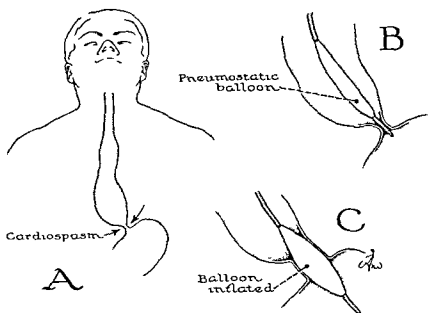


Fig 282—Cardiospasm dilated under x-ray control, using a pneumostatic balloon

ter after a suitable dilatation. The balloon is positioned and is then inflated with air or water under pressure and the narrowed segment is stretched. This method appears to be dependent on the uncontrolled rupture of the muscular coat in the spastic area. Advocates of pneumostatic or hydrostatic dilatations report good results, but the risk of rupture with its very serious sequelae is a reason in favor of operative treatment in most instances.

Some form of surgical treatment must be considered for those patients who fail to obtain relief from dilatation. Many operative procedures have been devised and, in particular, several types of cardioplasty have been tried. They have, however, the distinct disadvantage of destroying the competence of the cardiac sphincter, and reflux esophagitis is a frequent complication.

The Heller operation (1913) appears to give the most consistent and best results in the surgical management of advanced cardiospasm. It is similar in concept to the Fredet-Rammstedt operation for congenital hypertrophic pyloric stenosis. Originally, Heller made two longitudinal incisions, one anterior and one posterior, through the muscular layers of the area of cardiospasm (cardiomyotomy), allowing the submucosa and mucosa to bulge outward. In current practice, only an anterior incision is utilized, and when carefully performed, incompetence of the cardia, allowing esophageal reflux, is absent or very minimal. The operation may be done through the abdomen or thorax, the latter approach being preferable.

REFLUX ESOPHAGITIS

Chronic intermittent regurgitation of digestive juices into the lower esophagus produces a chemical inflammation, designated reflux or peptic esophagitis. In most instances this entity appears to be the result of diffuse inflammatory erosion, ulceration, and scarring produced by prolonged reflux of gastric juice in the presence of an incompetent cardioesophageal sphincter and frequently associated with hiatus hernia. Reflux of alkaline juices into the esophagus, after surgical procedures such as esophagojejunostomy, can also cause an identical type of esophagitis. This entity



Fig. 283—Benign esophageal stricture caused by prolonged nasogastric intubation

is an important cause of chronic dyspepsia, so-called heartburn and dysphagia, and it seems likely that its incidence is much higher than has been previously suspected.

Etiology.—In general, the methods of production of reflux esophagitis and the various factors involved are understood. However, there is no agreement on the factor in the digestive juices specifically responsible. Abundant experimental evidence demonstrates the susceptibility of the esophageal mucosa to acid-peptic digestion. The following fact may be etiologic:

- 1 Incompetence of the cardioesophageal mechanism in the presence of sliding hernia or following operations involving the cardioesophageal junction
- 2 Gastric hypersecretion and peptic ulcer diathesis
- 3 Protracted vomiting, nasogastric intubation, and pyloric obstruction
- 4 Ectopic gastric mucosa occurring in association with a short esophagus
- 5 Individual susceptibility

Reflux esophagitis is most often associated with sliding hiatus hernia where the esophago-gastric junction is displaced and the normal mechanism preventing reflux is absent. Regurgitation of gastric contents may then occur, especially when accentuated by pyloric obstruction or spasm, increased intra-abdominal pressure associated with obesity, or merely the assumption of the reclining position. The association of peptic duodenal ulceration with reflux esophagitis is high (40%) and a true ulcer diathesis with its associated hyperchlorhydric state, nervous tension, and individual susceptibility is generally agreed to be present in most cases. Frequently, the symptoms of esophagitis may be identical to those of duodenal ulcer.

Pathology.—The typical lesion occurs in the lower third of the esophagus, and four stages in its natural history are described, i.e., esophagitis, esophagitis with acute ulceration, esophagitis with chronic ulceration, and healed fibrous stenosis. At first the esophagus retains its natural elasticity and the associated hernia can be reduced when the patient is upright. Chronic ulceration progresses and the inflammatory reaction results in chronic submucous fibrosis around the lower third of the esophagus. Later this fibrotic tissue contracts and produces not only stenosis but also shortening of the esophagus, which draws the stomach up through the hiatus and fixes it in this position. The acid gastric juice continues to act unopposed on the esophageal mucosa, and the condition is continuously aggravated. The histologic appearance usually includes superficial ulceration with granulation tissue, extensive submucous fibrosis with edema and thickening of the wall, and infiltration of the musculature with chronic inflammatory cells.

Symptoms.—The clinical features are dominated by a common form of indigestion consisting mainly of heartburn, gastric flatulence, and postural regurgitation. Three varieties of pain may be noted: (1) high epigastric discomfort associated with high acid secretion, (2) burning retrosternal pain from acid irritation of the inflamed esophagus, and (3) a deep, boring pain made worse by flexing the spine. In adults, the commonest symptom is dysphagia followed by dyspepsia and less commonly by bleeding. Loss of weight is pro-

portional to the degree of obstruction and starvation. Pain may be precipitated by lying down or stooping forward, especially after eating, and relief is obtained by drinking milk or alkaline beverages. With advanced esophageal inflammation, intermittent dysphagia and bouts of partial or complete obstruction occur.

Diagnosis.—This is established by the history and by the radiologic and esophagoscopy examinations. The radiologic demonstration of esophagitis may be difficult. Advanced cases may show stenosis and stricture formation in the lower esophagus with proximal dilatation. In early cases, close inspection may reveal fuzziness of the mucosal pattern with spasm as evidenced by lack of distensibility of the



Fig. 284.—Reflux esophagitis with stricture. Arrows indicate peptic esophageal ulcer, sliding hiatus hernia, and diaphragm.

esophagus and sometimes small areas of ulceration. Frequently it is impossible to locate the cardioesophageal junction since the stomach may be pulled through the hiatus like a sleeve, and there is no clear demarcation between the esophagus and stomach. This knowledge is important for the surgeon. In the cases associated with hiatus hernia, a reflux of the barium into the lower esophagus may be easily seen in the early cases. Sometimes a hiatus hernia may be difficult to demonstrate, and examination in the Trendelenburg position should always be attempted for this reason.

Esophagosopic examination should be carried out in all suspected cases of esophagitis for the following reasons: (1) the degree of esophagitis, ulceration, and stenosis can be evaluated, (2) carcinoma may be ruled out by biopsy, (3) the level of the cardioesophageal junction can be accurately established and verified by the application of a silver clip which may be later demonstrated by x-ray to be above the diaphragm, and (4) information can be gained concerning the amount of fixation of the lower esophagus to the mediastinal structures.

The *endoscopic examination* shows a characteristic hyperemia and edema of the mucosa in the lower third of the esophagus which frequently bleeds easily on contact. Ulcers may be noted and the cardia is lax and patulous, the instrument passing easily into the stomach without deviation.

Treatment.—The treatment of this condition may be medical or surgical. All early cases of esophagitis and those with symptoms of moderate degree should be given a trial of *medical therapy* consisting essentially of an ulcer regime including an antacid, an antispasmodic, a bland diet, and sedatives. Conservative treatment may considerably relieve symptoms. Patients are advised to remain erect after meals, avoid stooping and bending, and to sleep with the head of the bed elevated. *Weight reduction and the removal of constricting belts and tight clothing* may be necessary to decrease intra-abdominal pressure. Many patients will obtain symptomatic relief, but those who fail to respond and those with destruction of the mucosa and progressive fibrotic stenosis may require surgical treatment.

The *surgical treatment* has as its first objective, prevention of reflux by restoring the normal cardioesophageal relationship, i.e., by correcting an existing hiatus hernia. The technique of Allison has been universally favored in this respect. Second, rapid drainage of the esophagus is secured by relieving any obstruction distal to the esophagus, such as pylorospasm. This has resulted in the use of the Finney pyloroplasty for the relief of symptoms associated with this condition, and this appears of benefit in the early phase of the disease. A third objective is the reduction of gastric acidity. For this Wangenstein has proposed subtotal gastrectomy to eliminate the acid-peptone secretions. He claims great success for this operation even in patients with stricture formation.

Obstruction of the esophagus itself must be relieved, and, in addition, other associated gastrointestinal disease must be eliminated. When esophagitis has progressed to a fixed, shortened esophagus with fibrous stricture, dilatation and bouginage may be performed. Extreme stenosis with dysphagia and weight loss require radical surgery. Excision of the stenotic portion with radical proximal gastrectomy is performed. Continuity is re-established by rolling the distal stomach segment into a tube and anastomosing it high to the esophagus. The operation is completed by pyloroplasty, which allows rapid emptying of the stomach remnant. Other operations that utilize isoperistaltic Roux loops of small bowel to by-pass the stomach or to serve as valvular units between the stomach and esophagus are also advocated. These, however, appear to carry a much higher incidence of digestive disturbances, especially those which by-pass the stomach altogether.

TUMORS

Benign tumors are uncommon at any age, whereas malignant neoplasms (carcinoma) are relatively common in adults.

Benign Tumors

Most of this group arise from the non-epithelial tissues of the esophageal wall. Of these, the leiomyoma occurs most commonly. The average age incidence is 40 years, in con-

trast to that for carcinoma which is 60 years. Males are affected twice as often as females.

Symptoms.—Contrary to general belief, these lesions frequently give rise to symptoms, the commonest being pain and dysphagia of varying severity. Bleeding is not a characteristic feature because of the extramucosal origin of the tumor. However, ulceration of the overlying mucosa with hemorrhage may occur.

They are most commonly found in the lowest third and least commonly in the upper third of the esophagus. The tumor is ovoid in shape and averages 6 by 4 cm in size. Malignant changes producing a leiomyosarcoma rarely occur.

Diagnosis.—The diagnosis suggested by the history is established on barium swallow examination and esophagoscopy. A barium-filled esophagus will characteristically show a smooth-filling defect with sharp outlines between the upper and lower limits of the tumor, with a normal esophagus above and below it. An esophagoscopy will generally reveal a firm extramucosal tumor encroaching on the lumen, with an intact, healthy overlying mucosa, although, rarely, ulceration may be present. A biopsy, if sufficiently deep, will establish the histologic nature of the lesion.

Treatment.—The majority of these tumors are removed by simple enucleation through a thoracotomy. A tumor in the lower third is best approached through the left chest, while those in the middle and upper thirds are more easily removed from the right side. Rarely, a tumor may be so large and adherent to the wall that simple enucleation is not feasible and esophageal resection is necessary.

Other less frequently encountered benign tumors include fibromas, lipomas, myxomas, and neurofibromas.

Bronchogenic or esophageal cysts are congenital lesions arising from the primitive foregut. Bronchogenic cysts are generally found near the upper end of the thoracic esophagus, are lined by ciliated columnar epithelium, and usually contain cartilage in the wall. A cyst of esophageal origin situated in the mid-thoracic region may also contain ciliated epithelium, but when located near the diaphragm the lining is usually squamous in type. Cartilage is not present in the wall. The diagnosis and treatment are similar to those for leiomyoma.

Carcinoma

Carcinoma of the esophagus, on this continent, accounts for approximately 4% of all gastrointestinal malignant growths and 2% of all deaths from cancer. Its incidence is proportionately higher in males than females in the ratio of 5:1, reaching its peak in the 6th and 7th decades of life. In Sweden and Finland, where the Plummer-Vinson syndrome occurs commonly in the female, the incidence in the two sexes is practically equal. In China 50% of carcinomas involving the alimentary tract arise in the esophagus.

The location of the primary growth is as follows: upper third (cervical) 20%, mid-third (supra-aortic, infra-aortic) 33%, and lower third (lower thoracic, abdominal) 45%.

Pathology.—Most esophageal cancers are epidermoid or undifferentiated in cell type and arise from the mucous membrane. Adenocarcinoma, when it occurs, is generally found at the lower end of the esophagus. It may represent an upward extension of a primary adenocarcinoma arising in the cardia or may originate in ectopic gastric mucosa or from mucous glands present in the esophageal wall. The gross appearance of the cancer varies from a bulky fungating tumor projecting into the lumen to a growth spreading superficially and infiltrating the mucosa without obstruction to the passage. Submucosal infiltration may occur through the rich intramural lymphatics for some distance beyond the apparent borders of the lesion. This is only recognized on histologic examination.

Direct extension to important structures within the neck, thorax, and abdomen is common. When the primary growth is situated in the upper third, the trachea, recurrent laryngeal nerves, and great vessels may be invaded. When the carcinoma originates in the middle and lower thirds, infiltration of the left bronchus, thoracic duct, aortic arch, descending aorta, pericardium, and diaphragm may be present. Perforation into the mediastinum or pleural cavity produces mediastinitis or a pyopneumothorax.

The routes of possible lymphatic spread have been previously described. Metastases to the lung, liver, brain, bones, and kidneys by way of the blood vessels may also occur.

Symptoms—Early symptoms are often indefinite and cause little suspicion or alarm. There may be a sensation of dryness, tickling in the throat, or a vague discomfort on swallowing. Substernal pressure or distress associated with a sensation of fullness is sometimes present. The most significant symptom is dysphagia, at first with solid and later with soft and liquid foods. In time, progressive weight loss, weakness, and malnutrition follow. Pain is not an early symptom; when present and persistent it generally indicates extension of the growth beyond the esophageal wall.

type are established by esophagoscopy with biopsy.

Treatment—The management depends on the general state of the patient, the site of the primary growth, and the extent of its spread.

Surgical excision offers the best chance of cure for the early cases of lower third and cervical growths, whereas for those cancers in the mid-thoracic portion, the results of surgical procedures have been disappointing, and the recent use of high-voltage rotational radiation therapy has been securing promising results.

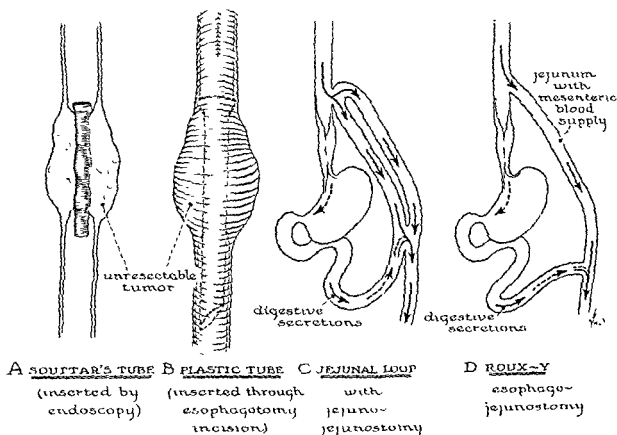


Fig. 285 —Palliative procedures for esophageal obstruction due to malignant disease

Diagnosis—Diagnosis of an esophageal cancer in an early stage is only possible by employing a barium swallow and esophagoscopy in any patient who presents suspicious symptoms. Clinical examination provides little assistance and the earliest lesions can only be diagnosed by endoscopy. The extent of the tumor is best determined by x-ray examination, while the presence of cancer and its histologic

The primary objective of the radical surgical procedures is the complete removal of the growth, together with a length of normal adjacent esophagus above and below the neoplasm, and all the adjacent lymph nodes, with the re-establishment of the continuity of the digestive tract. Unfortunately, when first seen only 50-60% of patients are operable. The remainder present one or more features

which immediately establish the inoperability of the condition, such as metastases to the supraclavicular lymph nodes, paralysis of a vocal cord, Horner's syndrome, tracheo- or broncho-esophageal fistula, evidence of metastases to liver, lungs, bones or other organs, or endoscopic evidence of the fixation of the lesion to adjacent structures.

Because of earlier diagnosis, safer anesthesia, improved operative techniques, and general supportive measures, an increasing number of cases are being explored. Formerly, multiple-stage procedures were performed and included esophagostomy, gastrostomy, resection of the lesion, and, finally, the joining of the upper esophageal segment to the stomach by means of extrathoracic tubular skin grafts (Grey-Turner). In recent years the left thoracoabdominal approach has enabled successful anastomoses to be performed as high as the cervical portion of the esophagus. Continuity is re-established by esophagogastrostomy or esophagojejunostomy after mobilization of the stomach or Roux loop of jejunum. This approach and the associated anastomoses have been especially successful for growths of the lower third of the esophagus. A right transpleural approach is favored for growths of the upper and middle thirds, and similar types of anastomoses are utilized.

A substitute esophagus consisting of an isolated mobilized segment of jejunum with an intact blood supply may be passed through a tunnel in the loose tissue immediately behind the sternum and anastomosed to the esophagus proximally and to the stomach distally.

Recently, anastomosis with the jejunum has been made after passing the loop of intestine through a tunnel in the loose tissue lying just beneath the sternum.

Specialized techniques have been developed for resection of the growths involving the cervical esophagus. When spread to the larynx or cervical nodes is present, concomitant laryngectomy or radical neck dissection is required. The Trotter or Wookey procedure consists of excision of the cervical esophagus and reconstruction by means of a full-thickness skin graft taken from the lateral cervical region, rolled into a tube, and anastomosed between the severed ends of the esophagus.

Others have used a skin graft wrapped around a cone-shaped tantalum-mesh framework which generally can be removed through the mouth in 2-3 weeks, leaving behind a skin-lined tube to bridge the defect.

For the inoperable cases, palliative procedures to minimize the distressing pain, dysphagia, regurgitation, inanition, and associated complications must be considered.

For the purpose of feeding the patient, a Souttar's tube may be placed by endoscopy through the constricted lumen, or a plastic tube with a proximal shoulder may be forced directly through the growth area when thoracotomy proves it to be unresectable. The opening made in the normal esophagus above the growth for the insertion of the tube is sutured. In cases where direct examination indicates that the growth is inoperable, a bypass procedure can be carried out. This is usually accomplished by anastomosing a segment or loop of jejunum to the esophagus above the obstruction.

In other cases a gastrostomy is performed. This was formerly the routine treatment, but it is now known that it in no way prolongs life and alternate procedures are under trial. Associated with the above measures, high-voltage radiation therapy is given and affords symptomatic relief.

Results.—The prognosis in carcinoma of the esophagus is improving but remains poor.

In general, approximately 42-68% of the cases seen by the surgeon are suitable for surgery. Resection is possible in about 78% of these cases, that is, in 53% of all cases. If cases of carcinoma involving the cardiac end of the stomach are included, resection can be carried out in 70% of all cases encountered.

The immediate operative mortality rate is 14% for lesions below the aortic arch and 33% for those located above this level. The average operative mortality varies from 20-25%. Long-term survival statistics indicate that if one includes all types of carcinoma of the esophagus, there is an over-all 24% 3-year survival rate with a 10% 5-year survival rate.

Mustard (1956) reported the experience at the Toronto General Hospital (1937-1953) with 381 cases. For the entire group 8 cases,

or 3%, survived 5 years Cervical growths
had the best prognosis

86 untreated patients—2 survived 2 years
100 patients with palliative gastrostomy—6 sur-
vived 1 year
125 patients with radiation therapy—1 survived
5 years and 4 survived shorter periods

Exploration in 133 cases with 100 excised

Operative mortality, 36.1%
5-year survival for those surviving operations,
19.4%

Garlock and Sweet have separately reported
a significant series of cases with a 5-year sur-
vival record considerably more encouraging

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Chapter 18

Stomach and Small Intestine

Donald R. Webster, MD

STOMACH

Introduction

Primitive man was probably more aware of his stomach than any other internal organ. All the disturbances in his peritoneal cavity, as well as hunger and satiety, were interpreted by the reaction of the stomach. Despite this, our knowledge has grown very slowly and it has been difficult to correlate physiologic, clinical, and pathologic information. As all good surgery must rest on a sound anatomic and physiologic basis, so gastric surgery has progressed only with advances in such fundamental knowledge.

It was the United States army surgeon, William Beaumont, in 1825, who had the imagination and perseverance to study Alexis St. Martin, the Canadian *voyageur* who developed a gastric fistula from a gunshot wound. This is one of the most fascinating stories in medical experience and laid the foundation of gastric physiology. Beaumont collected gastric juice, described the properties, and recognized the influence of emotions on the circulation, secretion, and motility of the stomach. This fundamental work has its modern counterpart in a monograph by Wolf and Wolff describing their subject "Tom," and confirming and extending Beaumont's original observations. The great Russian physiologist Pavlov devised the innervated gastric pouch, demonstrated the psychic phase of gastric secretion, and proved the vagal pathways

of the stimulus. Many modern physiologists, such as Babkin, a pupil of Pavlov's, Cannon, Carlson, Ivy, and others, have gradually extended our knowledge, but much remains to be done. We do not know exactly how the hydrochloric acid of the gastric juice is formed, why the stomach does not digest itself, how painful sensations in the stomach are transmitted, and why ulcers form and heal in some persons and not in others.

Embryology

The stomach appears in the 4th week of embryonic life as a spindle-shaped dilatation of the primitive gut, caudal to the lung bud. Just distal to this the liver bud appears, and from this two dilatations arise which form the gall bladder and the anterior part of the pancreas (the posterior portion of the pancreas arises as a separate bud). There are both dorsal and ventral mesenteries, the latter, however, extending only as far as the umbilicus. The dorsal border and esophageal end of the stomach grow more rapidly than the pyloric end, and a rotation occurs. The dorsal surface turns to the left, and the ventral and pyloric end to the right, following the growing liver. Thus the ventral side becomes the lesser curvature, the dorsal surface the greater curvature, the left side the anterior surface, and the right side the posterior surface. The vagus nerves follow the rotation so that the left becomes anterior and the right posterior. The dorsal

mesentery forms part of the lesser sac and the great omentum. The ventral mesentery by the growth of the liver forms the lesser omentum and falciform ligament.

Anatomy

The stomach varies in size and shape, and must be viewed with the fluoroscope to visualize the individual differences. The cardia is fairly constantly situated at D12 and the pylorus at L1 or L2. The position of the greater curvature, however, depends upon the shape, content, and tonus of the stomach. The organ is described as steer-horn or J-shaped, and any gradation may occur between these extremes, depending on the habitus of the patient and the tone of the stomach at the time of examination.

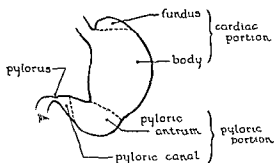


Fig 286—Regional anatomy of the stomach

The stomach is divided into cardia, fundus, body, and pylorus. The cardia is the area surrounding the esophageal opening. The fundus extends to the left and about 1" above the level of the cardia and is in contact with the left dome of the diaphragm. The body is limited by the fundus and the incisura angularis. The pyloric antrum is that portion between the incisura and the duodenum.

The wall of the stomach consists of a serosa of peritoneum, three muscular layers, and a mucosal lining. The serosa covers the stomach except at the lesser and greater curvatures, where it separates to form the lesser and greater omenta. Of the three muscular coats, the external longitudinal layer is continuous with that of the esophagus above and the pyloric sphincter below. Its fibers are scarce over the anterior and posterior surfaces. The middle

circular layer covers the whole stomach and ends as the pyloric sphincter. The inner oblique layer is continuous with the circular muscle layer of the esophagus and is well developed over the fundus of the stomach and fans out toward the incisura.

The mucosa covers the whole of the interior of the stomach. There is a gradual change in the character of the mucosa at the beginning of the pylorus which normally requires microscopic differentiation in human beings. Here the peptic glands are normally replaced by the pyloric glands secreting alkaline mucus. The peptic cells are incorporated into compound racemose glands, which contribute a complex product. The goblet cells of the surface secrete the visible mucus, while the neck cells probably secrete the dissolved mucin. The chief cells containing zymogen granules secrete the enzyme pepsin, and oxyntic cells, hydrochloric acid. Scattered through the gland are the argentaffine cells whose function is not known but may be related to the formation of the intrinsic factor of Castle. Over all is a tall simple columnar epithelial layer continuous with the stratified squamous epithelium of the esophagus above and the intestinal epithelium at the pylorus.

Arterial Supply.—The entire blood supply of the stomach comes from branches of the celiac axis. The left gastric artery runs toward the esophagus upon the diaphragm and passes downward along the lesser curvature of the stomach to end by anastomosing with the right gastric artery. The right gastric artery, a branch of the hepatic artery (occasionally of the gastroduodenal artery), passes to the left along the lesser curvature to anastomose with the left gastric artery. The hepatic artery also gives off the gastroduodenal artery which runs below the pylorus and first part of the duodenum and divides into the superior pancreaticoduodenal and right gastroepiploic artery, passing to the left in the greater omentum and giving off branches to the greater curvature. The left gastroepiploic artery, after arising from the splenic artery, supplies the greater curvature and also the greater omentum. Occasionally this artery anastomoses directly with the left gastroepiploic artery, but more often this anastomosis is made through smaller branches. Arteriovenous anastomoses are present and re-

ported to be decreased during secretory activity. Ligation of the majority of the larger arteries will not cause necrosis or interfere permanently with secretion.

Venous Drainage.—This is almost entirely into the portal system. The right gastroepiploic vein drains into the superior mesenteric vein, a tributary of the portal vein, while the gastric and splenic veins empty directly into the portal vein. The vicinity of the cardia and lower esophagus is one of the most important communication areas between the systemic and portal systems. Esophageal varices may develop from any obstruction in the portal and splenic venous drainage.

Nerve Supply.—The stomach is supplied by branches from the parasympathetic system through the two vagi, and from the sympathetic system by branches from the celiac

ganglion that accompany the blood vessels. The left vagus becomes the anterior vagus below the diaphragm, dividing into a number of branches. Some of these fibers anastomose with the right vagus, but the majority supply the anterior wall of the stomach and the lesser curvature. These disappear beneath the serosa and enter into the plexuses of Meissner and Auerbach. The right vagus nerve, now the posterior, sends branches to the celiac ganglion and breaks up to supply the posterior wall of the stomach, pylorus, and probably the whole intestinal tract, through the intrinsic plexuses. The celiac ganglion receives efferent fibers from the greater and lesser splanchnic nerves. Thus the stomach has a double nerve supply. The vagus is the main secretory nerve of the stomach, but in most other functions the two systems act in a reciprocal or occasionally in a supplementary manner.

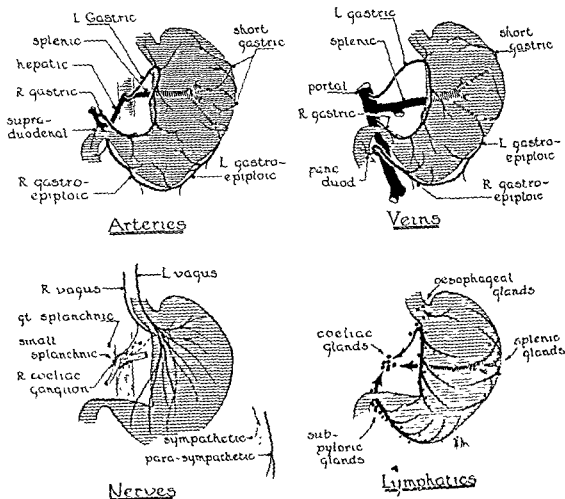


Fig. 287.—Arteries, veins, nerves, and lymphatics of stomach



HYPERSTHENIC

STHENIC

HYPOSTHENIC

ASTHENIC



HYPERTONIC STOMACH



ORTHOTONIC STOMACH

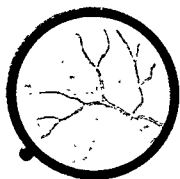


HYPOTONIC STOMACH



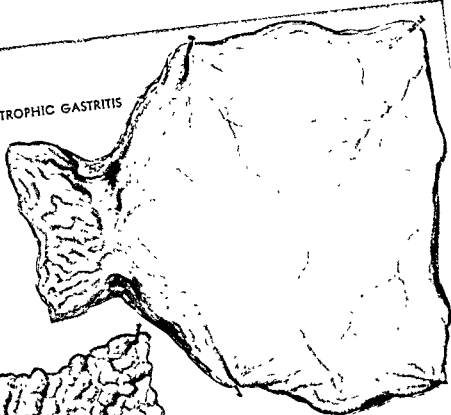
ATONIC STOMACH

Plate 17.—Surgical Relations and Variations of the Stomach.



GASTROSCOPIC VIEW

ATROPHIC GASTRITIS



HYPERTROPHIC GASTRITIS



GASTRITIS
WITH
SUPERFICIAL
EROSIONS

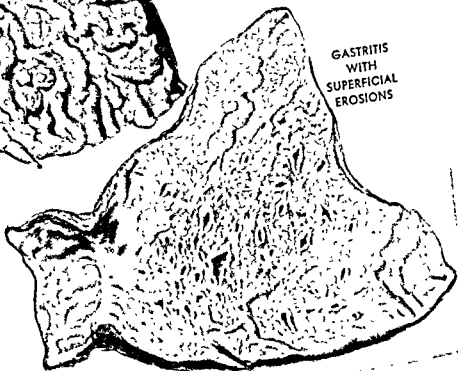


Plate 18.—Gastritis.

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Lymphatic Drainage.—The lymph gathers into radicles in the submucosa. These pierce the muscles and serosa, forming large vessels which, in general, follow the course of the veins. Thus the lymphatics of the lesser curvature drain away from the pylorus, following the coronary vein to the celiac nodes and communicating in their course with the lower esophageal lymphatics. There are numerous nodes scattered throughout the lesser omentum. The lymphatics on the greater curvature are much fewer in number than those on the lesser curvature. The fundus and upper part of the body drain to the hilus of the spleen, while the lower portion and pylorus empty into the subpyloric group and into the celiac nodes. The lymphatics of the pylorus and duodenum do not communicate, thus hindering the distal spread of malignant disease from the stomach.

Physiology

The stomach in addition to being a reservoir has important secretory and motor functions. The stomach secretes hydrochloric acid which was first identified by Prout in 1824. Despite intense investigation the exact mechanism of its formation is not known but is probably the result of a complicated enzymatic process. The hydrochloric acid concentration of pure gastric juice is about pH 1. It is probable that the parietal cells secrete the acid at the same concentration, and variations in acidity are due to diluting and buffering agents such as saliva, food, and mucus. The function of the acid is to provide an optimum pH for the action of the proteolytic enzyme pepsin. Hyperacidity is due to hypersecretion with less opportunity for dilution. This may also be a reflection of the number of actively secreting cells in response to different types of stimuli. The hypersecretion that occurs in duodenal ulcer is the result of the augmented secretory activity of a "large cell mass." Pepsin is secreted by the so-called chief cells. These lie scattered throughout the body of the gastric gland and in the resting state are full of granules that disappear with secretory activity. Pepsin is probably secreted in an inactive state as propepsin that passes into the lumen of the gland to be activated by the hydrochloric acid. The propepsin is also absorbed into the circulation

and excreted in the urine as uropepsin. This can be measured and is reported to be increased in peptic ulcer cases, especially accompanying exacerbations of ulcer activity.

Mucus is secreted in two forms, a visible material which is a product of the surface epithelium and a dissolved mucus secreted by the neck cells. This latter substance contains a mucoprotein probably associated with the so-called intrinsic factor that combines with vitamin B₁₂ to produce a substance essential for hematopoiesis.

The secretion of gastric juice consists of three phases: cephalic, chemical, and intestinal. The cephalic or *psychic phase* is initiated by the sight, smell, taste, or thought of food. The stimulus passes by the vagus nerves to the gastric glands, and this is abolished by atropine and vagotomy. The secretion produced by vagal stimulation accounts for about one half of the total secretion of the digestive period. It is much richer in enzymes than the secretion resulting from the chemical phase.

Hypoglycemia stimulates the vagal centers, producing a large flow of gastric juice. This is used as a test for completeness of the operation of vagotomy. Insulin is given in sufficient amounts to reduce the blood sugar to at least 50 mg %. If no secretion occurs in the stomach, it can be assumed that all vagal fibers have been interrupted, provided a control test produced a secretion before the operation. The lesser curvature of the stomach has a more active vagal innervation than the greater curvature. Stimulation of the sympathetic fibers has a slightly inhibitory effect on the secretion produced by vagal or chemical means. There is evidence that the vagal stimulus exerts its full action only if the pylorus is intact, and, conversely, chemical stimulation of the stomach is reduced if the vagi are cut. This suggests that there is an interdependence in their activities.

The two succeeding phases are the physiologic sequelae to the psychic secretions. The presence of food in the stomach results in the formation of a hormone (*gastrin*) that produces a flow of juice of high acidity but poor in enzymes. This was considered at one time to be histamine, but Komarov proved it to be a separate entity. The pyloric antrum appears to secrete gastrin mainly in an alkaline en-

vironment, and acid produces an inhibitory effect. Dragstedt has shown that if the pylorus is transplanted to the colon, a copious and continuous secretion from the stomach occurs. Harrison et al. have demonstrated that if a portion of the transplanted antrum is left in continuity with the stomach, the total amount of secretion is reduced but rises again if the remaining portion of the antrum is resected. This would indicate that the pyloric antrum has an inhibitory effect on secretion if in contact with the normal acid gastric juice and thus plays a part in the regulation of gastric secretory activity. This hormone appears to be formed mainly in the mucosa of the pyloric portion of the stomach, and it is for this reason that the pylorus should be removed when doing a subtotal gastrectomy. If it is left, gastrin may continue to be formed, and the resulting secretion from the remaining portion of the stomach will predispose to the formation of another ulcer. A similar hormone though less active (*enterogastrin*) is formed in the duodenum and upper jejunum.

Meat and protein extracts are the strongest stimulants of gastric secretion, while carbohydrates have very little effect. Fats stimulate a secretion of gastric juice while in the stomach but, after passing into the duodenum, stimulate the formation in the intestinal mucous membrane of a substance causing marked inhibition of gastric secretion. This substance has been extracted by Ivy and called "*enterogastrone*." There are other gastric secretory depressants formed in the intestinal tract, and Gray has extracted from the urine of normal persons an inhibitory substance termed *urogastrone*. Whether these substances play any part in the normal regulation of gastric secretion is not known.

It was shown by Gray et al. that ACTH will produce a secretion of gastric juice after a long latent period, following vagotomy and removal of the pyloric antrum, but not after adrenalectomy. Stimulation of the hypothalamus will also produce a secretion that is blocked by adrenalectomy. It has been suggested that this is the mechanism of the production of the so-called *Curling's ulcer*.

A diminished secretion and achlorhydria occur frequently in older persons or in persons with diabetes mellitus or carcinoma of the

stomach. Complete achlorhydria is always present in pernicious anemia. It is also found in some cases of hyperthyroidism, Addison's disease, arthritis, and some of the dermatoses.

The stomach exhibits tone and peristalsis. The tone keeps the stomach wall closely applied to the food mass. The peristaltic waves gradually move the liquefied bolus to the pyloric antrum and into the duodenum. There have been many suggestions as to the emptying mechanism of the pyloric valves, such as the acidity of the duodenal contents or the relaxation before an advancing peristaltic wave.

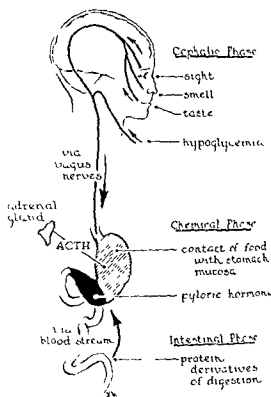


Fig. 288—Mechanism of the stimulation of gastric secretion

Recent work suggests that the pylorus is normally in a patulous state, and when the stomach contents are sufficiently liquefied and diluted, they pass into the duodenum. The bolus remains for a few moments in the first part of the duodenum, to allow perhaps for neutralization. This area is known as the *duodenal cap* when visualized by x-ray; failure to outline this region or demonstration of a deformity is often due to the presence of an ulcer.

The vagus is usually considered as motor to the stomach and the sympathetic as inhibitory, but either nerve may produce the same motor effect, depending on the state of the organ when the stimulus arrives. No "pacemaker" that initiates or controls peristaltic movements has ever been demonstrated.

Absorption.—Only small amounts of carbohydrate are absorbed from the stomach and no fat. Proteins, although split in the stomach, are only absorbed in negligible amounts. Water, alcohol, and probably hydrochloric acid are readily absorbed.

CONGENITAL ANOMALIES

(See Chapter 30)

TRAUMA, WOUNDS, AND FOREIGN BODIES INVOLVING THE STOMACH

Contusion

The stomach may be contused in association with other organs in crushing injuries. There is usually epigastric pain, blood-tinged vomitus, and rigidity of the abdominal wall. It may be difficult to be sure there is not a perforation, and the patient must be watched carefully. The abdomen should be x-rayed to exclude the presence of free gas in the peritoneal cavity suggesting a perforated viscus.

Rupture

Rupture usually results from a blow to the abdomen when the stomach is full of food or distended with gas. An old ulcer may rupture or a small split occur near the cardia. The signs are those of a perforated viscus, and the patient should be operated upon as soon as conditions permit.

Wounds

Gunshot and knives are the commonest agents producing wounds of the stomach. The direction of the missile can usually be estimated and often there is injury to other viscera. If there is no sign of exit of the missile, its position should be localized with x-ray. Operation should then be carried out, not to remove the bullet, but to repair the viscus, and the surgeon must be prepared to deal with any injuries that may be found in other organs.

Foreign Bodies

Innumerable varieties of foreign bodies have been found in the stomach. They can be generally classified into those swallowed:

1. **Accidentally.** These are usually small and will be passed without trouble, such as needles, pins, buttons, coins, teeth, marbles, etc.

2. **Intentionally.** This usually happens with psychotic patients, professional "sword swallowers," attempted suicides, or those hoping to conceal evidence. Articles removed have included nails, glass, razor blades, toys, money, parchment, hair, etc.

Accumulation of swallowed hair is called a bezoar. Vegetable fibers may become entangled with the hair until it fills the stomach, forming a perfect cast. The symptoms are a sense of fullness and foul breath; pyloric obstruction may ensue.

Occasionally penetration of the stomach wall may occur from foreign bodies in the abdominal cavity or neighboring organ, such as instruments, drains, or gallstones.

Treatment.—If the articles are small they will be passed. It is remarkable how large an object will go through the pylorus and ileocecal valve. Such things as open safety pins, needles, and razor blades should be observed with the x-ray, and if not passing satisfactorily should be removed surgically.

VOLVULUS

This is a rare condition where the stomach rotates about its longitudinal or sagittal axis. Radiologists report that a temporary volvulus is not infrequently seen during fluoroscopy. Many of these cases are described with ulcer, diaphragmatic hernia or eventration, neoplasm, or simple distention of the colon. There may be only symptoms of dyspepsia. In acute cases, however, there is vomiting or nausea with inability to vomit, upper abdominal distention, and collapse. Occasionally a stomach tube cannot be passed. A barium swallow will sometimes show the barium confined to a small pouch in the left upper quadrant.

The treatment of acute volvulus is reduction of the volvulus, examination of the stomach for necrosis, and resection if necessary. If the stomach is intact, fixation of the stomach or colon should be carried out.

INFECTIONS

Acute Infectious Gastritis

An inflammatory reaction of the gastric mucosa accompanies many febrile conditions. It is marked by burning epigastric pain, nausea, and vomiting. Achlorhydria may be present. Occasionally hemorrhage occurs which may be confused with bleeding ulcer. The condition disappears as the systemic condition improves.

Acute Suppurative or Phlegmonous Gastritis

Phlegmonous gastritis is a very rare condition in which a part or the whole of the stomach wall may be involved in an acute suppurative process.

It occurs most frequently in the course of some septic process elsewhere in the body but may follow ulceration or trauma of the stomach. The streptococcus is the commonest organism involved, but staphylococci, pneumococci, and the anaerobic groups have been found.

The process spreads in the submucosa, pus collects, and the whole mucous membrane of the stomach may be shed as a cast. The symptoms are those of an abdominal catastrophe, with agonizing steady epigastric pain, nausea, vomiting, rigidity, high fever, and collapse. Recovery may take place, but death usually ensues due to intoxication, or perforation with peritonitis. The differential diagnosis includes perforation, acute pancreatitis, and cholecystitis. Treatment consists of antibiotics, intravenous alimentation, and drainage of any localized abscess. Healing may be followed by a form of linitis plastica.

Acute Gastritis

Acute gastritis may result from the ingestion of corrosives or highly irritating substances. The extent of the reaction depends upon the corrosive action of the material and varies from congestion and edema to ulceration, necrosis, sloughing, and perforation. If the poison has not resulted in a marked corrosive effect on the pharynx and esophagus, or if its character is known, the stomach should be emptied by lavage or emetics. The appropriate antidote should be administered, fol-

lowed by catharsis. The patient may need circulatory support and sedation.

A milder form, sometimes referred to as exogenous gastritis, may occur with the taking of alcohol, extremely hot or cold beverages, of bacterial products, and some medicines such as quinine, iodine, bromides, etc. The symptoms are epigastric discomfort, burning, nausea, anorexia, vomiting, and diarrhea. Achlorhydria is common in the acute stage, and acidity gradually returns as the condition improves.

Treatment.—Usually vomiting has emptied the stomach. Sedation with an opiate is often required, followed by an antacid mixture. Return to a normal diet should be gradual.

Chronic Gastritis

Since the development of the flexible gastroscope by Schindler and others, gastritis has been diagnosed more frequently. Chronic gastritis may be loosely classified as hypertrophic, atrophic, and mixed types. In the hypertrophic type, the rugae are large and coarse and often the site of erosions that bleed easily. There is proliferation of the interstitial cells and lymph follicles, while the submucosa is fibrosed. Mucus is often secreted in large quantities. In the atrophic type, the mucosa is thin and the glandular elements inactive. The muscle is atrophied and fibrotic. All gradations of the condition may occur, representing different stages of the disease.

It has not been our experience that any widespread gastritis is present in stomachs resected for duodenal ulcer but is frequently present in cases of gastric ulcer. It is common in carcinoma of the stomach, which may account in part for the anorexia, achlorhydria, and epigastric discomfort.

Etiology.—There may be a definite and sometimes prolonged history of ingestion of irritants, notably alcohol, or hot, highly spiced foods, some drugs, or infected material from the nose, throat, or sinuses. It may also follow pyloric obstruction with stasis or anastomotic operations. There may be a neurologic mechanism involved. The atrophic variety is associated with some deficiency states, especially pernicious anemia, when achlorhydria is invariably present.

Symptoms.—These may vary from vague discomfort to an ulcerlike syndrome. The

erosions may bleed, varying from a slight ooze to massive hemorrhage. There may be vomiting, and in cases due to alcohol, the vomitus may contain large quantities of mucus.

Diagnosis.—It is usually made by a careful clinical history and by excluding other lesions by such examinations as x-ray, gastroscopy, and gastric analysis.

Treatment.—Treatment is directed toward removing underlying causes and giving a bland diet. Hydrochloric acid should be given in the achlorhydria cases and antacid mixtures in the hypersecretory ones.

Tuberculosis

Tuberculosis of the stomach is rare and is almost always associated with tuberculosis elsewhere in the body. It may result from swallowed sputum, infected milk, or hematogenous spread from a distant focus. Direct extension from another organ may occur. The lesion may ulcerate or form a tumor with caseation in the submucosa, which may then obstruct the pylorus.

Treatment.—The diagnosis is usually made following a laparotomy. Resection is not advised, and the patient should be treated by supportive measures. The prognosis is poor.

Syphilis

Syphilis of the stomach is very rare and occurs only in patients with active disease. There may be an ulcer, but usually the granulomatous process involves the stomach wall to produce the "linitis plastica" type of stomach. Symptoms usually simulate those of other lesions of the stomach and are not characteristic. The gastric crises of tabes may be a feature. The treatment is that used for syphilis, although at times a total gastrectomy may be indicated.

PEPTIC ULCER (GASTRIC AND DUODENAL ULCER)

Ulcerations of the stomach and duodenum have much in common both etiologically and pathologically but appear to be different entities. They are often termed peptic ulcers and in many respects can be discussed together. They occur only in tissues in contact with acid gastric juice, and therefore they are seen oc-

asionally adjacent to aberrant gastric mucosa such as occurs in the lower portion of the esophagus, in a Meckel's diverticulum, or in an anastomosis of the stomach to the jejunum.

While there are many similarities between duodenal and gastric ulcers, there are also marked differences. In duodenal ulcer there is often a marked hypersecretion of gastric juice with high acidity, whereas with gastric ulcer hypersecretion does not occur to the same extent and may show normal levels of acidity.

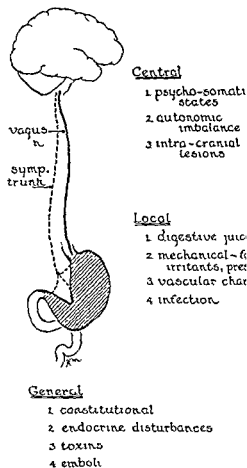


Fig 289—Suggested etiologic factors in the formation of peptic ulcers

The patient with a gastric ulcer is not usually the emotional type so often associated with duodenal ulcer. The most important difference, however, is that while malignancy in the part of the duodenum is extremely rare, malignant ulceration in the stomach is extremely common. The exact incidence of malignant degeneration in a gastric ulcer is not definitely known.

known but is probably in the neighborhood of 7%. Nevertheless, the lesion may be a primary ulcerating carcinoma which requires early surgery if healing is not prompt and permanent

Peptic ulcer is apparently a disease of modern times. While the older writers occasionally mentioned the symptoms suggestive of peptic ulceration, no definite cases were described until the 18th century, and these were described as unique lesions. In the middle of the 19th century several small series of cases were recorded. These were mostly gastric ulcers. Duodenal ulceration was apparently regarded as a rare disease. From 1900 onward there appears to have been a marked increase in the incidence of peptic ulcers all over the world. Patterson states that 12% of the American population have peptic ulcers at some period of their lives. The site of the ulcer has shifted from the stomach to the duodenum so that duodenal ulcers are about 10 times as common as gastric ulcers and far more common in men than in women.

Etiology.—Peptic ulcer appears to be the result of conflict between constitution and environment. The proponents of the neurogenic concept believe that sustained emotional states cause increased secretion of gastric juice and alterations in the gastrointestinal mucosa permitting digestion of the tissue. Cushing demonstrated that lesions of the midbrain could produce ulceration in the stomach and duodenum, and many psychiatrists think that a similar process is involved in some psychosomatic states. This is the basis for the operation of vagotomy.

Another school, originally led by the distinguished pathologist Virchow, believes that ulceration is a local disease due to some devitalization of the mucosa from trauma, gastritis, foci of infection, allergy, emboli, or vascular change in the wall itself. Bigelow and Keyes have demonstrated that stimulation of the peripheral end of the cut vagus produces sludging of blood in the area of the stomach where ulcers frequently occur, thus adding support to both theories. It has been shown on an experimental basis that an ulcer will occur in response to an increase in acid-pepsin production, inadequate neutralization, buffer-

ing, dilution, or a reduction in basic tissue resistance. There are innumerable theories offered for the causation of ulcer, but most evidence is in favor of increased secretion of acid gastric juice. Numerous attempts have been made to produce experimental ulcers in animals by trauma, chemicals, and the diversion of intestinal secretions or contents. These ulcers are usually acute, and if they do not perforate and nutrition is restored, they will heal readily. One must guard against concluding from this that lesions of the gastric or duodenal mucosa, which can be produced in animals by many different types of insults, have any relationship to the chronic ulcer in man.

Zollinger, in 1952 and 1955, reported hypersecretion and intractable and recurring peptic ulcers associated with an atypical alpha cell tumor of the pancreas. It is supposed that this tumor secretes a gastrinlike substance, producing hypersecretion, or that glucagon is a stimulant to gastric secretion. There seems to be agreement that whatever the fundamental cause, the hypersecretion of gastric juice mediated through the vagus is responsible for duodenal ulceration. Dragstedt believes that gastric ulcers are dependent on the hormonal mechanism of the antrum.

Many lengthy series of ulcer cases have been analyzed, and from these certain features are notable:

1. **Age.** While peptic ulcers have been reported in the very young, duodenal ulceration seldom occurs before 20 years of age, and gastric ulceration still later. The age peak of duodenal ulcers is in the fourth decade, and gastric ulcers in the fifth.

2. **Sex.** Peptic ulcers are about four times more common in men than in women.

3. **Occupation.** In the Western world ulcers are more prone to occur in people with financial and administrative worries.

4. **Constitution and heredity.** Duodenal ulcer patients are usually the high-strung worrying type and appear to have a larger secretion of gastric juice than the normal person, especially the fasting and night secretions. There are numerous instances of peptic ulcers occurring in several members of the same family, which may indicate a predisposition to the disease.

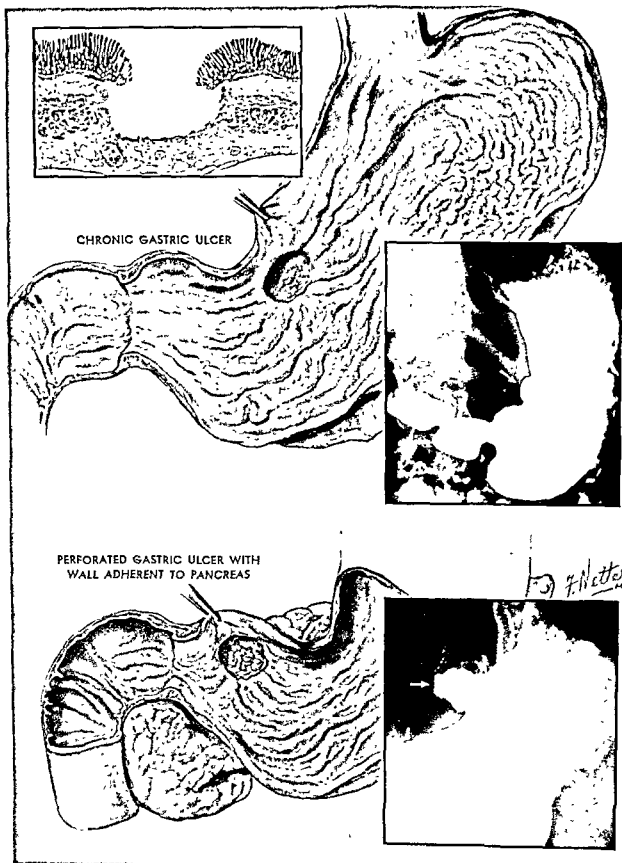


Plate 19.—Chronic Gastric Ulcer.

STOMACH AND SMALL INTESTINE

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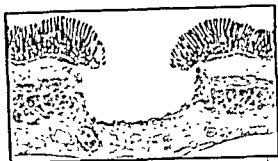
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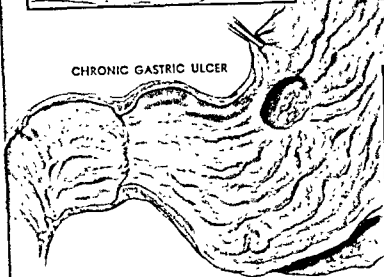
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CHRONIC GASTRIC ULCER



PERFORATED GASTRIC ULCER WITH
WALL ADHERENT TO PANCREAS



5. *Diet* There is no specific article of diet that can be proved to cause ulcer. There is some evidence that those who eat highly spiced foods, take alcohol on an empty stomach, and those who cannot properly masticate food are more prone to develop an ulcer. Lack of a food factor such as vitamin B₁₂, as suggested by Somerville, may play some part. In some regions, such as northern India, peptic ulceration is unknown. Protein deficiency increases the susceptibility to ulcers in the experimental animal, lengthening the gastric emptying time and increasing the incidence of perforation in these ulcers.

6. *Trauma* Peptic ulcers, usually duodenal, occasionally occur in cases of severe trauma or burns and are called "Curling's ulcers." These are probably due to the production of histamine like substances from the traumatized area.

7. *Allergy* There is no direct evidence available that this plays any part in human beings, although previous sensitization increases the incidence of experimental ulcer produced through histamine stimulation.

It is possible that not one but many etiological factors are responsible. There may be a deficiency of some protective substance represented perhaps by Ivy's enterogastrone or some basic endocrine or enzyme disturbance, such as an excess of lysozyme. Whatever the predisposing and initiating factor, it is the digestive action of the gastric juice that actually produces the ulcers and prevents their healing. This has led to the dictum of "no acid, no ulcer," and the reduction of acidity below the optimum for the action of pepsin has been the aim of the most successful medical and surgical forms of treatment.

Pathology.—Gastric ulcers are usually situated in the pylorus or in the lesser curvature in the region of the incisura, occasionally high in the cardiac region. These are the areas where there are few, if any, acid-secreting cells. Duodenal ulcers are most commonly seen in the first part of the duodenum, frequently so close to the pyloric ring that it is difficult to decide on which side the disease began.

Peptic ulcers may vary from minute erosions to complete destruction of all coats of the wall. The ulcer itself may be shallow or deep, depending on its activity. The edges

may be about shelving, or undermining. The base becomes indurated and covered with fibrinous exudate over which a gray slough can be seen. In large ulcers the base is often covered by masses of mucous membrane. If ulceration is formerly, the ulcer may erode through the wall; but the pancreas becomes involved and the base of the ulcer. If the ulcer is situated in the anterior wall, perforation may result into the peritoneal cavity, or it may become adherent to surrounding structures such as the liver or gall bladder. Bleeding may be minimal, but if a large vessel is eroded, serious or fatal hemorrhage may occur.

In chronic ulcers there is always an inflammatory area surrounding the lesion, especially at the base. This may be marked with edema and induration. Vascular disturbances occur with thrombosis and obliteration of the arteries. Evidence of healing can often be observed by proliferation of the epithelium at the edge of the lesion and production of granulation tissues in the base.

In the healing process cicatrization may cause serious complications. Those of the lesser curvature may give rise to "hourglass" stomach and those near the pylorus to obstruction preventing the emptying of the stomach.

Clinical Picture.—*Pain* is the most distressing feature of peptic ulceration. It may vary from a feeling of slight discomfort to a gnawing, burning ache that intrudes into the daily life of the victim and disturbs his rest at night. It can produce a facies depicting suffering and change a person into a hopeless hypochondriac. In the early stages of the disease the location of the pain is indefinite, but if long-continued and an inflammatory reaction is present, the gastric ulcer pain is in the mid-epigastrium and that of duodenal ulcer to the right of the epigastrium.

The pain of duodenal ulcer appears some 2-3 hours after a meal and is relieved by the taking of more food or alkalis. This sequence was described as the "ulcer rhythm" by Moynihan, who epitomized it as gastric ulcer—food, comfort, pain, comfort; and duodenal ulcer—food, comfort, pain. The pain is seldom typical, however, and symptoms may be indefinite.

Sometimes the pain is accompanied by belching, nausea, and occasionally vomiting, which

may relieve the pain. There is frequently a seasonal periodicity to the symptoms, increasing in the spring and autumn, and also a poorly understood individual rhythm, where the patient may be completely free of pain for weeks or months. These are usually periods of transient healing when the crater may disappear in the x-ray picture. Attacks may be precipitated by worry, fatigue, alcoholic beverages, and excessive smoking, and also by certain specific foods, especially coarse vegetables and fried food, which the patient soon learns to avoid. Realizing that food or alkalies will relieve the pain, he often carries some medication with him and to his bedside at night.

stomach is empty. Many investigators have attempted to correlate the hunger contraction of the stomach with the waves of pain in ulcer, but again the relation is not consistent. Others feel that ulcers are only painful when an inflammatory reaction is present. The instant relief produced by vagotomy may be explained by a diminished secretion of gastric juice or the reduction in the motor activity of the stomach.

Physical Signs.—There are no definite physical signs, but ulcer patients are frequently, though not always, thin, having lost weight through a self-imposed diet. The face occasionally portrays suffering or irritability

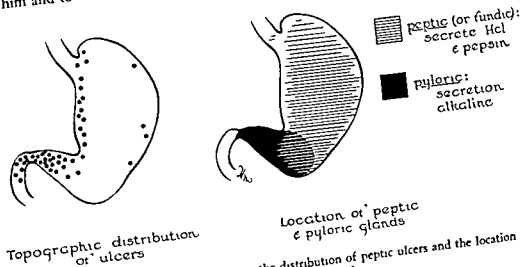


Fig. 290.—Diagrammatic comparison between the distribution of peptic ulcers and the location of peptic and pyloric glands

When the ulcer is active and penetrating anteriorly toward the serosa or posteriorly toward the pancreas, the pain is usually aggravated and assumes a boring, gnawing character which may become continuous, unrelieved by food. This may suggest a dangerous change in the activity of the ulcer, often indicating a progression of the penetration. The mechanism of this pain is not satisfactorily explained. There are many factors that may influence the sensation of pain, such as the size and location of the ulcer, the degree of penetration, and the involvement of adjacent structures, and the pain threshold of the patient. However, the explanation of the pain in uncomplicated ulcer is not satisfactory. Theories such as the contact of hydrochloric acid with the ulcer surface do not explain the presence of pain when the

Pallor may be present if bleeding or malnutrition has been prolonged.

Locally there is usually hyperesthesia over the epigastrium and tenderness on deep palpation. There may be some guarding of the muscles over the ulcer and tenderness over the lower dorsal vertebrae, particularly if the ulcer has involved the pancreas or posterior abdominal wall. If pyloric obstruction is present, a succussion splash can be elicited and visible peristalsis observed.

The palpation of a mass suggests a marked inflammatory reaction about a penetration, and malignancy must be excluded.

Diagnosis.—A careful history and observation of the patient are often adequate to suggest the diagnosis, and confirmatory in-

vestigations can be carried out. Laboratory aids are most helpful, and with their proper utilization a high percentage of correct diagnoses can be obtained.

X-ray studies by means of a barium meal furnish the most useful diagnostic weapon we possess. These yield information about the

lesion, if in the stomach, although the "blind areas" of the fundus limit its usefulness.

Gastric Analysis.—The examination of the stomach contents is at times a valuable aid in establishing a diagnosis. A tube is introduced into the stomach, and the fasting contents are withdrawn. If the volume is large, or if there

Royal Victoria Hospital Gastric Analysis

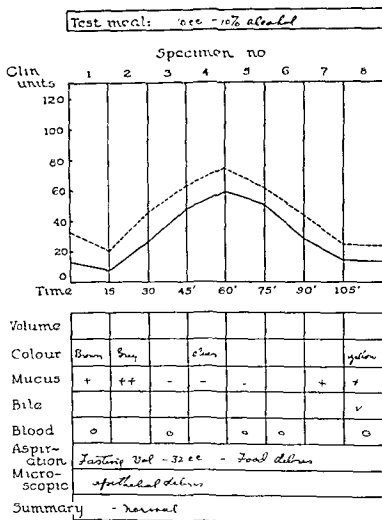


Fig 291—Sample chart of normal gastric analysis

size and location of the ulcer, the patency of the pylorus, the dilatation of the stomach, the deformity of the duodenal cap, or the presence of polyps or diverticula

Gastroscopy—The gastroscope in the hands of experienced observers will frequently demonstrate the site and the character of the

are remains of food taken many hours previously, it is evidence of pyloric obstruction. A test meal of either food or alcohol, which is seldom used now, or a chemical such as histamine or insulin is given, and the contents are aspirated every 15 minutes for 2-3 hours afterward.

The acidity and volume are measured; the presence or absence of blood is determined, and if there is a suspicion of malignancy, a cytologic examination can be carried out. Large quantities of mucus suggest gastritis. If bile is present, the pylorus must be patent. The acidity and volume are usually higher in duodenal ulcer than in gastric ulcer cases, and very high levels suggest an active duodenal ulcer. The absence of free hydrochloric acid or a low acidity in the presence of a gastric ulcer is suggestive of malignancy.

Occult Blood—This is usually positive in all active ulcers.

Treatment.—Surgery is required for those cases of ulcer that resist medical treatment and where complications make it imperative. In uncomplicated ulcer, persistent pain despite adequate rest, diet, and medication is the symptom for which the patient demands relief. Every case, however, must be individually studied. The patient's background, his home, habits, and occupation must be reviewed to eliminate any adverse factors if a high percentage of success is to be attained.

Gastric ulcers, especially in the prepyloric area of the stomach, require surgery if after 3 weeks of intensive medical therapy the ulcer is not completely healed clinically and radiologically. This is necessary because carcinoma develops in 75% of benign gastric ulcers and also because in many cases it is impossible to distinguish benign from malignant ulcers without laparotomy and histologic study.

Gastric ulcers situated near the cardia offer a difficult surgical problem. Their excision with a so-called proximal gastrectomy may be a technically formidable procedure. Dragstedt has suggested that such cases should first have a gastrotomy and a biopsy taken. If the rapid histologic section shows no evidence of malignancy, a low gastrectomy is carried out, including all the antrum. Once the antral factor has been eliminated, the ulcer should heal. It is too early to give an evaluation of this procedure.

The treatment of uncomplicated duodenal ulcer, since malignancy is rare in this area, is primarily medical. Eighty-five per cent of patients respond to adequate medical therapy, thus leaving approximately 15% requiring surgery because of intractability. In both gastric

and duodenal ulcers, surgery is indicated for treatment of the complications, mainly perforation, bleeding, and obstruction. The medical therapy must include correct diet, sedation, antacids, antiparasymphathomimetic drugs, if indicated, and attempted control of home and work environment.

The surgical procedure is planned to remove the lesion where possible and to reduce the acidity to a point where peptic activity cannot take place. Gastric surgery has undergone a long and tedious development, sometimes on a trial-and-error basis, following advances in our knowledge of gastric physiology. The earliest operations attempted closure of perforations. The next step was to direct the gastric contents into the jejunum because the pylorus was stenosed or to prevent the contact of chyme with the duodenal ulcer.

Anterior gastroenterostomy was described by Wolfier in 1881, and posterior gastroenterostomy by Von Hacker in 1885. The immediate results of the operation were good, but a high percentage of recurrence followed after some years. Billroth, in 1881, performed the first partial gastrectomy, uniting the open ends of the duodenum and stomach. As this was not always feasible, he later anastomosed the jejunum to the posterior surface of the stomach. These are commonly spoken of as the Billroth I and Billroth II operations. Later, in 1911, Polya demonstrated his method of anastomosing the jejunum directly to the open end of the resected stomach. This left too large a stoma, and various techniques for reducing the size of the opening and fashioning the anastomoses have been described by Balfour, Finsterer, Hofmeister, and many others.

In cases of pyloric stenosis, pyloroplasties were performed by several surgeons whose methods still bear their names, such as Finney, Heineke, and Mikulicz. These operations alone have now been abandoned in the treatment of peptic ulcer. Ligation of the majority of the gastric vessels was advocated by Wilson Hey but has received little support in this country.

Recently several series of Billroth I procedures have been reported by Harkins, Tanner, and others. When technically possible, it would seem to offer a more physiologic restoration of the food pathway. Tanner, however, has reported several recurrent ulcers. Henley,

perforation
with closureanterior
gastro-enterostomyposterior
gastro-enterostomy

wedge resection



sleeve resection



Billroth I



Billroth II



Polya (posterior)



Polya (anterior)



Hofmeister-Finsterlin



pyloroplasty



total gastrectomy

Fig 292—Surgical procedures in the treatment of peptic ulcer

who interposed a length of jejunum between the resected stomach and duodenum, has reported a similar experience.

We believe the operation of choice is a subtotal gastrectomy of the Hofmeister-Finsterer type, removing about three quarters of the stomach, including the pylorus, and the ulcer site in the duodenum. The anastomosis is made posteriorly with a short loop of jejunum fixed high on the stomach so that angulation will not occur and with a stoma that will admit about two fingers. This procedure removes a larger portion of the fundus with its parietal cells, as well as the antrum with its local secretion and the hormonal mechanism. This reduces the stomach's production of acid while the flow of alkaline pancreatic and biliary secretions through the gastrojejunostomy further decreases the acidity of the gastric contents emptying into the jejunum. Occasionally, due to adhesions or activity, the ulcer cannot be removed, and a procedure involving removal of the pyloric mucosa has been recommended by Finsterer. This is known as an *exclusion operation*. In such difficult cases, however, a vagotomy and gastroenterostomy are safer. The mortality in gastrectomy usually results from a leaking duodenal stump, and the less radical procedure will often effect a cure.

Vagotomy, either thoracic or subdiaphragmatic, disconnects the stomach from cerebral stimuli, thus abolishing the psychic phase of gastric secretion and reducing motility. Because of this latter effect and because there is often some degree of pyloric stenosis, it is advisable to do a gastroenterostomy at the same time to assist in evacuation of the stomach. Some surgeons are combining partial gastrectomy with a subdiaphragmatic vagotomy. Vagotomy is not recommended for gastric ulcer because of the possibility of malignancy. There is still some dispute as to the ultimate results of vagotomy. Some observers report a marked disturbance of gastric and intestinal motility that worsens with time. However, in other centers the results appear satisfactory if combined with gastroenterostomy, and it is adopted as the procedure of choice in uncomplicated duodenal ulcer.

Dragstedt reported that in patients with duodenal ulcers the fasting nocturnal secretion

of gastric juice is 3-4 times that of the normal and that there is about twice as much acid secreted in response to histamine. He found that after complete vagotomy, the response to insulin hypoglycemia is abolished, the response to histamine decreased 60-80%, and the fasting secretion of hydrochloric acid is reduced to 20% of its previous level.

Sara Jordan, reporting for the American Association of Gastroenterologists after a thorough review of several thousand cases, states that subtotal gastrectomy alone or combined with vagotomy has produced somewhat better results in controlling peptic ulcer than gastroenterostomy combined with vagotomy or vagotomy alone. In all cases reviewed the addition of vagotomy to gastric resection has not improved the subjective or objective results of ulcer treatment.

We believe that vagotomy alone should be reserved for the treatment of gastrojejunal ulcer. It is recommended in conjunction with gastroenterostomy in cases where the ulcer is involved in an inflammatory mass where the removal and closure of the duodenum would be hazardous. It may be useful in conjunction with subtotal gastrectomy in the occasional young subject with an abnormally high preoperative acidity.

Complications

The most serious complications of peptic ulcers are perforation, obstruction, and hemorrhage, and in the case of gastric ulcers, malignant degeneration.

Perforation

An ulcer that has penetrated through all coats of the organ is said to have perforated. This may occur slowly, and the inflammatory reaction about the lesion produces adhesions between it and the neighboring organs, so that general peritoneal soiling does not take place, and the adjacent tissue becomes the base of the ulcerative process. This is more likely to occur in posterior duodenal ulcers where the pancreas and posterior abdominal wall may be involved. In anterior ulcers, the liver, gall bladder, or omentum may be the adherent organs. This process is sometimes referred to as subacute perforation. To differentiate such

ulcers from the acutely perforated ones, the term penetrating ulcer is frequently used.

Occasionally a few drops of gastric contents may escape, giving symptoms of acute perforation that rapidly improve, or if posteriorly, may form an abscess in the lesser sac. This is inaptly referred to as chronic perforation.

Acute Perforation.—Acute perforation of an ulcer into the general abdominal cavity is one of the most dramatic of abdominal catastrophes. Frequently, without warning, sometimes following aggravation of ulcer symptoms, the patient is seized with agonizing epigastric pain that soon spreads over the abdomen. He lies on his back with his knees drawn up, the face pale, glistening with beads of perspiration, and sometimes cyanotic. The patient often vomits. The temperature is subnormal, and the pulse is small but not usually over 100. The breathing is entirely thoracic and when one examines the abdomen, it is typically scaphoid, rigid or "boardlike," and tender. The tenderness may be more marked on the right side. X-ray or percussion usually reveals pneumoperitoneum, and the liver dullness is obliterated.

The initial shock usually passes off and the general condition and symptoms improve. The pain is eased, color improves, and the pulse is stronger. The abdomen, however, remains rigid and tender, and the respirations shallow. This is referred to as the stage of reaction which soon merges into that of general peritonitis with distention, hiccough, vomiting, and intestinal paralysis.

If the condition of the patient or circumstances do not permit immediate operation, the stomach should be kept empty by continuous suction, intravenous fluids should be given, and the pain controlled by sedatives. As soon as possible an attempt should be made to close the perforation. It is remarkable that about one third of the patients who have had a perforation repaired by simple closure have no further ulcer symptoms. When the operation is performed within 6 hours of the perforation, subtotal gastrectomy may be carried out, obviating the necessity of a subsequent operation for persistent ulcer symptoms. The treatment of perforation by suction alone should be attempted only by a team skilled in maintaining continuous drainage.

Pyloric Obstruction

As most ulcers are near the pylorus on either the duodenal or stomach side, obstruction may result from spasm, edema, or cicatricial contraction in the healing process. As obstruction develops, the pain is not relieved by food or alkalies, and vomiting becomes a prominent symptom, the vomitus often containing food eaten many hours before.

Dilatation of the stomach is a stimulus to secretion, and the resultant hypersecretion lost to the body because it cannot be reabsorbed results in alkalosis, hypochloremia, and azotemia. X-ray with barium will demonstrate the dilatation and retention.

Continuous aspiration of the stomach contents and the administration of antispasmodic drugs will sometimes relieve the obstruction, indicating that it is due to spasm or edema. If obstruction persists, it is probably due to fibrotic contraction, and complete stenosis is an absolute indication for operation.

Hourglass Contraction

This deformity, dividing the stomach into two cavities, is due to a gastric ulcer, usually involving the lesser curvature. Previous perforations and perigastric adhesions or spasm may occasionally play some part, but the great majority are the result of reaction about a large, lesser curvature ulcer that involves both walls of the stomach—the so-called saddle ulcer. The fibrosis gradually produces a contraction that reduces the gastric pathway to a narrow channel close to the lesser curvature. The symptoms are usually those associated with the ulcer, although fullness, nausea, and regurgitation may be present.

Hemorrhage

Hemorrhage varies from a slight ooze that can be detected as "occult blood" in the stools to a profuse hemorrhage that can be fatal. Small amounts of blood lost continuously may produce a secondary anemia, although this is unusual in ulcer cases.

Frequently the ulcer symptoms are aggravated before hemorrhage occurs and almost completely relieved afterward. This probably represents increased activity and congestion

may vary from a slight ooze to severe hemorrhage. Systemic signs of acute inflammation may be present, and occasionally the inflammatory mass can be palpated.

Diagnosis.—This is made on the history and demonstration of the ulcer by x-ray.

Treatment.—If the symptoms are not controlled by a strict medical regime, operation becomes necessary. The most popular method today is a vagotomy, because if successful it eliminates the more hazardous procedure of a secondary gastrectomy. This latter operation may be very difficult if a primary posterior anastomosis has been carried out

brought into proper nutritional balance, and if conditions permit, a primary radical resection is carried out. If inanition is marked, a loop colostomy proximal to the lesion will lessen the diarrhea and usually result in marked improvement in the patient's weight and general condition. The curative procedure is accomplished by freeing the colon from the mass and either closing the fistula or resecting the involved portion. The gastrojejunostomy is then undone and the involved area of the small bowel is resected. This is followed by a higher gastrectomy and a new gastrojejunostomy.

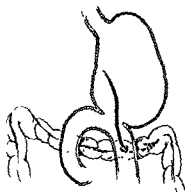


Fig. 294—Gastrojejunocolic fistula

Gastrojejunocolic Fistula

Gastrojejunocolic fistula, as the name implies, is a fistulous connection between the stomach, jejunum, and transverse colon. Except in the rare case where a gastric ulcer perforates into an adherent colon, it is a complication of gastrojejunal ulceration. The colon and enterostomy site are usually bound together in an inflammatory mass, and the fistulous openings are quite close to one another.

Symptoms.—The symptoms are loss of weight, marked diarrhea, foul eructations, and occasionally true fecal vomiting. The diagnosis is confirmed by a barium enema and a barium meal. Both should be done as there may be a valvelike action in one direction that prevents proper demonstration of the lesion.

Treatment.—Medical treatment is of no avail, and the patient will die of inanition unless surgical measures are taken. The patients, who are usually in poor condition, are

Dumping Syndrome

Under the heading of dumping syndrome is grouped a variety of symptoms that may occur after subtotal gastrectomy. These include *vasomotor symptoms* such as muscular weakness, vertigo, flushing, palpitation, sweating, and/or fainting, and *gastrointestinal disturbances* such as epigastric pain, nausea, vomiting, distention, and/or diarrhea. The etiology is not definite but some of the agencies suggested are the following:

- 1 Distention of duodenal loop
- 2 Hypoglycemia
- 3 Traction on mesentery
- 4 Rapid emptying of the irritative gastric contents into the duodenum
- 5 Rapid shift of electrolytes, with potassium deficiency
- 6 Functional

None of these explain all cases. Symptoms usually appear a few weeks after operation and tend to disappear after some months. It is stated that there are fewer cases after a Billroth I anastomosis than after a Polya type of operation. The conversion of a Polya to a Billroth I anastomosis either directly or by the interposition of a jejunal loop has been reported to alleviate the symptoms. Eating a high protein, low carbohydrate diet, with restriction of fluids at meals, and lying down immediately after eating control most symptoms and signs of the dumping syndrome.

CARCINOMA

Carcinoma of the stomach is responsible for one fifth to one quarter of all cancer deaths

It is one of the most frequent of all malignant growths. Livingstone and Pack in 1939 stated: "There are more deaths from cancer of the stomach than from all malignant tumors of lip, tongue, cheek, tonsil, pharynx, larynx, salivary glands, thyroid, male and female breast, ovary, uterine cervix, and corpus uteri combined." It is rare in childhood, uncommon in early adult life, and almost twice as frequent in men as in women. Its insidious growth and trivial symptomatology in the early stages postpone the inevitable visit to the doctor, and even then the frequent vagueness of the complaints often results in palliative treatment for some time before the tragic presence of advanced carcinoma is demonstrated. Until every case of dyspepsia in patients over 35 years of age is suspected, and is examined radiologically, there will be little improvement in our results.

Etiology.—The cause of cancer of the stomach, like cancer elsewhere in the body, is quite unknown. Although heredity and familial predisposition to cancer are suggestive, a causal relationship is statistically difficult to prove. It is essentially a human disease. While other forms of cancer are common in animals, gastric cancer is almost unknown. McCoy examined the gastrointestinal tract of 100,000 rats without finding a single neoplasm. In 381 baboons only one adenoma was found, and it is reported that no case of gastric carcinoma has been found in the enormous amount of material observed in the Chicago stockyards. This marked difference between man and the lower animals has never been satisfactorily explained.

There are geographic and racial differences in the incidence of gastric cancer, but after many studies Pack and McNeer conclude that on the basis of our present knowledge diet offers few clues to the cause of gastric cancer.

The production of adenocarcinoma of the stomach in animals has been very disappointing. The carcinogenic hydrocarbons, deficiency diets, endocrines, parasites, bacterial products, and viruses seem to have no action.

There are, however, three conditions that should be considered precancerous or precursors of cancer of the stomach. These are gastric ulcer, gastritis, and some benign tumors, especially polyps.

Gastric Ulcers.—It has been conclusively demonstrated that cancer may develop in a benign chronic gastric ulcer. It is difficult to estimate the percentage, but it is probably not high. Many ulcers thought to be benign are found to contain cancer cells, when studied after removal. Finsterer reported a series where this was found to be about 20%. It is for this reason that any gastric ulcer that does not heal completely or recurs should be resected.

Gastritis.—While definite proof is lacking that gastritis is a precursor of cancer, the atrophic form of gastritis with achlorhydria, has a higher incidence of cancer than the normal or hypersecreting stomach.

Polyps.—Polyps of the stomach usually occur in association with achlorhydria, and cancer-in-situ has been frequently found in them. While the malignant degeneration is not so apparent as in rectal polyps, they must be regarded as precancerous in a certain number of cases.

Pathology.—Two thirds of gastric cancers arise in the prepyloric area. The lesion generally stops short of the pylorus proper, and invasion of the duodenum is rare. Cancer of the lesser curvature is next in frequency and may arise high in the region of the cardia. Only 5% of gastric carcinomas occur on the greater curvature, but ulcers situated in this area are almost always malignant.

Gastric carcinoma can be divided into four main groups.

1. The ulcerative form is the commonest and arises in the pyloric area or lesser curvature. It varies considerably in size and depth and characteristically has a hard everted edge with a dark red necrotic base which bleeds easily. Microscopically the groups of epithelial cells lie imbedded in abundant fibrous tissue. Infiltration tends to be rapid, although the muscle coat is seldom completely destroyed. The neighboring lymph nodes are invaded very early. It may begin in an apparently normal stomach or at the edge of a chronic peptic ulcer.

2. The papillary or polypoid form is less common and presents as a sharply limited soft mass, projecting into the lumen of the stomach near the pylorus. It may represent malignant degeneration of a benign polypoid

lesion Enlargement is rapid, and ulceration of the mucosa occurs early. As the tumor outgrows its blood supply, necrosis and infection may follow. Microscopically it is an adenocarcinoma but may assume the so-called encephaloid form with scanty stroma and spheroidal cells. It spreads slowly, and may remain localized to the mucosa for a long time.

3. In the infiltrating type a fibrous thickening chiefly in the submucous and subserous coats may cause a stenosis of the pylorus. As the process extends, the body and finally the whole stomach may be involved. It is to this latter type that the term *linitis plastica* or leather-bottle stomach has been given. Ulceration may be present but is superficial. Microscopically it is a scirrhous cancer. Metastases are infrequent and occur late, and this form offers a better prognosis than the other neoplastic lesions of the stomach.

4. Colloid or mucoid carcinoma is an adenocarcinoma but contains large quantities of mucus. It usually appears in the pyloric region, giving a gelatinous appearance to the thickened wall. It soon infiltrates the wall, producing colloid cancer of the peritoneum and the so-called "Krukenberg tumor" described below.

Spread.—Cancer of the stomach spreads by direct extension in the stomach wall and to neighboring organs, by the lymphatics, and by the blood stream. Since most carcinomas occur along the lesser curvature, the lymphatic drainage is to the coronary, hepatic, and pyloric groups of nodes. As the disease progresses, more lymphatics are invaded, creeping along the vessels to the celiac axis to eventually involve the aortic group and the nodes in the hilus of the liver. Occasionally spread from the celiac nodes may occur along the thoracic duct or by way of other mediastinal lymphatics to give rise to metastases in the supraclavicular area (Virchow node). Invasion of the blood stream by malignant emboli may set up metastatic foci in the liver, lungs, bone, and brain in that order of frequency, but this is usually late. Lymphatic involvement of the liver is usually of a spreading pattern from the hilus, while blood-borne metastases are scattered and often numerous.

Extension to the pancreas, liver, colon, and omentum may occur when the growth finally

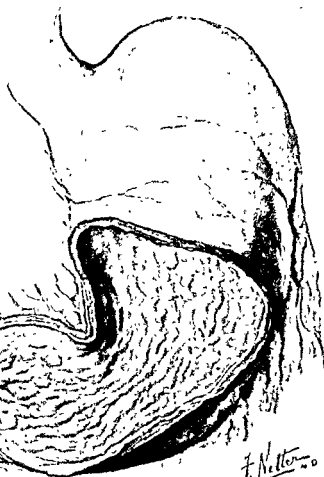
penetrates the wall of the stomach. This is a late sequel, as the serosa appears to offer considerable resistance to the direct spread. Serosal involvement also sets cells free in the peritoneal cavity which may find a foothold on any peritoneal surface. The ovary appears to be particularly susceptible to transperitoneal implantation, producing a bulky secondary ovarian lesion (Krukenberg tumor) characterized by a mucoid cellular degeneration with displacement of the nucleus to form the "signet ring" appearance. If the cells lodge on the pelvic peritoneum, the so-called *rectal shelf* is formed, which can be felt on rectal examination.

Symptoms.—Unless the growth bleeds, or being near the cardia or pylorus causes obstructive symptoms, cancer of the stomach is remarkably free of symptoms. In a mass survey recently reported, Morgan concludes that a much longer asymptomatic period exists than was formerly suspected, lasting perhaps 3 years before the first mild symptoms of indigestion occur. It is for this reason that the onset of dyspepsia, commencing in a patient over 40 years of age who has been previously well, must be regarded as cancer until this has been disproved. Early symptoms are commonly a diminution in appetite and a feeling of distention after a meal. Recurrent attacks of nausea may occur. Pain is late and may be similar to that of peptic ulcer. Insidious hemorrhage usually gives rise to lethargy and pallor, although massive bleeding may occur with a hematemesis or melena. Vomiting is uncommon in the early case unless there is obstruction to the pylorus, while with lesions in the cardia dysphagia is a prominent symptom. Only too often, however, these early symptoms are overlooked until marked cachexia and a palpable epigastric mass place the cancer in the incurable category. Any patient therefore who has dyspepsia, loss of weight, or anemia occurring at or beyond middle age must have a complete examination of the stomach to exclude carcinoma, if he is to be saved by adequate surgery.

If there is a malignant degeneration in a previously benign gastric ulcer, there is a rather sudden change in the dyspepsia. The periodicity of the pain is lost, it becomes more persistent, and is not relieved by food and



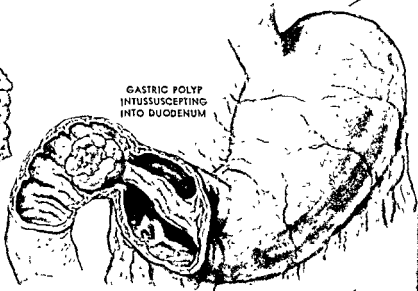
POLYP WITH
PEDICLE



F. Netter M.D.



CIRCUMSCRIBED POLYPOID
ADENOMATA



GASTRIC POLYP
INTUSSUSCEPTING
INTO DUODENUM

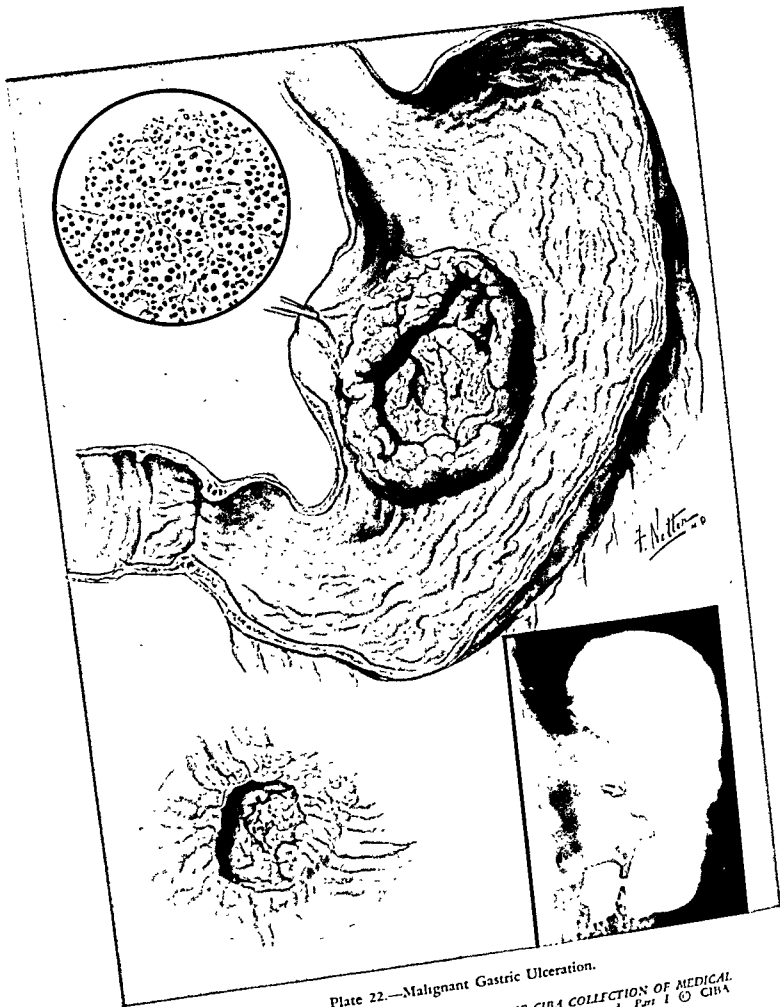


Plate 22.—Malignant Gastric Ulceration.

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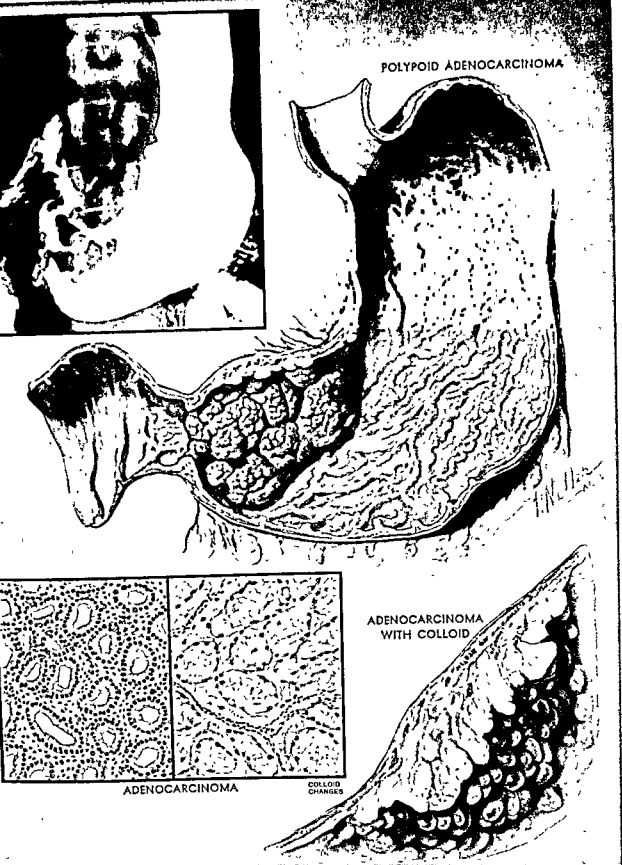
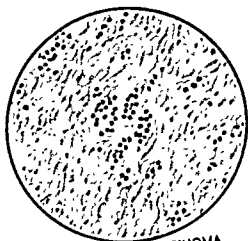


Plate 23 — Polypoid Form of Adenocarcinoma of the Stomach.



DIFFUSE SCIRRHOUS
INFILTRATION OF
GASTRIC WALL

H. Netter M.D.



SCIRRHOUS CARCINOMA
(MICROSCOPICALLY)



MALIGNANT INFILTRATION LIMITED
TO PYLORUS

Plate 24.—Scirrhous or Infiltrating Form of Gastric Carcinoma

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alkali. In a series of cases analyzed at the Royal Victoria Hospital it was shown that the longer the duration of symptoms before treatment was sought, the longer the ultimate survival. This finding means only that the growth was very slow-growing and should not have any bearing on treatment. The following chart shows the signs and symptoms in order of frequency in which they occurred in this series.

SIGNS AND SYMPTOMS IN ORDER OF FREQUENCY OF OCCURRENCE

SYMPTOMS	NO	%
Loss of weight	296	69
Abdominal pain (epigastric distress)	289	67
Vomiting	168	39
Abdominal tenderness	144	33
Anorexia	123	28
Epigastric mass	120	28
Weakness	95	22
Pallor	85	19
Abdominal fullness	70	16
Nausea (alone)	68	16
Constipation	63	15
Cachexia	61	14
Gas eructations	53	12
Dysphagia	35	8
Acute bleeding (vomiting of bright, red blood)	15	3
Tarry stools	50	11

Special Diagnostic Measures.—

Gastric Analysis—In approximately half the cases of cancer there is an achlorhydria or hypochlorhydria. However, the remaining cases show free hydrochloric acid within the normal range.

Gastroscopic Examination—In experienced hands the use of the gastroscope, which allows visualization of the tumor, makes the diagnosis certain in a percentage of cases and may also serve to exclude the diagnosis of cancer. However, the gastroscope has three blind spots, the fundus, the pylorus, and the greater curvature opposite the cardia, which greatly limits its usefulness. A negative gastroscopic examination therefore does not exclude the presence of carcinoma.

Occult Blood—This is often positive, and the test should never be omitted in any gastrointestinal investigation.

Roentgenography—Careful radiologic examination of the stomach with particular attention to the mucosal pattern will demonstrate

the carcinoma in a high percentage of cases. However, repeated examinations may be necessary as the tumor may not be visualized in early cases.

Cytologic Examination—Cancer cells in stomach washings may be detected by special stains. Brushes or abrasive balloons passed to the site of the suspected lesion under fluoroscopic control remove the superficial cells into the lumen of the stomach from which they are aspirated. This method has not as high an accuracy as x-ray examination but is a useful adjunct.

Electrogastrography—An electrode is placed by means of a 2 mm. plastic tube into the lower half of the stomach and the other electrode attached to the skin of the arm as a reference. By this means Morton has shown that carcinoma of the stomach gives a characteristic electric pattern, irregular in amplitude and rhythm and different from benign gastric or duodenal ulcer.

Treatment—The treatment of cancer of the stomach is entirely surgical. While past results have not been too encouraging, the survival rate is gradually rising due to improved technique and the widening of the criteria of operability. However, no large gain will be made until cases are diagnosed and treated earlier.

In a study of 427 consecutive patients with gastric carcinoma admitted to the Royal Victoria Hospital from 1941-1950, the operability and survival rate was summarized as shown below.

EXPLORATION—RESECTION—SURVIVAL
(STUDY OF 427 CASES)

Total number of cases	100%
Explored	82%
Resected	38%
Survived resection	34%
Survived 5 years	11 7%
Survived 5 years (corrected for age)	13 6%

Surgical treatment may be roughly divided into (1) resection for cure, and (2) resection for palliation.

The first may be a subtotal resection, removing the greater and lesser omenta and lymphatics, including all visible disease, or it may be a total resection including all involved areas. The second may be palliative, i.e., a

resection, a gastroenterostomy, or even a total resection to enable the patient to empty or fill the stomach and at least make himself more comfortable. It is frequently of value even in the presence of involved lymph nodes or liver metastases.

Many patients with palliative resections do not die of recurrence in the remaining pouch but of local or distant metastases. They are relieved of the obstruction and the presence of a sloughing lesion within the stomach. They may escape hemorrhage or progressive anemia and the effects of septic absorption from the ulcerating mass and obstruction if the tumor is near the pylorus.

If the lesion is situated in the cardia or proximal portion of the stomach, it may extend into the esophagus. The operative procedure of choice is resection of the lesion with at least 5 cm. of normal tissue, including the lesser curvature, and an esophagogastrostomy. This can be done through a thoracic or thoracoabdominal incision.

Lesions in the distal portion of the stomach are best treated by a subtotal gastrectomy, including the first part of the duodenum. It has not been proved that total gastrectomy gives a longer survival than the less radical procedure. Moreover, patients with total gastrectomies frequently present severe nutritional problems and are difficult to rehabilitate to a useful life.

Total gastrectomy is indicated for patients with extensive involvement such as linitis plastica, if there are no generalized metastases.

Wangenstein and others have been practicing re-exploring patients after some months and removing any accessible metastatic nodes. Radiation has not been found to be of value as treatment. It is apparent that only early diagnosis and aggressive surgery will improve the prognosis of the cancer victim.

BENIGN TUMORS

Such growths are uncommon and consist of adenomatous polyps which may be single or multiple. Submucous lipoma, fibroma, neurofibromas or myofibromas may occur. Pancreatic rests are occasionally found in the gastric wall near the pylorus.

Adenomatous polyps may undergo malignant changes, usually beginning at the periphery of the polyp. Tumors arising from the nerve sheaths are the neurolemmas and the neurofibromas, the latter sometimes being associated with generalized neurofibromatosis. Giant hypertrophy of the epithelium may occur, giving the appearance of polyposis. Hypertrophy of the pyloric mucosa may act as a valve producing intermittent pyloric obstruction. The symptoms are usually vague epigastric discomfort, signs of obstruction and hemorrhage. Achlorhydria may occur. The treatment is surgical removal, and total gastrectomy may be necessary in cases of diffuse polyposis.

SMALL INTESTINE

DUODENUM

The name *duodenum* is derived from the approximate length of this portion of the bowel, the breadth of twelve fingers. It extends from the pylorus to the ligament of Treitz making almost a complete loop. It is arbitrarily divided into four portions.

The first or superior portion, called by the radiologists the bulb or cap, is related above to the neck of the gall bladder and the under-surface of the liver.

The second part descends from the 1st to 3rd lumbar vertebra, forming a curve about the head of the pancreas. It receives the common bile duct and main pancreatic (Wirsung) duct in the ampulla of Vater, and the accessory pancreatic (Santorini) duct about 1-3 cm above this. Posteriorly it rests on the right kidney, renal vessels, ureter, and psoas muscle.

The third portion crosses transversely to the 3rd lumbar vertebra where, as the fourth part, it turns abruptly forward to join the jejunum. This junction is supported by the ligament of Treitz. Posteriorly it is related to the vertebra, aorta, and left renal vessels.

Blood Supply.—The superior pancreaticoduodenal from the gastroduodenal branch of the hepatic artery anastomoses with the inferior pancreaticoduodenal, a branch of the superior mesenteric artery. These vessels run along the concavity of the duodenum, giving off numerous branches to the anterior and pos-

terior surfaces. A separate branch from the gastroduodenal or the hepatic artery supplies the duodenal cap.

Innervation.—The duodenum, like the rest of the gastrointestinal tract, is innervated with sympathetic and parasympathetic fibers through the splanchnic and vagi nerves. These pass to the bowel in the coats of the blood vessels and probably act in a reciprocal fashion. Distention of the duodenum produces nausea and pain referred to the epigastrium.

Motility.—The duodenum exhibits peristalsis and rhythmic segmentation. The gastric peristaltic waves usually stop at the pylorus, but Ivy has described them as passing over to the duodenum. Reversed peristaltic waves are commonly seen with the duodenal contents regurgitated into the stomach.

Secretion and Absorption.—The duodenal mucosa, like that of the small intestine, has the remarkable ability to secrete and absorb simultaneously. Substances such as water, alcohol, and glucose are rapidly absorbed in isolated loops, but it is questionable whether this plays much part under normal conditions. Besides the digestive enzymes, the duodenum secretes such hormones as secretin, a form of gastrin, and other substances affecting the gall bladder, the regulation of gastric secretion and hematopoiesis. These have not been completely elucidated.

Congenital Anomalies

Stenosis and Atresia.—These conditions may occur in any part of the duodenum, and the symptoms of high obstruction appear shortly after birth. If the stenosis is below the ampulla of Vater, the vomitus will contain bile.

Malrotation.—These anomalies have been grouped under three headings:

1. Duodenum dextra. This is usually associated with failure of rotation.
2. Duodenum mobile. This is a persistence of a duodenal mesentery to any or all parts of the duodenum.
3. Duodenum inversum. Here the second portion curves upward to the left.

These conditions are found in the course of investigation for other conditions. It is doubtful that many cases cause symptoms, but

when such occur, they result from partial, intermittent obstruction, with crampy upper abdominal pain and vomiting of bile-stained material.

Bands and Membranes.—These peritoneal folds extend from the gall bladder and under-surface of the liver to the proximal portion of the duodenum. Such a fold is sometimes called Harris' membrane. Occasionally the membrane passes to a point below the duodenum, compressing it, giving symptoms similar to stenosis. This occasionally occurs in infants and results in early vomiting of green-colored material.

Diverticula.—These are out-pouchings usually from the second or third portion of the duodenum. They may be true diverticula, involving all coats of the bowel, or the longitudinal and circular coats may be missing. There is no peritoneal covering unless they are situated anteriorly. If the neck is wide, no symptoms are caused, but if narrow and the diverticulum is in a dependent position, food collects in the pouch and diverticulitis may result, giving epigastric distress and tenderness. If symptoms are severe, the diverticulum may be excised, although when situated in the region of the ampulla of Vater, this may be difficult or impossible. (See also Chapter 30.)

Duodenitis

Duodenitis may occur as an isolated entity or in association with gastritis. It may be part of an inflammatory or neoplastic process of neighboring organs such as the gall bladder or pancreas. It may be the result of stasis and dilatation caused by intermittent obstruction from congenital bands or malposition. It is seldom diagnosed.

New Growths

New growths of the duodenum are extremely rare. Excluding those of the ampulla of Vater, which may arise in the common duct or the rare case of extension of a pyloric or pancreatic cancer, duodenal tumors have an incidence of a fraction of 1%. The benign tumors include adenomas (polyps), myomas, lipomas, hemangiomas, and pancreatic rests.

The pain is inconstant and not related to meals. External fistulas occasionally follow appendectomy. Abscesses may track into the pararectal space.

Diagnosis.—This is made on the symptoms, course of the disease, and the x-ray appearance. A barium enema will exclude a lesion of the large bowel such as carcinoma or diverticulitis, and a barium drink followed by x-ray will often demonstrate the narrowed, rigid ileum.

Treatment.—An expectant approach may be taken when the acute process is found at laparotomy performed on the diagnosis of appendicitis. Some cases apparently arrested without the development of obstruction, abscesses, or fistulas. Excision of the affected portion of the bowel is the best treatment. If there is too much involvement of the mesentery, a short-circuiting operation may be done, such as an ileotransverse colostomy. The involved intestine may be removed later. Good results have been reported by Garlock by doing a short-circuiting operation and bringing the distal loop out as a mucous fistula. The prognosis must be guarded as fistulas sometimes occur after any operative procedure. Toxic signs may be present after a short-circuiting operation, and recurrence may take place, even in apparently healthy bowel.

Tumors

Benign Tumors

Tumors of the small bowel are remarkably rare. The commonest benign growths are adenomas, which may be single or multiple, fibromas, which may arise in any layer; and lipomas, which may be single or multiple and may be pedunculated. Leiomyoma forms a mass projecting into the lumen of the bowel or into the peritoneal cavity. Sarcomatous changes occasionally occur in the growth. Pancreatic rests usually occur as small patches situated anywhere in the intestinal mucosa. They too may undergo malignant changes.

Symptoms.—The symptoms of tumors of the small intestine are usually due to complications such as obstruction, hemorrhage, or intussusception.

Treatment.—When discovered, small tumors may be removed by excision, but if this

is impossible, due to their size, resection of the involved segment of the intestine is indicated.

Malignant Tumors

Adenocarcinomas.—These may arise *de novo* or from degeneration of a benign tumor or pancreatic rest. They may cause obstruction, melena, or an annular stricture. Metastases usually occur early. The treatment is wide excision of the involved area with as much mesentery as possible. If metastases are present, the prognosis is poor. Short-circuiting procedures may be useful in relieving obstruction in cases not suitable for operation.

Sarcoma.—These develop as leiomyosarcomas, round cell sarcomas, and lymphoid tumors. They frequently grow quite large and metastasize early. The treatment is wide excision and the prognosis is bad.

Carcinoid.—These tumors arise from the argentaffine cells and occur most frequently in the submucosa of the appendix and terminal ileum. They may be single or multiple solid nodules which have a golden yellow appearance on section. They may grow to an appreciable size and cause obstruction. A certain number, which some authors place as high as 20%, metastasize to the lymph nodes and liver.

Mesenteric Lymphadenitis

Mesenteric lymphadenitis, which usually occurs in children, is characterized by attacks of crampy abdominal pain, nausea, occasional vomiting, and moderate elevation of temperature and white blood cell count. It is usually diagnosed as appendicitis. At operation the mesenteric lymph nodes are markedly enlarged, reddened, and succulent, and free fluid is often present in the peritoneal cavity. The nodes if removed at operation show only catarrhal inflammation on section, and cultures are sterile. Suppuration is rarely present. The etiology is obscure but it is probably due to a virus. It is frequently associated with lymphadenopathy elsewhere in the body. There is no evidence that it is due to trauma, tuberculosis, parasites, or enteritis.

Diagnosis.—This can usually be made on a history of attacks of colicky pain and tenderness not characteristically situated over the

appendix region. The tender area will sometimes shift to the midline when the patient lies on the left side, due to the falling of the mesentery toward the left (Klein's test).

Treatment.—The attacks tend to disappear as the child grows older. The difficulty, however, is in excluding appendicitis with certainty, and the anxiety of the parents frequently leads to appendectomy. It is remarkable that many patients are completely relieved and others have fewer attacks. While mesenteric lymphadenitis is apparently a self-limited disease, appendectomy appears to have a beneficial effect on the course of the disease.

Mesenteric Vascular Occlusion Mesenteric Thrombosis

The mesenteric veins may be the site of thrombosis occurring in association with cirrhosis of the liver, following splenectomy for some blood dyscrasias, external compression from tumors, and extension of thrombophlebotic processes in the ileocolic or hemorrhoidal veins. The mesenteric arteries, usually the superior, may be occluded by an embolus arising from the mitral valve in endocarditis, the left auricle, the pulmonary veins, or arteriosclerotic plaques from one of the major vessels.

Pathology.—In *thrombosis* the bowel becomes congested, swollen, and cyanosed. There may be a temporary diarrhea due to anoxia, but the bowel soon becomes paralyzed, and gangrene is the usual outcome. However, collateral venous drainage may be established and the patient survives. In *embolism* the extent of the infarction depends on the site of the obstruction. Small areas may be saved by collateral channels, but if the superior mesenteric artery is involved, the whole small bowel and the proximal half of the colon may die. Occasionally circulation is re-established, and patients regarded as hopeless at operation have recovered.

Clinical Picture.—The condition usually appears in middle age, in a debilitated patient, following operation, or suffering from some other lesion as mentioned above. There are signs of an abdominal catastrophe, often with agonizing pain and collapse. There may be vomiting of blood and melena. Death may quickly ensue or follow soon after from obstruction or peritonitis.

Treatment.—The patient must be treated by transfusions and made ready for operation. The involved bowel must then be resected if possible. It is remarkable that many patients have survived with only a small segment of intestine remaining and have lived a reasonably normal life, so that the surgeon need not despair if huge segments of bowel have to be removed. It is possible, in cases of embolism, that the obstruction may be removed from the vessel and that circulation may be restored.

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Film References

Title	Running Time	Sound or Silent	Procureable From
Radical Resection for Carcinoma of the Stomach (1951) (By Samuel F. Marshall, M.D., Boston)	41 min	Sound Color	Ethicon, Inc. New Brunswick, N. J.
Surgery for Massive Hemorrhage from Gastroduodenal Ulcer (Presents a clinical and technical analysis of the management of acute, massive bleeding gastroduodenal ulcer based on two illustrative cases) (1952) (By John D. Stewart, M.D., Buffalo)	33 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.
Gastric Resection for Duodenal Ulcer (A simplified technique for calloused, penetrating ulcer. A retrocolic, antiperistaltic anastomosis is performed) (1949) (By G. Gavin Miller, M.D., Montreal)	27 min	Silent Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.
Gastric Vagotomy and Gastroenterostomy in Treatment of Duodenal Ulcer (Presents the physiologic principles upon which operation is based, anatomy of the vagus nerves at lower end of the esophagus, and a technique for adequate division of these nerves) (1954) (By Lester R. Dragstedt, M.D., Chicago)	26 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.

Liver and Portal Hypertension

James F. Hopkirk, M.D., Richard C. Long, M.D., et al.

THE LIVER

Anatomy

The liver is the largest glandular organ in the body and weighs normally 1,450-1,750 grams. The portal fissure is situated almost in the middle of the inferior surface, and through this enter the portal vein, hepatic artery, and the hepatic plexus of nerves. From the fissure course the hepatic veins, bile ducts, and lymphatics. The liver is covered by a fibrous sheath, Glisson's capsule, which gives rise to the intrahepatic connective tissue. Along with the vessels, the connective tissue subdivides to form the structural support for the hepatic cells segregating them into units known as the liver lobules. The hepatic lobule is the histologic unit, but the functional unit probably consists of two cords of hepatic cells with an enclosed sinusoid on one side and a bile capillary on the other.

Blood Supply—The liver receives its blood supply from two sources: the portal vein and the hepatic artery. Venous blood leaves the liver by means of the hepatic veins which have their origin in the tiny efferent veins that drain each lobule. The lobule receives blood from as many as five portal vein radicles but has only one efferent vein, which arrangement secures a more intimate contact of the blood with the hepatic cells. The portal vein carries to the liver blood which has already drained the stomach, spleen, pancreas, and the large and small intestines. During digestion and

with increase in body metabolic processes, the flow of blood in the portal system is greatly increased. Soon after entering the liver the portal vein divides into successively smaller branches, the smallest of which are found at the corners of the hepatic lobules in the interlobular spaces.

The question has been raised as to whether portal blood is essential for the normal structure and function of the liver. In animals, loss of this source of blood supply produces hepatic atrophy and deranges hepatic function. In human beings, the portal vein has been resected, with survival of the patient.

The branches of the hepatic artery accompany those of the portal vein and supply the connective tissue, the walls of the portal venules, and the bile capillaries, as well as the liver cells themselves. The amount of blood carried by the artery is about 25% of that carried by the portal system. However, the hepatic arterial blood carries an oxygen tension of 85% as compared to 30% in the portal system and is almost solely responsible for the oxygenation of the liver cells.

The hepatic and portal circulations merge via a capillary system in the interlobular spaces of the liver, and in this way mixed arterial and venous blood is supplied to the sinusoids and the liver parenchyma. The pressure differential in the two systems is equalized in the capillary bed. Within the liver lobules the blood flows between columns of liver cells in the sinusoids which are lined with undifferentiated

cells and Kupffer cells of the reticuloendothelial system

The *liver lobule* is a vascular unit, the exit of which is the central or hepatic vein. Each lobule is a collection of cells lying between the central vein and the portal branches with the cells radiating out from the central vein in a spokelike fashion. The cells of each column surround a lumen, the bile capillary

Normal Physiology

Effect of Hepatectomy.—Complete removal of the liver in an animal gives rise to a characteristic syndrome. For 3-8 hours the animal appears normal. Then it begins to show muscular weakness and flaccidity followed in about an hour by muscle twitching, convulsions, and death. These changes are associated with a progressive and profound fall in the blood sugar. By giving intravenous glucose it is possible to keep the animal alive for 18-30 hours. Associated with the lowered glucose curve there is a progressive drop in blood urea and an increase in amino acids, in uric acid, and in serum bilirubin levels.

Functions of the Liver

Excretory.—The liver forms and excretes about 500-1,000 ml. of bile per day at a rate of 15 ml./kilo of body weight with a secretory pressure of 200-341 mm. H₂O. The rate of flow is increased by the choleretic action of bile salts. Stimulation of the sympathetic and vagus nerves has a similar effect, although the latter also exhibits an inhibitory action. The chief components of the bile are bile pigments, the alkali salts of bile acids, organic salts, cholesterol, and mucoprotein. Its reaction is alkaline (pH 7.8-8.6).

Bilirubin.—The formation of bilirubin takes place in the reticuloendothelial system throughout the body from the breakdown of hemoglobin and probably myoglobin and the respiratory enzyme cytochrome-C. It is then excreted by the parenchymal liver cells. The cells alter the bilirubin to some extent as shown by the direct and indirect van den Bergh reaction. The bile pigment has no useful function and is purely an excretory product. Once formed in the reticuloendothelial system the bilirubin is absorbed onto protein fractions in the blood

serum which prevents its excretion by the kidney. This tie is broken by the liver cells, and free bilirubin is excreted in the bile. In the bowel the reducing action of *Escherichia coli* changes it to urobilinogen.

Bile Salts.—The bile salts are a specific secretion of the liver. All are derivatives of cholane, a polycyclic hydrocarbon, which is conjugated in the liver cells with the amino acids, glycine and taurine, to form glycocholic and taurocholic acids. Bile salts have a number of functions; e.g., they aid the digestion of fat by emulsification; the stimulation of the production of pancreatic enzymes, amylase, trypsin, and especially lipase; the absorption of fat soluble vitamins A, D, E, and K. They also help to keep the cholesterol of the bile in solution.

Metabolic.

Carbohydrate.—The liver stores carbohydrate in the form of glycogen and also supplies glucose to the body as it is needed by converting its glycogen reserve. It can also manufacture glucose from protein and to a lesser extent from fat. The entire activity of the liver in carbohydrate metabolism is designed to maintain a normal blood sugar.

Protein and Nitrogen Metabolism.—The liver stores protein, and it manufactures albumin, fibrinogen, prothrombin, and a portion of the globulin fraction. It is the site of urea formation as the end product of the deamination of protein.

Fat Metabolism.—Varying amounts of fat are stored in the liver, depending upon the nutritional state of the individual. The liver also desaturates fatty acids and manufactures ketone bodies which are catabolized by the tissues. Cholesterol is combined with fatty acids in the liver to form cholesterol esters, and this is one of the media for the transportation of fat.

Water Metabolism.—The liver acts as a storage organ for water and as a reservoir for blood and so assists in the regulation of the blood volume.

Vitamin Metabolism.—It manufactures vitamin A from carotene and stores it. It also stores vitamin B and can convert vitamin K to prothrombin.

Pigment and Porphyrin Metabolism.—The liver excretes porphyrins and reconverts some urobilinogen to bilirubin.

Blood Formation.—In the embryo the liver is one of the sites of red blood cell formation. In adults it serves as storage center for the antipernicious anemia factor and for copper and iron.

Blood Coagulation.—It is the site of formation of fibrinogen and prothrombin.

Ammonia Metabolism.—There are three sources of ammonia:

1 From absorption from the gastrointestinal tract of the ammonia derived from proteolytic enzymes and bacteria acting on ingested food.

2 From the functional activity of the kidney, converting glutamin into ammonia and glutamic acid by a special renal enzyme, *glutaminase*. Most of the ammonia is excreted in the urine but some passes back into the circulation.

3 From deamination in protein metabolism. Ammonia is eliminated as urea and uric acid after their synthesis in the liver. High blood ammonia levels are important to the surgeon as they are found in (a) patients who have undergone shunting procedures to ameliorate portal hypertension, and (b) those who have chronic liver disease associated with a gastrointestinal hemorrhage.

INJURIES TO THE LIVER

Etiology.—Injury may occur as the result of blunt trauma to the right upper abdomen or lower thorax, from penetrating wounds, or from operative trauma.

Pathology.—The right lobe is usually involved. The degree of damage varies from simple contusion to subcapsular or even complete rupture. Associated injury of the abdominal and thoracic viscera commonly occurs.

Clinical Picture.—In the absence of an external wound, there may be some difficulty in making the diagnosis. Abdominal pain with tenderness in the right hypochondrium is characteristic. If the tear is large, signs of hemorrhagic shock may appear, and dullness in the flanks may result from the blood in the peritoneal cavity. An x-ray of the chest and abdomen should always be taken to help rule out injury to other viscera.

Treatment.—In the absence of injury to other abdominal viscera, the treatment should be conservative. Whole blood transfusions may be necessary to restore the blood volume and overcome shock. Uncontrolled hemorrhage requires laparotomy and suture of the liver tear.

INFECTIONS OF THE LIVER

Liver Abscess

Etiology.—Abscesses develop from infection which reaches the liver from the systemic circulation in general pyemia, from the portal vein in septic thrombophlebitis, from the bile duct in acute cholangitis, or from a neighboring focus by direct spread.

Pyemic Abscess

Etiology.—In any case of general pyemia, multiple septic emboli are set free, some of which reach the liver via the hepatic artery, causing numerous small abscesses. These are seldom suspected during life unless jaundice occurs. The treatment is that of the general systemic infection.

Pylephlebitic Abscess

Etiology.—Any suppurative process within the abdomen may cause a septic thrombophlebitis in the portal vein. The appendix is the commonest site of origin. The infected thrombus extends along the vein to the liver, or emboli are set free, which are carried to the liver by the portal system.

Pathology.—The abscesses are usually multiple, vary greatly in size, and are more common in the right lobe. If the abscess is situated close to the surface of the liver, it may rupture into the peritoneal or pleural cavity.

Clinical Features.—The antecedent suppurative process is followed after an interval by the secondary development of septicemia with severe chills and fever. Eventually hepatic involvement occurs with right upper quadrant pain, an enlarged tender liver, and jaundice. If the splenic vein is involved, splenomegaly may occur.

Signs of intrathoracic disease indicated by a raised right diaphragm on x-ray examination and signs of pleural involvement are common.

Treatment.—The early treatment of intra-abdominal suppuration by adequate surgery

and appropriate antibiotic therapy will usually prevent pylephlebitis. If the laparotomy reveals evidence of thrombosis of the portal system, the involved veins may be ligated to prevent proximal spread, and a course of anticoagulant therapy should be given.

If liver abscesses develop, they should be drained preferably by an extraperitoneal approach. Anticoagulants and antibiotic therapy should be given in maximum dosage.

Cholangitic Abscess

These abscesses form secondary to acute suppurative cholangitis and hence occur along the intrahepatic bile ducts. The clinical picture is one of intermittent fever, chills, jaundice, and a tender enlarged liver.

The treatment is drainage of the common duct and antibiotic therapy. If any large abscesses are present, they should be drained extraperitoneally.

Amebic Abscess

Etiology.—The *Entamoeba histolytica*, found in many parts of the world, may cause a hepatitis which in some cases goes on to abscess formation.

Pathology.—Amebic abscesses are usually solitary and occur or develop in the right lobe of the liver. The amebae reach the liver via the portal circulation from the primary intestinal ulcers.

The abscess is formed by liquefaction of an area of hepatitis by a proteolytic toxin produced by the amebae. The abscess is typically sterile, and only by scraping the wall can amebae be demonstrated.

Clinical Picture.—An abscess may develop at any stage of the disease. The symptoms are fever, profuse sweating, weakness, and marked gastrointestinal upset. There is pain in the right upper quadrant and an enlarged tender liver. X-ray shows an elevation and immobilization of the right diaphragm. The presence of the *E. histolytica* in the stool confirms the diagnosis.

Treatment.—Emetine and chloroquine have been used in the treatment of the intestinal ulceration and hepatitis. When abscesses form, drainage of the abscess is essential, taking every precaution to maintain the sterility of

the abscess. This drainage may best be done by aspiration at laparotomy.

Actinomycosis

Actinomycosis of the liver is very rare and develops from a focus of infection in the abdomen or thorax (most commonly in the ileocecal region).

Pathology.—The liver is enlarged and adherent to surrounding structures. It is pitted with numerous small abscesses, giving it a honey-combed appearance. The disease may invade the abdominal wall with resulting sinus formation.

Clinical Picture.—The initial symptoms are those of the primary disease. Upper abdominal pain, fever, and an enlarged tender liver are signs of hepatic involvement.

Treatment.—Antibiotic therapy will arrest the disease in many instances. (See Bacteriology, p. 65.) If the abscess is accessible, it should be drained and the necrotic tissue excised.

CYSTS OF THE LIVER

Retention Cysts

Retention cysts are small single cysts, lined with a flattened epithelium, and are thought to be due to a malformation of the smaller bile ducts. They are usually asymptomatic unless large enough to press upon surrounding structures.

Treatment.—The cyst should be excised if symptoms develop.

Polycystic Disease

Polycystic disease frequently occurs in association with polycystic kidneys. The cysts are multiple, vary greatly in size, and contain a clear albuminous fluid.

Treatment.—Treatment is usually unsatisfactory because of the wide distribution of the cysts in the liver and in other organs.

Parasitic Cysts (Hydatid Disease)

Etiology.—The *Taenia echinococcus* is ingested by man in food contaminated by the excreta of dogs and other canines. The embryos penetrate the intestinal wall and are carried to the liver by the portal vein. The

great majority of the embryos are arrested in the liver but may pass through into the systemic circulation and lodge in the lungs or other organs.

Pathology.—When the embryo reaches the liver, it becomes encysted and the cyst gradually increases in size. The fully developed cyst has two layers, an outer laminated fibrous layer and an inner germinal layer from whose cells grow daughter cysts. The parent cyst is usually solitary and contains a clear gelatinous material in which are imbedded many daughter or granddaughter cysts.

Clinical Picture.—When the cysts are small, they are asymptomatic; but as the disease progresses, the cysts enlarge and symptoms develop. Dull pain in the right hypochondrium is common and jaundice may occur. Physical examination will usually reveal a palpable mass on the surface of the liver.

Urticaria, eosinophilia, and other manifestations of the developing allergic phenomena are seen. As calcification of the cyst wall is common, a plain film of the abdomen may be of help in the diagnosis.

There are two confirmatory tests:

1. The Casoni reaction—the intradermal injection of the fresh fluid from animal hydatid cysts.

2. The Chedini-Weinberg complement-fixation reaction.

The cysts may rupture into (1) the peritoneal cavity, (2) the pleural cavity, lung, or bronchus, with the development of a bronchobiliary fistula, or (3) the gastrointestinal tract.

Treatment.—Operation is the treatment of choice. Careful localization of the cyst is essential in order that the incision may be placed correctly. When the cyst is exposed and carefully packed off, it is aspirated and the contents are replaced by a solution of 10% formalin. The cyst is opened and the inner layers are carefully excised. The cavity is closed and the abdomen is sutured without drainage. Partial hepatectomy may be required if the cysts are multiple.

TUMORS OF THE LIVER

Benign.—Adenoma, lymphangioma, hemangioma, myoma, teratoma, fibroma.

Malignant.—(1) Primary carcinoma (hepatoma, cholangioma), sarcoma; (2) secondary.

Benign Tumors

Benign tumors are an uncommon cause of symptoms although they are frequently found at operation and post mortem. Adenomas and hemangiomas may become large enough to present as a mass in the upper abdomen or may press upon neighboring viscera to cause symptoms.

The treatment of these tumors should be excision if possible.

Malignant Tumors

Primary Carcinoma

Primary carcinoma of the liver is a rare disease. It arises from either the liver cells (hepatoma) or from the cells of bile ducts (cholangioma). The *hepatoma* is commoner in the male and is frequently preceded by portal cirrhosis. The *cholangioma* is commoner in females, and this fact may have some relationship to the greater incidence of gall bladder disease in this sex.

Pathology.—The tumor may take the form of multiple nodules, a single large mass, or a diffusely infiltrating growth. In hepatoma the cells are arranged in cords or solid alveoli, and contain bile pigment. Cholangiomas consist of closely packed tubules with a highly vascular stroma. Metastases to other organs are rare.

Clinical Picture.—The onset is insidious with anorexia, loss of weight, and anemia. An enlarged liver, emaciation, ascites, and perhaps jaundice make the diagnosis more obvious. The disease runs a rapid and downhill course with death occurring within 3-4 months after the onset of symptoms. Liver biopsy is necessary to establish a correct pathologic diagnosis.

Treatment.—In the diffuse form or where multiple nodules are present, nothing can be done. Rarely, when a solitary primary tumor is found, resection of a part or all of a lobe may be undertaken. X-ray therapy is without effect.

Primary Sarcoma

Primary sarcoma is an extremely rare lesion. The treatment is one of palliation, although some temporary relief may be expected from x-ray therapy.

Secondary Carcinoma

Secondary carcinoma of the liver is very common in patients with malignant disease. The primary growth may lie in any part of the gastrointestinal tract drained by the portal vein; it may arise elsewhere in the body, e.g., uterus, bronchus, breast, and reach the liver by the systemic circulation; or it may invade the liver by direct extension or by the lymphatics.

The metastases are nearly always multiple. The liver progressively enlarges and presents a nodular surface. Pain, jaundice, and ascites eventually occur.

Treatment.—Treatment should be directed toward the alleviation of symptoms. However, in the rare case where the primary growth has been adequately excised and a solitary metastasis develops in the liver, resection of the involved lobe may be advisable.

SURGICAL JAUNDICE

Jaundice is a manifestation of an increase in the bilirubin level of the blood which gives a yellow discoloration of the skin, sclerae, and mucous membranes. The hyperbilirubinemia results from a number of diverse mechanisms, not all of which are associated with the liver and biliary system.

Pathogenesis of Jaundice.—When the red blood cell is broken down, it divides into an iron-free molecule and an iron-containing portion (hemosiderin). It is the iron-free molecule which is the precursor of bilirubin. The cells of the reticuloendothelial system convert this molecule to bilirubin-globin, which is removed from the blood stream by the liver and is excreted as sodium bilirubinate by the liver cells as one of the components of the bile. In the gastrointestinal tract the bilirubin is acted upon by bacteria and is reduced to urobilinogen, most of which is excreted in the stool. The remainder is reabsorbed and carried by the portal circulation to the liver. Here the greater part is converted to bilirubin, but some reaches the systemic circulation and is excreted in the urine. The unabsorbed urobilinogen in the bowel is oxidized to urobilin which is partly responsible for the color of normal stools.

Jaundice is due either to an increased rate of bilirubin production or to a decrease in its rate of excretion. An increased rate of production may be due to:

1. Increased susceptibility of the red blood cells to hemolysis, e.g., congenital hemolytic jaundice
 2. Presence of hemolysin, e.g., incompatible blood transfusions
 3. An overactive reticuloendothelial system
- A decreased rate of excretion may be due to:
1. Damage to liver parenchymal cells
 - a. In infections, e.g., infectious hepatitis
 - b. Toxic agents, e.g., phosphorus, arsenic
 - c. In cirrhosis
 2. Obstruction to the flow of bile which is usually extrahepatic, e.g., stone or tumor

Classification of Jaundice.—There are three main types; however, it is difficult to differentiate these exactly, as there is frequently an overlap of one type with another.

1 Hemolytic due to the excessive production of bilirubin from the destruction of large numbers of erythrocytes, e.g., congenital hemolytic jaundice

2 Hepatocellular due to a disturbance in the liver cells from

- a. Infections due to virus infection, e.g., leptospiral infection, syphilis, etc.
- b. Chemical poisons: alcohol, arsenic, chloroform, carbon tetrachloride
- c. Biologic substances: incompatible blood
- d. Miscellaneous: portal cirrhosis

3 Obstructive due to some obstruction of the flow of bile

- a. Within the lumen of the bile duct: e.g., stone, inflammatory exudate, parasites
- b. Changes in the wall of the duct: e.g., stricture, inflammation, neoplasm, or spasm of the sphincter of Oddi
- c. Pressure on the ducts from within: pancreatic lesions; hepatic lesions; enlarged glands at portal fissure, duodenal and gastric lesions

4 Mixed: When the initial lesion, e.g., obstruction, is followed by secondary changes in the liver cells which contribute to the duration and severity of the jaundice

The Problem of Diagnosis.—There are many causes of jaundice, but there are only four which commonly confront the surgeon

1. Stone in the common duct
2. Carcinoma of the pancreas
3. Infectious hepatitis
4. Hemolytic jaundice

The differential diagnosis of jaundice presents great difficulty. Much can be learned from an accurate history, but certain laboratory procedures are an essential aid in ascertaining the etiology.

In jaundice due to common duct stone, there frequently is a history of biliary dyspepsia which may be associated with one or

more attacks of biliary colic. The onset of the jaundice is commonly preceded by such an attack. Because the obstructive factor can be intermittent, the depth of the jaundice fluctuates. Slight variation in its depth usually is not detectable clinically, and repeated estimation of the serum bilirubin must be made. The icterus is usually accompanied by pruritus due to the deposition of bile salts in the skin. The latter symptom does not occur in the nonobstructive types of jaundice. Physical examination is of little help although there may be

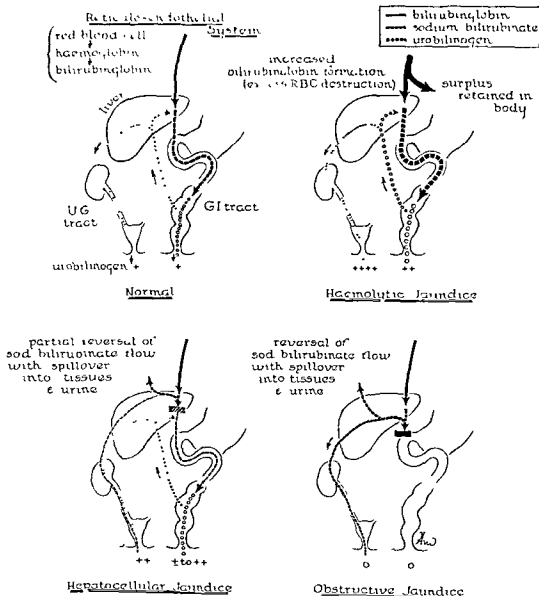


Fig 295—Diagrammatic representation of normal and abnormal bile metabolism. The size of the arrows shows in an approximate fashion the magnitude of abnormal bile or bile product flow.

tenderness over the gall bladder area during the acute attack. The stools are clay-colored at the height of the jaundice but vary in color with the fluctuation in the degree of icterus. The urine is strongly bile-colored, and this is one of the earliest signs of jaundice.

The *jaundice due to malignant obstruction of the common duct* develops slowly and progressively and is not typically associated with attacks of biliary colic, although dull upper abdominal pain is frequent. There are commonly symptoms of anorexia, loss of weight, and a general lack of well-being. If the obstruction is distal to the entrance of the cystic duct, the undiseased gall bladder becomes distended with bile and may be palpable (Courvoisier's law). Occasionally, the jaundice fluctuates due to ulceration of the tumor or to a ball valve action of a pedunculated neoplasm. As in the jaundice of common duct stone, the stools are clay-colored, the urine is strongly stained with bile, and pruritus is a frequent symptom. Small amounts of occult blood may be present in the stools.

The *jaundice of infectious hepatitis* is preceded by a period of anorexia, nausea, and vomiting and is not associated with severe pain. Enlargement of the liver causes a dull ache in the right upper quadrant due to stretching of the liver capsule. The jaundice deepens rapidly, usually lasts for 3-4 weeks, and then gradually disappears. During this period the stools are clay-colored and the urine bile-stained. The liver is enlarged and tender unless yellow atrophy supervenes. Pruritus is absent.

Hemolytic jaundice is due to an excessive destruction of red blood cells. It is characterized by an elevation in the serum bilirubin and the urobilinogen content of the stool and urine. (See Chapter 22, Spleen.)

Liver Function Studies

No single test has been found that will measure liver function accurately, and it is therefore necessary to employ a group of procedures, each of which measures a different aspect of liver physiology. If obstructive jaundice is of long standing, liver function studies are unreliable because of the secondary hepatocellular damage. Once the diagnosis has been established, these tests may be utilized to follow the progress of the disease.

Classification

1. Tests based upon the metabolism of bile pigments
 - a. Quantitative serum bilirubin (van den Bergh)
 - b. Urobilinogen content of urine and feces
2. Tests based upon the excretory function of the liver
 - a. Bromsulphalein
3. Tests based upon the activity of the liver in protein metabolism
 - a. Serum protein: total, albumin, and globulin
 - b. Blood prothrombin time and its response to vitamin K
 - c. Cephalin-cholesterol flocculation test
 - d. Thymol turbidity and thymol flocculation
4. Tests based on the activity of the liver lipid metabolism
 - a. Total cholesterol level of the blood
 - b. Ratio of total cholesterol to cholesterol esters
5. Tests based upon the detoxifying function of the liver
 - a. Hippuric acid synthesis
6. Miscellaneous
 - a. Alkaline phosphatase level

Quantitative Serum Bilirubin.—(Normal: 0.1-0.8 mg/100 ml.) Jaundice is best measured by the quantitative estimation of the serum bilirubin. A slight degree of hyperbilirubinemia may not cause visible discoloration of the tissues. However, jaundice becomes clinically evident when the serum bilirubin level reaches 2.5-3.0 mg/100 ml. The total bilirubin is made up of two fractions, the direct and the indirect. The *indirect van den Bergh* is the measure of bilirubin before it has passed through the liver cells (bilirubin-globin). It is so called because it is chemically inactive and will not combine with the reagent (Ehrlich's diazo reagent) without the addition of a substance which lowers its surface tension. Once it has passed through the liver cells, it is changed to sodium bilirubinate which readily reacts with the reagent and gives the *direct reaction*. Normally 40-75% of the total bilirubin is of the direct variety. An *indirect reaction* is found in cases of hemolytic jaundice.

TABLE 20
URINE AND STOOL BILE AND UROBILINOGEN LEVELS IN JAUNDICED PATIENTS

	URINE		FECES
	BILE	UROBILINOGEN	UROBILINOGEN
Normal	0	+	+
Hemolytic jaundice	0	+++	++
Hepatocellular jaundice	+	0	+ or ++
Obstructive jaundice	++	0	0

TABLE 21
LIVER FUNCTION TEST VALUES IN JAUNDICE

	NORMAL VALUES	HEMOLYTIC JAUNDICE	OBSTRUCTIVE JAUNDICE	HEPATOCELLULAR JAUNDICE
Serum protein	6.9-8.5 Gm./100 ml	Normal	Normal	Decreased
A/G ratio	3:1	Normal	Normal	Reversed
Prothrombin	100%	Normal	Decreased	Decreased
Cephalin-cholesterol	0-1+	Normal	Normal or 2 plus	2 or more plus
Thymol turbidity	< 4 units	Normal	Normal	Increased
Thymol flocculation	0-1+	Normal	0-1 plus	Over 1 plus
Cholesterol total	150-250 mg/100 ml	Normal	Increased	Decreased
Cholesterol esters	75-175 mg/100 ml	Normal	Increased	Decreased
Hippuric acid synthesis	> 1 Gm	Normal	Normal	Decreased
Alkaline phosphatase	3-13 units	Normal	Increased	Normal

while a direct reaction is found in both obstructive and hepatocellular jaundice.

Urobilinogen Content of Urine and Feces—(Normal Urine, 0.3-5 mg./day. Feces, 40-280 mg./day) The estimation of the urobilinogen content of the urine and stools is of great value in the differential diagnosis of jaundice and in evaluating the progress of the disease. In complete obstructive jaundice, where no bilirubin reaches the intestine, urobilinogen will be absent from both the feces and the urine. The stools are clay-colored. In hepatocellular jaundice the ability of the liver to re-excrete the urobilinogen is impaired, the blood level rises, and the amount excreted in the urine is increased. In hemolytic jaundice, with consequent increase in erythrocyte destruction and thus an increased bilirubin formation, the level of the urobilinogen in stool and urine is markedly increased.

Bromsulphalein Test.—(Normal, 95% of dye excreted in 30 minutes) The liver has the capacity to remove certain dyes from the circulation and excrete them in the bile. Liver disease prevents this excretion, and a high percentage of the dye may be retained. As this is a colorimetric test, it is invalidated in the presence of jaundice, and because of the added burden placed upon the liver, its use

for patients suspected of having obstructive jaundice is contraindicated. Its chief value lies in the assessment of liver function in the patients without jaundice. This test is performed by an intravenous injection of 5 mg. Bromsulphalein per kilogram of body weight.

Serum Protein.—(Normal: total 6.9-8.5 Gm./100 ml.; albumin 4.4-6.0 Gm./100 ml.; globulin 1.5-3.0 Gm./100 ml.)

Albumin, globulin, fibrinogen, and prothrombin are formed in the liver. Changes in the metabolism of these protein fractions occur in liver disease. In advanced liver disease a marked decrease occurs in the total serum proteins, and this is chiefly, if not entirely, in the albumin fraction. Occasionally there may be a rise in the serum globulin level which leads to a reversal of the normal albumin-globulin ratio and may even result in an increase in the level of the total serum protein level.

Prothrombin.—Prothrombin is formed by the liver in the presence of an adequate supply of vitamin K. Two factors are therefore necessary for its production—adequate intake and absorption of vitamin K, and a functioning liver. In simple obstructive jaundice the exclusion of bile salts from the intestine and the consequent lack of absorption of the fat-soluble vitamin K result in a decreased forma-

tion of prothrombin. In such cases parenteral administration of vitamin K will bring the prothrombin concentration to normal. In severe hepatocellular damage there is also a decreased prothrombin formation, but this is refractory to the administration of vitamin K. This failure to respond occurs only in the presence of severe liver damage.

Cephalin-Cholesterol Flocculation Test.—In hepatocellular damage, gamma globulin is produced in increased amount, while serum albumin formation is decreased. This change in the serum proteins causes a flocculation of a cephalin-cholesterol emulsion. This test is unaffected by jaundice and is a very sensitive test of hepatic function. Its chief value lies in differentiating obstructive from hepatocellular jaundice. In hepatocellular jaundice strongly positive reactions (3 or 4 plus) are the rule. In liver damage due to long-standing biliary obstruction, the test is positive, but not usually more than 1 or 2 plus.

Thymol Turbidity Test and Thymol Flocculation Test.—(Normal: turbidity, less than 4 units; flocculation 0-1 plus.) This is a flocculation test similar to the cephalin-cholesterol test but is considerably less sensitive. It too is unaffected by the presence of jaundice. In jaundice due to extrahepatic biliary obstruction, the values are within normal limits. In hepatocellular jaundice the values are elevated. The thymol flocculation test is a continuation of the thymol turbidity procedure and in the absence of jaundice is much more sensitive.

Plasma Cholesterol and Cholesterol Esters.—(Normal: total cholesterol 150-250 mg / 100 ml, cholesterol esters, 75-175 mg / 100 ml.) Hypercholesterolemia commonly occurs in obstructive jaundice, and the ratio of total cholesterol to cholesterol esters is normal. In very severe hepatocellular damage, lowered values frequently occur, and the proportion of cholesterol esters is diminished.

Hippuric Acid Synthesis.—(Normal: over 1 Gm.) Benzoic acid is conjugated in the liver with glycine to form hippuric acid which is excreted in the urine. The amount excreted is subnormal in patients with hepatocellular jaundice but is normal in uncomplicated obstruc-

tion to the common duct. The renal function must be normal if this test is to be accurate.

Alkaline Phosphatase.—(Normal: 3-13 units [King-Armstrong].) Alkaline phosphatase is an enzyme produced by the osteoblasts at the site of bone formation. It is excreted by the liver in the bile. In obstructive jaundice the alkaline phosphatase is markedly increased but is normal or very slightly elevated in hepatocellular jaundice.

Liver Biopsy

In combination with liver function studies, a liver biopsy may assist materially in establishing the cause of jaundice. This may be done by aspiration needle biopsy or under direct vision at the time of laparotomy. This diagnostic procedure carries with it the risk of fatality, usually due to intra-abdominal bleeding. The onset of hemorrhage may be delayed for some hours or even days. Bile peritonitis, infection, and pneumothorax are rare complications. To be of value, a representative piece of liver must be obtained, and this is not always possible in a single aspiration. The familiar laboratory cork borer is a useful biopsy instrument.

PORTAL HYPERTENSION

Portal hypertension is a manifestation of a variety of disease processes having in common obstruction to the portal circulation, either within or without the liver (see also Chapter 30).

McMichael (1931) introduced the term *portal hypertension* and suggested that such a derangement might occur in the absence of cirrhosis. This concept was further developed by Whipple and Rousselot (1937), who demonstrated by manometric measurements greatly increased pressure in various portal tributaries in many patients presenting congestive splenomegaly, in whom the liver appeared normal when examined at laparotomy.

The surgical significance of portal hypertension is based on the fact that repeated and ultimately fatal hemorrhages from esophageal or gastric varices may complicate the course of this disorder. To circumvent this eventuality, attempts were made to create an adequate by-

pass or shunt between a major portal tributary and a systemic vein. This was achieved in 1943 by Whipple and his colleagues, and since that time increased interest and experience have made such a surgical procedure an accepted form of treatment in carefully selected cases.

Pathogenesis.—The obstruction of the portal system may be either *congenital* or *acquired*, and the sites of the obstructive factor may be either *intrahepatic* or *extrahepatic*.

In the majority of cases of *intrahepatic block* this is due to cirrhosis.

The *extrahepatic block* follows thrombosis of the portal, superior mesenteric, or splenic vein, which is usually due to *pylephlebitis* sub-

sequent to an inflammatory lesion in the alimentary tract or omphalitis in the newborn child.

Rousselot has divided the obstructions into two major groups, *intrahepatic* and *extrahepatic*, as follows.

1. *Intrahepatic*

a. Cirrhosis of the liver

- (1) Laennec's cirrhosis
- (2) Schistosomiasis
- (3) Biliary cirrhosis
- (4) Infectious hepatitis

2. *Extrahepatic*

a. Stenosis of vein

- (1) Congenital
- (2) Acquired (phleboscclerosis)

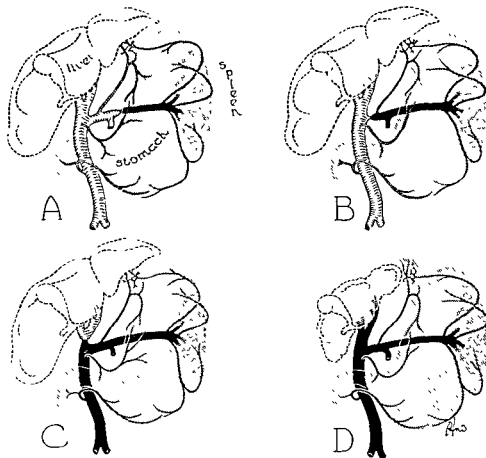


Fig 296—Sites of obstruction causing increased hydrostatic pressure in the portal venous bed

- A, Obstruction of the splenic vein distal to the entrance of the inferior mesenteric vein
- B, Obstruction of the splenic vein proximal to the entrance of the inferior mesenteric and coronary veins
- C, Obstruction of the portal vein
- D, Intrahepatic obstruction due to cirrhosis of the liver

LIVER AND PORTAL HYPERTENSION

- b Compression of vein
 (1) Inflammatory (e.g., pancreatitis)
 (2) Pancreatic cyst
 (3) Tumor
 (4) Aneurysm of splenic artery
- c Thrombosis of vein
 (1) Inflammatory
 (2) Traumatic
- d. Cavernomatous transformation of vein (portal)

The Liver Circulation in Cirrhosis.—Associated with the widespread fibrosis there is a marked distortion of the portal and hepatic

different stages of the disease. It has recently been shown (Madden, 1954) that in cirrhosis with irreversible ascites there is an increase in the arterial and the portal beds and a decrease in the hepatic venous bed. In cirrhosis without ascites or in those cases where the ascites is reversible, the entire vascular bed is decreased.

Clinical Picture.—The principal clinical manifestations of portal hypertension are splenomegaly and the development of collaterals between the portal and systemic veins. These develop characteristically in the esophagus where they give rise to esophageal varices;

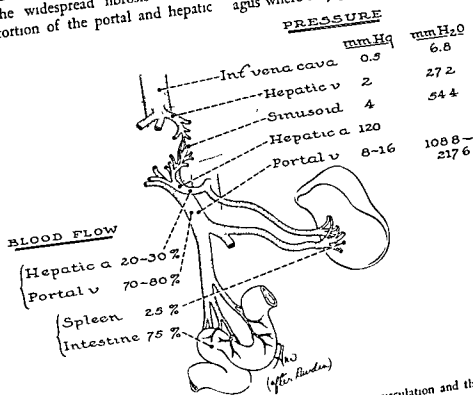


Fig. 297.—Pressures in the various components of the hepatic circulation and the amount of blood contributed by each component (After Ravdin, I S. Ann Roy. Coll Surgeons England 20: 71-98, 1957)

veins, and consequently the liver becomes increasingly dependent upon the hepatic artery as a source of its blood supply. As a result, extensive shunts develop between the branches of the hepatic artery and the portal vein and also between the branches of the portal and hepatic veins. The intrahepatic shunts divert a large portion of the portal blood flow past the liver cells. These shunts in combination with the extrahepatic venous shunts form multiple internal and external Eck fistulas. This phenomenon is not static, varying with

at the anus with the development of hemorrhoids, and in the abdominal sites where the abdominal contents become retroperitoneal developmentally or adhere to the abdominal wall as the result of a pathologic process. It should be realized, however, that portal hypertension may exist without the development of esophageal varices, although other collateral channels may exist. There is an associated anemia, leukopenia, and thrombocytopenia, probably due to hyperfunction of the spleen, i.e., hypersplenism.

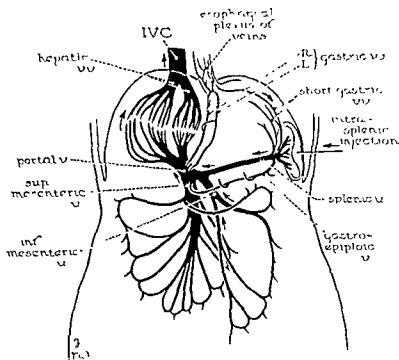


Fig 298A—Normal portal venous system which can be partially visualized by x-ray examination after intrasplenic injection of a radiopaque substance such as Diodrast. In cases of portal hypertension, anastomotic channels, esophageal varices, and abnormalities in the portal and splenic veins can be demonstrated.



Fig 298B—Splenoportogram demonstrating intrahepatic obstruction. *a*, Dye injected by needle into splenic pulp. *b*, Tortuous splenic vein. *c*, Markedly dilated coronary vein. *d*, Gross esophageal varices. *e*, Portal vein.

If the block is *intrahepatic* the liver is usually shrunken and its tests of function abnormal. Total serum proteins are decreased and the albumin-globulin ratio may be reversed. Ascites, swollen legs, and the other manifestations of cirrhosis such as palmar erythema and gynecomastia are usually apparent.

If *extrahepatic* the liver is normal except in the rare instances of purely coincidental cirrhosis.

The presence of esophageal varices can be confirmed by radiologic examination and by esophagoscopy. The dilated anal veins are readily seen on proctoscopic examination. It is interesting that in most cases one or another

Surgical Treatment.—The surgical treatment is primarily directed toward the arrest of hemorrhage and the prevention of its recurrence by measures which reduce the portal pressure or obliterate esophageal varices. Splenectomy will also correct the hematologic manifestations of hypersplenism.

In the intrahepatic type, the selection of patients for operative treatment should be carefully made, as in this group the results of surgery are not as satisfactory as for those of the extrahepatic variety. Severe impairment of liver function as revealed by ascites, jaundice, hepatic coma, or a serum albumin below 3 Gm. % are usually contraindications. However,

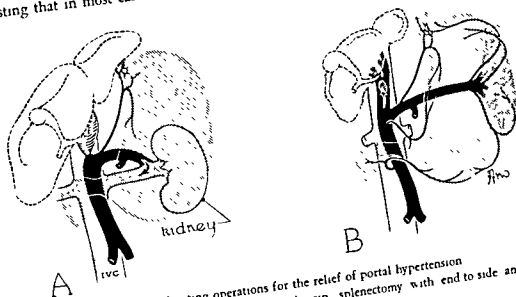


Fig. 299—Standard shunting operations for the relief of portal hypertension
 A, Where the obstruction is in the portal vein, splenectomy with end to side anastomosis of the splenic to the left renal vein is carried out
 B, Anastomosis, side-to-side, of the portal vein and inferior vena cava, in the presence of intrahepatic portal obstruction

of these collateral sites is involved, not necessarily both. The site of the obstruction can often be determined by the use of *portal venography*. This test is of considerable value, therefore, in indicating which of the shunt procedures should be carried out. It is accomplished by the intrasplenic injection of a contrast medium such as Diodrast, with immediate serial x-ray exposures, performed immediately prior to operation because of the danger of serious hemorrhage from the spleen. This gives an outline of the splenic and portal veins with the normal and abnormal anastomotic vessels.

many cirrhotics who bleed can be improved by medical management to the point where they are suitable for surgery. All patients with esophageal varices do not present with hemorrhage, although it can be assumed that if they live long enough bleeding will eventually occur. The immediate cause of the hemorrhage is acid peptic ulceration of the thin-walled varices as a result of an incompetent cardiac sphincter with reflux of the gastric contents. The following procedures may be used:

- 1 To control hemorrhage:
 - a Emergency intra-esophageal tamponade using Sengstaken-Blakemore balloon

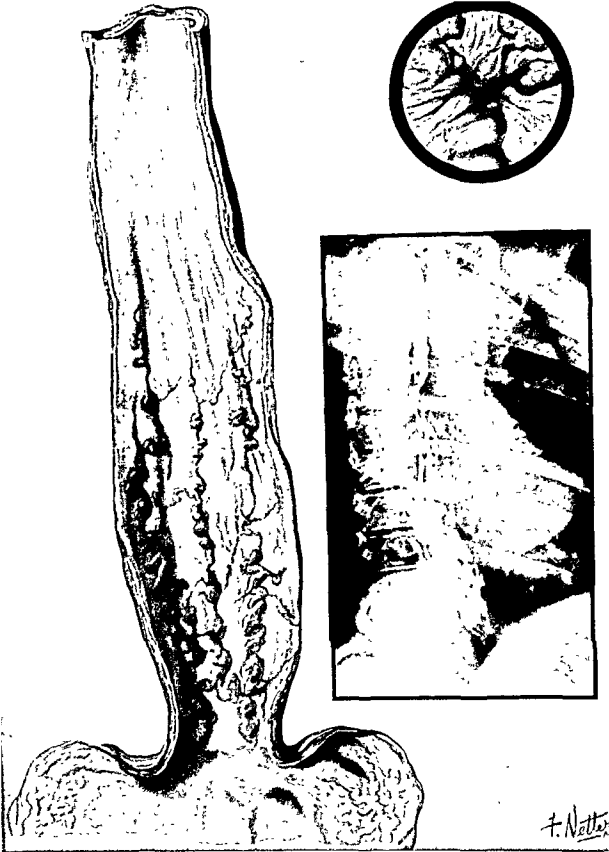


Plate 25.—Esophageal Varices.

The x-ray clearly demonstrates irregular filling defects caused by distended veins in the lower third of this esophagus. The varicose condition, however, commonly extends to the level of the aortic arch or even higher

b. Obliteration of the esophageal varices by direct suture

c. Resection of the lower end of the esophagus and cardiac end of the stomach

2. To reduce the portal pressure:

a. Portacaval anastomosis, either end-to-side or side-to-side (Tck fistula)

b. Splenorenal anastomosis with removal of the spleen and end-to-side anastomosis of the splenic to the left renal vein; left kidney preserved

c. Splenectomy; only of value if the obstruction is situated in the splenic vein between the spleen and the entrance of the inferior mesenteric vein

In order to select the correct shunt procedure it is essential that the surgeon determine the pressure in the different radicles of the portal system. This is done by the insertion of a needle with an attached manometer at the time of laparotomy. The normal pressure is 12-16 cm. of saline, and this may be raised in cases of portal hypertension to 50-60 cm. of saline

If the pressure is elevated in the splenic vein but normal in the superior mesenteric vein, the block is limited to the splenic vein, and the portal vein is clear. If pressures are increased in all radicles of the portal system, then the block is intrahepatic or in the portal vein adjacent to the liver.

At the present time the consensus is that the best procedure in cases of intrahepatic block is portacaval anastomosis. In cases due to an extrahepatic block, this procedure is rarely feasible, and splenorenal anastomosis must be performed. The study of the preoperative portogram is of great assistance in deciding the type of anastomosis for the case in question. Experience has shown that if the establishment of a venous shunt is successful in reducing the portal pressure to a level below 30 cm. of saline, recurrence of serious hemorrhage from esophageal varices is a rare event. It should be realized, however, that the course of associated disease processes such as cirrhosis of the liver is not affected by the operation.

ACUTE HEPATIC FAILURE

Failure of the cellular function of the liver can occur in almost any form of liver disease.

It is found most commonly (1) following operation on the patient with viral hepatitis, (2) in patients with portal cirrhosis with bleeding esophageal varices, and (3) following accidental ligation of the hepatic artery.

Clinical Features.—Hepatic failure is characterized clinically by the development of jaundice, fever, ascites, and neurologic changes (coma).

Hepatic coma is commonly associated with esophageal bleeding in patients with cirrhosis. The symptoms are associated with an increase in blood ammonia secondary to its increased production in the gastrointestinal tract, from the breakdown of the blood protein. This ammonia by-passes the liver, where it is normally detoxified, and causes a functional abnormality in the brain.

Such patients are confused, may have hallucinations, or be frankly comatose and show a characteristic flapping tremor. There may be clonus, an extensor plantar response, convulsions, and sometimes death.

Treatment.—The principle of treatment is the reduction of the protein in the gastrointestinal tract by (1) control of the hemorrhage, (2) removal of the accumulated blood by catharsis, (3) restriction of protein intake, (4) use of antibiotics to diminish the activity of intestinal bacteria which produce ammonia, and (5) the administration of glutamic acid which combines with ammonia to form glutamine.

The prognosis in these cases should be guarded. The process is reversible, and recovery can occur even from severe coma, particularly if the precipitating factor can be removed.

ASCITES

Ascites is an accumulation of fluid in the peritoneal cavity. The commonest causes, in the absence of some local etiologic factor (e.g., carcinomatosis), are cirrhosis and cardiac failure.

Pathogenesis.—The development of ascites is dependent upon a number of factors.

1. Decrease in the serum osmotic pressure due to a lowering of the serum albumin.

2. Retention of the sodium ion. There is almost complete absence of urinary sodium in

REFERENCES

patients with cirrhosis and ascites. Although the serum sodium concentration may be low in these patients, relatively large quantities of salt may be held in the extravascular fluid compartments. The mechanism of this retention is not completely clear, but it is probably due to increased renal tubular absorption, possibly to an antidiuretic hormone, and may also be due to adrenocortical activity.

3. Obstruction to the hepatic venous outflow. This obstruction may be due to obliterative fibrosis of the veins in those patients with irreversible ascites, or to intrahepatic cellular edema in those patients with reversible ascites from either cirrhosis or cardiac failure. In malignancy there may be blockage of these veins by tumor.

Treatment.—Primarily, the treatment of ascites is medical, i.e., the restriction of sodium intake, the administration of ionic exchange resins, and a diet high in protein, carbohydrate, and vitamins. Intravenous serum albumin is of limited value, as it rapidly goes into equilibrium with the albumin of the ascitic fluid. The surgical treatment at the present time is unsatisfactory. There are two promising avenues of approach: (1) the establishment of an isolated segment of ileum with the mucosa exposed to the peritoneal cavity, which effectively absorbs the ascitic fluid, and (2) the establishment of a collateral circulation or shunt to bridge the blocked outflow tract. The use of extrahepatic portacaval shunts and ligation of the hepatic artery will at times prevent the accumulation of ascitic fluid.

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Film References

Title	Running Time	Sound or Silent	Procured from
Splenorenal Anastomosis for Portal Hypertension (Depicts portacaval anastomosis between the end of the splenic vein and the side of the renal vein) (1930) (By C. Stuart Welch, M.D., Albany)	18 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.
Portacaval Shunt (Depicts the technique of portacaval shunt as performed through a right thoracoabdominal approach) (1951) (By John P. Heaney, M.D., Houston)	31 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.
Transesophageal Ligation of Esophageal Varices (1951) (By George Crile, Cleveland)	15 min	Silent Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.

Chapter 20

Biliary System

James F. Hopkirk, MD, and Richard C Long, MD

ANATOMY

The hepatic duct is formed in the depth of the transverse fissure of the liver by the union of the ducts draining the right and left lobes. It runs inferiorly and posteriorly in the edge of the lesser omentum, lying anterior and to the right of the portal vein. The hepatic artery usually lies a short distance from its left margin.

The gall bladder is a thin-walled, musculo-membranous organ about 10 cm in length which lies in a fossa on the inferior surface of the right lobe of the liver. It is attached to the liver by loose connective tissue and is covered with peritoneum that is reflected from its sides onto the liver. The organ is divided into three parts: fundus, body, and neck. The wide end or fundus usually reaches the anterior border of the liver and may come in contact with the anterior abdominal wall. The normal gall bladder is never palpable, but if distended, it may be palpable in the angle between the right rectus muscle and the costal margin. The body lies in close relation to the duodenum, pyloric end of the stomach, and transverse colon. The neck is an S-shaped tube which empties into the cystic duct. Usually there is a sacculcation at the neck, known as Hartmann's pouch.

The mucosa of the gall bladder consists of a single layer of columnar epithelium and may evaginate and project into the muscularis mucosae. These evaginations, which are found

in the normal gall bladder, are known as Rokitsansky-Aschoff sinuses.

The cystic duct runs posteriorly and medially to join the hepatic duct.

The common bile duct is the direct continuation of the hepatic duct after its junction with the cystic duct. It passes inferiorly in the edge of the lesser omentum, then behind the first part of the duodenum to run in a groove in the head of the pancreas and enter the second part of the duodenum at the ampulla of Vater, 2½ cm below the pylorus.

Blood Supply.—The common hepatic artery arises from the celiac axis, passes superiorly in the lesser omentum, and divides near the liver into the right and left hepatic arteries. The cystic artery usually arises from the right hepatic branch. It lies a short distance to the left of the cystic duct and supplies both the duct and the gall bladder. It should be emphasized that the course of the artery and its relation to the adjacent structures are subject to wide variation, and it is important therefore that all structures should be clearly visualized at operation.

Lymphatic Drainage.—Most of the lymphatic drainage converges on the cystic lymph node, which lies close to the junction of the cystic and common ducts, and from this node to the nodes in relation to the head of the pancreas and portal vein. Some of the lymphatics pass across the bare area of the gall bladder wall directly into the liver.

Nerve Supply.—The innervation of the gall bladder and bile ducts is through the splanchnic nerves and the right branch of the vagus nerve

EMBRYOLOGY

During the 4th week of fetal life a solid budlike outpouching forms on the ventral wall of the primitive foregut, from which develop the liver and the extrahepatic biliary system. This solid bud forms vacuoles which coalesce to form the gall bladder and ductal system. Any arrest of development at this stage may result in stricture or a congenital absence of the ducts or gall bladder. If, on the other hand, the bud splits in an abnormal manner, a double gall bladder, aberrant ducts, or diverticula of the gall bladder may result

directly into the duodenum. The liver secretes 500-1,000 ml. in 24 hours, and although the gall bladder holds but 30-50 ml, its concentrating power enables it to hold almost all the bile secreted during that period with the addition of some mucus which is secreted by the mucosa.

The ingestion of food stimulates the production in the duodenum and upper jejunum of the hormone *cholecystokinin* which produces a tonic contraction of the gall bladder. There is a reciprocal relaxation of the sphincter of Oddi, either in response to direct stimulation to acid chyme or to a nervous mechanism, allowing the concentrated gall bladder bile to enter the duodenum

The components of the bile are bile salts, bile pigments, and cholesterol. The bile salts,

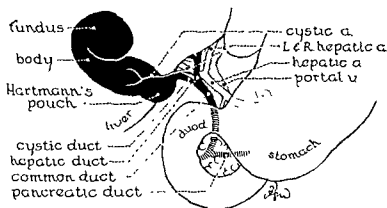


Fig. 300—Normal anatomy of the extrahepatic biliary system. Note how the intrapancreatic portion of the common duct can be occluded by disease in the head of the pancreas

PHYSIOLOGY

The chief functions of the gall bladder are to serve as a reservoir for liver bile, to control the pressure within the biliary tree, and to concentrate bile. The gall bladder also secretes mucus and at times cholesterol. The liver continually secretes a thin watery bile which does not contain mucin. The secretory pressure of the liver is 300-360 mm. of bile. If the intraductal pressure rises above that level, the liver ceases to secrete and jaundice occurs. Closure of the sphincter of Oddi at the terminal end of the common duct allows the pressure within the duct to rise sufficiently to force the bile into the gall bladder where it is concentrated 4-10 times. When the gall bladder is filled, the sphincter relaxes and the liver bile enters

sodium taurocholate and sodium glycocholate, are the most important constituents. They help to keep the cholesterol of the bile in solution, facilitate the digestive action of the pancreatic enzymes, particularly lipase, assist in the absorption of the products of fat cleavage, and stimulate the liver to produce additional bile. When bile salts fail to enter the intestines, 25-75% of ingested fat is lost in the feces.

DIAGNOSTIC PROCEDURES

Simple Roentgenography.—Most gallstones are not shown on plain roentgenograms, as only those with a high calcium content cast a definite positive shadow. Cholesterol and pigment stones are not radiopaque. Therefore, a negative roentgenogram is of little signifi-

cance in excluding disease of the gall bladder. Plain films of the abdomen may also demonstrate calcification of the gall bladder, gas in the biliary tree, a concentrated mixture of bile and calcium carbonate, or occasionally a soft-tissue shadow when carcinoma of the gall bladder is present

Cholecystography.—The concentrating power of the gall bladder is of great clinical importance because the Graham-Cole test for cholecystic disease is based on the assumption that a diseased gall bladder will not concen-

1. Failure to absorb the dye from the gastrointestinal tract, e.g., in vomiting or diarrhea
2. Failure of the liver to remove the dye from the blood stream and excrete it, e.g., in liver disease
3. Obstruction to the cystic duct
4. Premature emptying of the gall bladder before films are made, e.g., following ingestion of food prior to radiography
5. Presence of other shadows in the right upper quadrant which interfere with interpretation of the gall bladder shadow

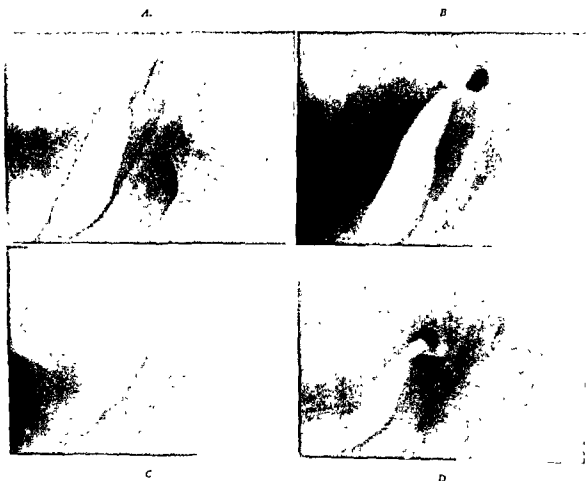


Fig 301—Cholecystograms of normal gall bladder showing the contraction which occurs after a fatty meal

A, Before fat

B, Ten minutes after feeding

C, Twenty minutes after feeding

D, Thirty minutes after feeding

trate radiopaque dye that is excreted by the liver. In a relatively small number of patients several factors may influence the accuracy of the test

6. Failure of the gall bladder to concentrate the dye to an extent sufficient to produce a shadow
7. Failure of the patient to take the dye

The original contrast medium was tetraiodophenolphthalein, but this has been replaced by iodoalphonic acid (Priodax) and iopanoic acid (Telepaque). These are preferably given by mouth. This test will give a positive diagnosis of cholelithiasis in a high percentage of cases. The accuracy of the test is considerably less in noncalculous disease of the gall bladder. Normally 12-14 hours are required following the ingestion of the dye for a satisfactory concentration to occur in the gall bladder.

Intravenous Cholangiography.—An intravenous dye having a very high iodine content (Cholografin) is a useful tool in demonstrating the various parts of the biliary tree. This is concentrated by the liver so that the hepatic and common bile ducts are visualized before the gall bladder is seen. It is particularly useful (1) after cholecystectomy, to demonstrate dilatation of the common duct, retained stones, or cystic duct remnant, (2) as a preoperative cholecystogram when oral cholecystography fails, (3) for children, and (4) for the investigation of tumors of the extrahepatic biliary system or pancreas which may partially occlude the common duct. However, the distal end of the common bile duct is often poorly visualized by this means, and the gall bladder shadow is not as dense as that visualized with Telepaque. The dye should not be used in the presence of jaundice, liver damage, or for those patients who have an iodine idiosyncrasy. If there is more than 30% retention using the Bromsulphalein test, the dye will not be excreted. In order to visualize the extrahepatic ductal system, several films must be taken within the first hour as the excretion is rapid.

Cholangiography.—A radiopaque solution, e.g., iodized oil, injected into the gall bladder or bile ducts, yields valuable information concerning the shape, content, and emptying of the main bile ducts. This procedure may be carried out during the operation where there is obstruction to the common duct or at a later date to ensure patency of the common duct prior to removal of the indwelling T tube.

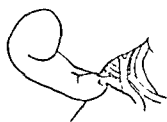
Duodenal Drainage.—Duodenal intubation is of value as a diagnostic measure. A tube is passed into the duodenum, and gall bladder contraction is stimulated by the introduction of magnesium sulfate or olive oil. The pres-

ence of crystals of cholesterol and calcium bilirubinate in the aspirated material is confirmatory evidence of biliary tract disease.

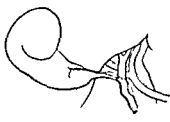
PATHOGENESIS OF GALL BLADDER DISEASE

The experimental and clinical data on the bacteriology of the normal and inflamed gall bladder make untenable the common belief that bacterial infection is the usual cause of cholecystitis. While positive cultures have been obtained from the gall bladder wall, from the cystic lymph gland, and from the bile, there is no certain evidence that this phenomenon indicates more than secondary infection of the previously damaged viscus.

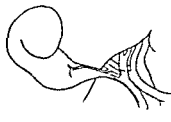
The existing evidence indicates that *cholecystitis* is nearly always due to chemical agents that are normally present in the body, namely, pancreatic juice and bile salts. While reflux of pancreatic juice can cause cholecystitis in man, proof is lacking that this is a common occurrence. Bile salts serve to keep cholesterol in solution, and hence if there is any significant lowering of the level of bile acids, cholesterol will be precipitated. Bile acids may pass through the mucosa of an inflamed gall bladder, thus decreasing the concentration and interfering with the bile salt-cholesterol ratio to cause precipitation of cholesterol. The cholesterol-bile salt ratio may also be increased in conditions which give a hypercholesteremia, such as in pregnancy, high fat diets, and in the premenstrual phase of the menstrual cycle. Overconcentration of the bile salts or other constituents of the bile, which may result from partial or complete obstruction of the cystic duct, exerts a damaging effect on the mucosa of the gall bladder. The obstruction may be due to a variety of factors, e.g., anomalies of the valves of Heister, of the cystic duct, adjacent adhesions, stone, etc. It has been shown that partial obstruction of the common duct will cause the formation of gallstones in the experimental animal, and there is some evidence that spasm or hypertrophy of the ampulla of Vater will cause enough resistance to the biliary flow to produce gallstones. It is also known that gall bladders will develop gallstones after choledochocenterostomy, presumably, in this case, due to bacterial action.



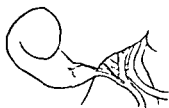
short large cystic duct



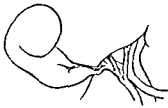
mobile common duct



cystic duct parallel to hepatic duct



accessory hepatic duct

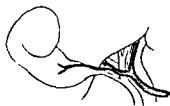


cystic duct joins R hepatic duct

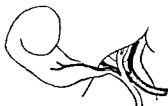


large pendulous Hartmann's pouch

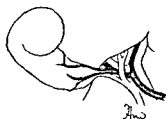
Fig 302—Variations in the position of the extrahepatic bile ducts. In operations in this area extra care must be taken to identify each structure prior to its ligation or section



R hepatic a.
anterior to
hepatic duct



R hepatic a.
lying close to
cystic duct

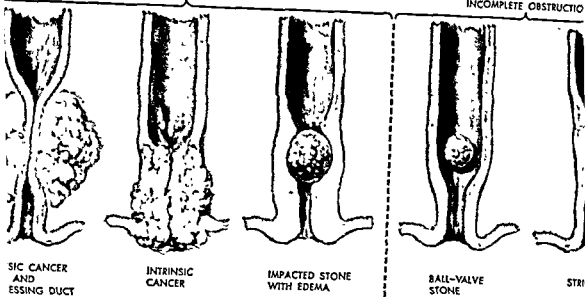


two cystic arteries

Fig 303—Variations in the position of right hepatic and cystic arteries. Ligation of the right hepatic artery may occur when anomalies are present. This accident, although no longer believed lethal, should never occur.

COMPLETE OBSTRUCTION

INCOMPLETE OBSTRUCTION



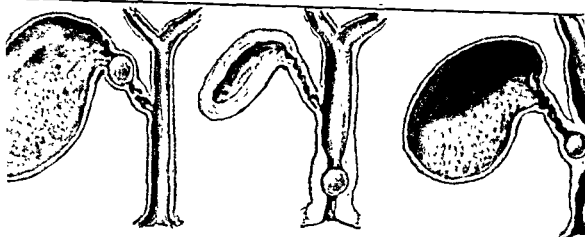
SIC CANCER
AND
ESSING DUCT

INTRINSIC
CANCR

IMPACTED STONE
WITH EDEMA

BALL-VALVE
STONE

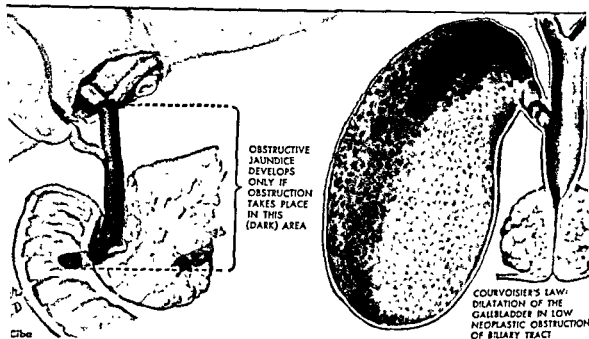
STRICTURE



STRICT DUCT OBSTRUCTION
DROPS

COMMON DUCT OBSTRUCTION:
JAUNDICE

TERMINAL CYSTIC DUCT OBSTRUCTION:
HYDROPS AND JAUNDICE



OBSTRUCTIVE
JAUNDICE
DEVELOPS
ONLY IF
OBSTRUCTION
TAKES PLACE
IN THIS
(DARK) AREA

COURVOISIER'S LAW:
DILATATION OF THE
GALLBLADDER IN LOW
NEOPLASTIC OBSTRUCTION
OF BILIARY TRACT

Plate 27.—Extrahepatic Biliary Obstruction.



Fig 304.—Calcified gall bladder.



Fig 305 —Cholecystograms A, Functioning gall bladder containing radiotranslucent (cholesterol) stones B, Multiple radiopaque calculi C, Functioning gall bladder with typical calcium ovalate gallstones D, Laminated gallstone.

The damaged mucosa is particularly liable to invasion by pathogenic bacteria, chiefly staphylococci, *Escherichia coli*, streptococci, and occasionally *Salmonella typhi*.

Chemical analysis reveals that *gallstones* are derived from the normal chemical constituents of the bile and are formed almost exclusively in the gall bladder. Cholesterol, calcium bilirubinate, and calcium carbonate are the principal stone-forming substances. A stone may be composed almost entirely of one of these compounds, e.g., pure cholesterol stone or a mixture of two or three.

Gallstones are likely to form when any one of the stone-forming constituents is in excess in the bile. Pigment stones composed of calcium bilirubinate are formed because of an increased excretion of bilirubin such as occurs in hemolytic jaundice. Cholesterol stones occur when there is a change in cholesterol metabolism. There is a hypercholesteremia in pregnancy which may account for the increased incidence of cholelithiasis in multiparous women. Stones also form when gall bladder function is so altered that the solvents (bile acids) are absorbed faster than the stone-forming constituents, and this leads to precipitation and crystallization. The pH of the bile may be of some importance, as an increased alkalinity tends to precipitate calcium carbonate. It seems probable, therefore, that no one mechanism can explain the occurrence of the different types of human gallstones.

Biliary tract disease is frequently associated with pancreatitis and appears to be more common in patients with diabetes mellitus.

DISEASES OF THE GALL BLADDER

Acute Cholecystitis

Etiology.—Acute cholecystitis is dependent in the great majority of cases upon an obstruction to the outlet of the gall bladder. In most instances this is due to a calculus impacted in the neck of the gall bladder or in the cystic duct. The obstruction blocks the outflow of bile and causes an accumulation of the normal secretions within the gall bladder. The resulting distention plus the edema at the site of obstruction impairs the arterial supply and the venous and lymphatic drainage of

the gall bladder. The resulting ischemia leads to necrosis and ulceration, which may go on to gangrene and perforation.

An overconcentration of bile salts is capable of injuring normal gall bladder tissue. This may be an added etiologic factor. Such an injured gall bladder wall is particularly liable to secondary bacterial invasion by *Streptococcus hemolyticus*.

Pathology.—The pathologic picture varies from mild inflammation to ischemia and gangrene of the gall bladder wall. In the typical case the wall of the gall bladder is red, thickened, and edematous. The serosal surface is congested and is often covered with fibrin. The mucosa is bright red. If the obstruction to the cystic duct is complete, the gall bladder is distended with what appears to be purulent fluid—the so-called *empyema of the gall bladder*. This fluid may be a sterile emulsion of cholesterol, mucus, and calcium carbonate, but true pus may be present.

Edema is responsible for most of the thickening of the wall. The inflammatory exudate is most marked in the outer layers and is characterized by a relatively small number of polymorphonuclear cells. A hemorrhagic exudate is a prominent feature. Not infrequently there is an associated pancreatitis.

A considerable number of acutely inflamed gall bladders yield positive bacterial cultures. The common organisms found are *E. coli*, streptococcus and staphylococcus, and gram-negative bacilli.

Clinical Picture.—There is frequently a history suggestive of previous gall bladder disease. The onset of symptoms in the acute attack is usually sudden, with pain in the right hypochondrium, reaching its peak in 24 hours. The pain may radiate to the right scapular region and to the right shoulder. Nausea and vomiting are common. Tenderness over the gall bladder is constant and is associated with muscle spasm. The gall bladder is usually palpable despite this muscle resistance. Fever is common and is accompanied by tachycardia and leukocytosis. Jaundice may occur occasionally if there is an associated hepatitis, cholangitis, or common duct obstruction due to stone, inflammatory edema, or pressure on the common duct from swelling of Hartmann's pouch.

The acute symptoms usually subside in 2-3 days, but tenderness, rigidity, and a palpable mass are likely to persist for a considerable period. However, the improvement in the clinical picture is often misleading and cannot be directly correlated with a corresponding improvement in the disease process. The condition may go on to abscess formation and even gangrene.

Differential Diagnosis.—Acute cholecystitis must be differentiated from perforated or penetrating peptic ulcer, acute appendicitis, acute pancreatitis, and coronary thrombosis.

With perforated peptic ulcer, there is often a previous history of epigastric pain, which was relieved by food and alkalis. The tenderness is acute and the rigidity is boardlike; both are more generalized, and there is usually a loss of liver dullness; an x-ray of the abdomen may show air in the peritoneal cavity. The differentiation from acute pancreatitis is difficult. The pain and tenderness in pancreatitis are more diffuse, tend to radiate to the left lumbar region, and the vomiting is more severe. An elevated serum amylase is of diagnostic significance.

In acute appendicitis the location of maximum pain and tenderness is in the right iliac fossa or loin and is associated with rectal tenderness. In the high-lying retrocecal appendix the differentiation may be impossible. In coronary thrombosis the pain frequently radiates down the left arm. There is hypotension with typical electrocardiographic changes. However, similar changes in the electrocardiogram may result from an attack of acute cholecystitis.

Treatment.—Operative treatment is indicated for all cases of acute cholecystitis, although there is wide divergence of opinion as to the optimum time for operation. If the patient is seen early, the gall bladder should be removed. This eliminates the source of infection and risk of perforation, gives a smoother convalescence, avoids the need for future operation, and can be performed without difficulty. If the patient is seen late when the symptoms are subsiding, conservative measures are preferable as they carry the least risk. Operation should not be delayed if there is increased pain, tenderness, and rigidity, and

if the pulse rate, temperature, and leukocyte count do not quickly return to normal.

Cholecystostomy is indicated (1) when the patient's general condition is so serious that only the simplest and quickest operation can be considered, and (2) in patients with edema or adhesions about the common duct of such a nature that a cholecystectomy is particularly difficult.

Adequate antibiotic therapy should be used in all cases. Dehydration is not uncommon, and this necessitates adequate fluid prior to operation. The glycogen and protein reserves of the liver should be replenished by the parenteral administration of the substances.

If the early treatment has been of a conservative nature, cholecystectomy should be performed at a later date when the acute process has completely subsided.

Chronic Cholecystitis and Cholelithiasis

Etiology.—In the section on pathogenesis of gall bladder disease it has been pointed out that *cholecystitis is the result of chemical irritation, metabolic disturbance, or enzyme action*. Infection, if present, is probably a secondary factor. Occasionally chronic cholecystitis may manifest itself after an acute attack.

Pathology.—The healthy gall bladder is translucent, and one of the earliest signs of pathologic change is the development of opacity. In the later stages of cholecystitis, the wall of the gall bladder becomes thickened and opaque. Inflammatory adhesions frequently develop between the gall bladder and adjacent viscera. The microscopic appearance may be one of fibrosis or chronic cellular inflammatory changes. Gallstones cause or largely contribute to the pathologic change in the gall bladder by predisposing to biliary stasis, by trauma to the mucosa, and by cystic duct obstruction.

Hydrops of the gall bladder results if the cystic duct becomes obstructed in the absence of acute inflammation. The bile pigments and salts are gradually absorbed and are replaced by a watery secretion, white bile, from the mucosal surface. The gall bladder may become very distended and may contain as much as 500 ml. of fluid.

Cholesterosis of the gall bladder is the result of a local or general change in cholesterol metabolism, characterized by the deposition of large amounts of cholesterol or cholesterol esters in the mucous membrane. The resulting small yellow specks, like strawberry seeds, have given the condition the name of "strawberry gall bladder." Stones may be present and are frequently of the pure cholesterol type. The symptoms here are similar to those in other forms of gall bladder disease.

of gallstones appears to increase with age, many patients with stones apparently are asymptomatic or exhibit only minor symptoms.

The symptoms of gall bladder disease are those of gastrointestinal upset and pain. The gastrointestinal symptoms are primarily due to pylorospasm and spasm of the sphincter of Oddi with resultant delay in the emptying of the stomach and biliary tract. This results in epigastric fullness and postprandial distress which is relieved by belching. Many types of

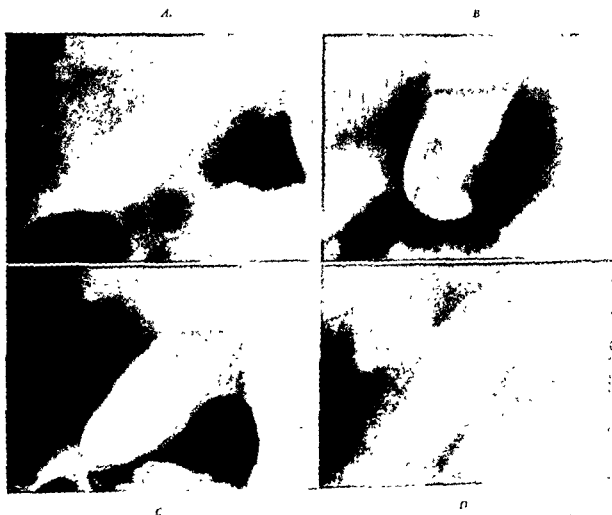


Fig. 306--Cholecystograms showing a functioning gall bladder with gallstones. Note that the gallstones are not visible in the supine film. Contraction of the gall bladder following a fatty meal pushes the gallstones toward the cystic duct. It can be readily seen how one or several of the gallstones can enter the biliary ductal system, causing obstruction.

A, Supine before fat
B, Upright before fat

C, Fifteen minutes after fat
D, Thirty minutes after fat

Clinical Picture.—Cholecystitis and cholelithiasis most commonly occur in the obese middle-aged female but are also found in males and even in children. As the incidence

of food may cause distress, but it is especially true for those of a fatty nature.

The pain may be mild and limited to a dull right upper quadrant ache or severe with radi-

tion to the right scapular region, the right shoulder tip, and the right flank. Most patients with chronic disease of the gall bladder will have one or more attacks of biliary colic during the course of the disease. In its typical form this is pathognomonic of biliary tract disorders. This pain is due to distention of the gall bladder and to peritoneal irritation. It begins suddenly with the usual radiation, is steady, and slowly progresses in intensity. There is usually a history of a mild epigastric ache which precedes the pain due to distention of the gall bladder, and the pain is only felt in the gall bladder area when the distended viscus comes in contact with the parietal peritoneum. The attack is frequently accompanied by nausea and vomiting.

with radiologic confirmation by cholecystography. Tenderness over the gall bladder may be present. This tenderness may be increased if the patient is made to inspire during palpation (Murphy's sign). Fever and leukocytosis are uncommon.

Differential Diagnosis.—Chronic gall bladder disease must be differentiated from peptic ulcer, chronic pancreatitis, disease of the upper urinary tract, recurrent appendicitis, and coronary artery disease.

Treatment.—If it can be demonstrated that chronic gall bladder disease with or without stones exists, cholecystectomy is the treatment of choice. If, however, the evidence is equivocal, the surgeon should hesitate to operate. Symptoms such as dyspepsia and flatulence



A



B

Fig. 307.—A plain film of the abdomen showing a stone in the cystic duct and a quantity of radiopaque material in the fundus of the gall bladder. No dye has been used.

A, Supine B, Upright

Occasionally there is a definite relationship between angina pectoris and gall bladder disease. In such cases cholecystectomy will cure the symptoms of gall bladder disease, the simulated anginal pain, and will cause an improvement in the electrocardiograph.

Physical examination reveals few signs, and the diagnosis is made on a careful history

are prone to persist after operation unless the pathologic changes in the gall bladder are definite. The operation of cholecystostomy is rarely if ever indicated.

Silent Gallstones

The increased use of radiography as an adjunct to routine physical examination has

resulted in the demonstration of gallstones in patients who did not complain of symptoms referable to the biliary tract. These patients are often considered to have so-called silent gallstones in a functioning gall bladder. However, we feel that a careful interrogation will elicit a history of gall bladder disease.

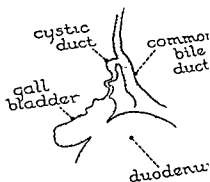
Many observers feel that nonoperative treatment, coupled with careful follow-up and repeated cholecystograms, is the treatment of choice. However, the risk of developing acute cholecystitis, acute pancreatitis, common duct stone, and conceivably carcinoma of the gall bladder is such that elective cholecystectomy should be advised in all patients of this type whose general physical condition does not increase the hazard of the operation.

spontaneous biliary fistulas follow an attack of acute cholecystitis. The gall bladder becomes adherent to the anterior abdominal wall, and the stoma develops by erosion through the muscle and skin. Postoperative external fistulas follow cholecystostomy, cholecystectomy, or operations on the common duct.

Clinical Picture.—In internal fistulas the symptoms are those of biliary tract disease. The stone may become impacted in the duodenum or terminal ileum and cause acute intestinal obstruction. Such a cause should be suspected in intestinal obstruction if air is seen in the biliary tract or a shadow of a stone in the scout film. Fever and jaundice may result from an associated cholangitis. Hematemesis and melena sometimes occur.



Fig. 308.—A plain film of the abdomen showing air in the gall bladder, duodenum, and common bile duct due to a fistula between the gall bladder and the duodenum.



Biliary Fistula

Etiology.—Biliary fistulas are uncommon. There are three principal types

- 1 Spontaneous internal
- 2 Spontaneous external
- 3 Postoperative external

Spontaneous fistulas, either internal or external, are associated in the majority of cases with gallstones and result from the erosion of a stone through the wall of the gall bladder, or common duct, generally into the duodenum, stomach, or colon. They may also follow penetrating peptic ulcer or carcinoma of the gall bladder, stomach, or pancreas. Most external

Spontaneous external fistulas occur near the umbilicus or in the right upper quadrant of the abdomen. The biliary tract should be investigated carefully in all patients who develop abscesses or sinuses in these areas. X-ray findings of air in the bile ducts or of barium in the biliary tract following a barium meal is strong evidence of biliary fistula.

Treatment.—In spontaneous fistulas and in those fistulas which follow cholecystostomy, the treatment is cholecystectomy, excision of the fistulous tract, and closure of the opening in the intestine. If intestinal obstruction is present, the offending stone should be removed. In fistulas following cholecystectomy

and operations on the common duct, the tract should be excised, any biliary obstruction relieved, and a T tube placed in the common duct.

Traumatic Rupture

Rupture of the gall bladder or ducts may occur following penetrating wounds or trauma to the abdominal wall. Adjacent abdominal viscera are frequently involved.

The clinical picture is one of initial shock, followed by a period of improvement. There is a dull pain in the upper abdomen, and within a short time the abdomen slowly distends with fluid. Jaundice due to absorption of bile from the peritoneal cavity comes on after a few days.

Treatment is usually cholecystectomy. If the tear is in the fundus, it may be sutured without drainage or a cholecystostomy may be performed. If the bile ducts are injured, they should be repaired over an indwelling T tube. Any bile found in the abdominal cavity should be sucked and even washed out. Chemotherapy should be instituted.

Bile Peritonitis

Sterile bile in the peritoneal cavity causes a chemical peritonitis which pursues a relatively benign course. However, as most cases follow operations on a diseased gall bladder, the bile is usually infected and the peritonitis while mild at first becomes progressively worse.

Etiology.—Bile peritonitis occurs following either spontaneous or traumatic rupture of the gall bladder or bile ducts or after an operative procedure.

Treatment.—Early operation is indicated. The bile in the peritoneal cavity should be aspirated, tears repaired, and a drain placed alongside the common duct so that any future leak may be aspirated. Most cases of bile peritonitis can be avoided by meticulous surgery, and drains should always be placed down to the operative site so that any bile leakage will drain to the exterior.

Tumors of the Gall Bladder

Benign

Benign tumors of the gall bladder, adenomas, lipoid polyps, and papillomas, are usu-

ally asymptomatic. True papillomas are extremely rare, and most recorded papillomas are merely polypoid swellings of the mucosa due to cholecystitis glandularis proliferans. The distinction between the true tumors and the pseudopolyps is almost impossible and of little practical importance. Both should be removed, as the true papilloma is precancerous and the pseudopolyp is an indication of chronic gall bladder disease.

Malignant

Carcinoma is, for all practical purposes, the only malignant tumor of the gall bladder. Sarcoma, melanoma, and endothelioma occur but are extremely rare.

Incidence.—Primary carcinoma of the gall bladder is an uncommon tumor of the digestive tract. It occurs predominantly in females in a ratio of 4:1. This ratio parallels the incidence of cholelithiasis in the two sexes.

Etiology.—The presence of papillomas and calculi appears to be related to the development of carcinoma. While papillomas have the capacity to undergo malignant change, this transformation rarely occurs in the gall bladder. The relationship of cholelithiasis to a carcinoma of the gall bladder is interesting and probably important, as the great majority of cases show the presence of stones prior to the development of the neoplasm.

Pathology.—Carcinoma of the gall bladder is divided into two main groups: adenocarcinoma and squamous cell carcinoma. The majority of cases are varieties of adenocarcinoma, papillary, mucoid, and scirrhous. The wall of the gall bladder is thick, hard, and contracted, and at operation the condition may be mistaken for chronic cholecystitis. The tumor metastasizes early with direct spread to the liver, to adjacent organs, or along the bile passages. Distant spread may occur to lungs, bones, and other systemic organs.

Clinical Picture.—The preoperative diagnosis of carcinoma of the gall bladder is seldom made. The symptoms are usually those of long-standing biliary tract disease, although with the onset of malignancy they become more severe. Pain is a prominent feature. Occasionally the onset is insidious, and the diag-

nosis is made only when metastases develop. Jaundice may occur from hepatic involvement or from blockage of the ducts. A palpable mass in the gall bladder region is a late sign. Acute perforation may occasionally occur.

Differential Diagnosis.—It is seldom possible to differentiate carcinoma of the gall bladder from chronic cholecystitis. Cholecystography is of little help; it merely demonstrates gall bladder dysfunction of varying degree. The advanced case must be differentiated from other neoplasms in the upper abdomen.

Treatment.—Cholecystectomy offers the only hope of cure. This procedure is frequently impossible because of the extension of the disease. The removal of the calculous gall bladder may be of value in the prevention of malignant change.

DISEASES OF THE BILE DUCTS

The clinical manifestations of biliary duct disease are the result of either (1) obstruction, (2) infection, or (3) functional motor disturbances.

OBSTRUCTION

The cause of the obstruction may be (a) in the wall (congenital stricture, choledochus cyst, acquired stricture, inflammatory edema) (see also Chapter 30), (b) in the lumen (stone, tumor), or (c) outside the duct (chronic pancreatitis, carcinoma of the pancreas).

Congenital Anomalies

Congenital Cystic Dilatation of the Common Duct (Choledochus Cyst)

Choledochus cyst is a rare anomaly characterized by a localized dilatation of the common duct and probably due to a congenital weakness of the wall, to which has been added an obstructive factor, such as stenosis, angulation, inflammation, or achalasia.

Pathology.—The cyst is retroperitoneal and arises from the supraduodenal portion of the common duct. Its wall consists of dense fibrous tissue without a lining epithelium. The cyst varies greatly in size, may have a capacity of several liters, and contains bile-stained fluid.

Clinical Picture.—Choledochus cyst occurs predominantly in the female. It rarely produces symptoms before the age of 6 months. The clinical features are intermittent jaundice, colicky right upper abdominal pain, and a cystic swelling in the right hypochondrium. Clay-colored stools occur.

Treatment.—The treatment of choice is anastomosis between the cyst and duodenum. If this is not feasible, a cholecystoduodenostomy may be performed.

Congenital Atresia of the Bile Ducts

The obliterative process may involve the bile channels within the liver, the hepatic ducts, the cystic duct, the common duct, or the gall bladder. The involved portions may be completely absent or may be represented by cords of fibrous tissue. There is an associated widespread biliary cirrhosis.

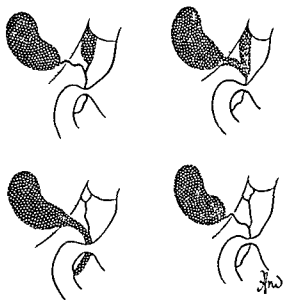


Fig. 309—Degrees and locations of congenital atresia of the extrahepatic bile ducts.

Clinical Picture.—Jaundice is present from birth and progressively deepens for the duration of life. As the obstruction is complete, the stools are clay colored from birth. The abdomen may appear prominent due to enlargement of the liver and spleen. Ascites is present in late cases. In the untreated patient, death inevitably occurs but only after many months.

Differential Diagnosis.—The condition must be differentiated from other causes of jaundice in infancy, principally icterus neonatorum and erythroblastosis fetalis.

Treatment.—The treatment consists of an anastomosis between the proximal patent portion of the biliary system and the duodenum, although in many cases this is not possible because of the type or extent of the atresia. As in all jaundiced patients, care must be taken preoperatively to control the bleeding tendency with adequate amounts of vitamin K and transfused whole blood.

Inspissated Bile or Mucus

These may plug the biliary ducts and give a picture that is indistinguishable from congenital atresia of the bile ducts.

Treatment.—Laparotomy with irrigation of the duct system will usually dislodge the obstructing plug and cure the condition.

Acquired Anomalies

Cholelithiasis

Approximately 15% of patients who undergo laparotomy for cholelithiasis have one or more stones in the common duct. These stones usually originate in a diseased gall bladder but occasionally develop within the ductal system. They may remain in the ducts or pass through the ampulla of Vater without producing symptoms. However, if they become impacted, symptoms of biliary tract obstruction occur.

Pathology.—Obstructing stones are usually single, but once obstruction occurs, secondary stones and gravel accumulate. The degree of dilatation of the ductal system depends upon the duration and completeness of the obstruction and whether it is intermittent or continuous. Mucosal ulceration occurs at the site of an impacted stone and may result in stricture or, more rarely, perforation. Inflammatory changes occur in the duct walls which may go on to thickening and fibrosis.

The effects on the liver depend upon the duration of the obstruction. If this is of long standing, varying degrees of hepatocellular damage occur, leading to derangement of liver function.

Clinical Picture.—The history of intermittent, colicky, sometimes severe, right upper quadrant pain, intermittent fever, and fluctuating jaundice is characteristic of biliary duct stone. The ball valve action of the stone is responsible for the intermittent character of the symptoms. Occasionally the obstruction is complete, leading to a steadily deepening jaundice. The pain is more severe than that associated with acute cholecystitis and frequently radiates through to the right scapular region and the right shoulder. It may radiate to the left hypochondrium and epigastrium. If the obstruction is complete, the pain is continuous and steady. The fever characteristically is of the intermittent type (Charcot's intermittent biliary fever), accompanies an exacerbation of the pain, and is commonly associated with a chill. There may be an associated *E. coli* septicemia. The jaundice varies in degree and usually fluctuates, although it may be steadily progressive if the stone is firmly impacted. The urine contains large amounts of urobilinogen and bile pigments, while the stool is characteristically pale. The biochemical findings are typical of obstructive jaundice, namely, an elevation of the serum bilirubin, an elevated alkaline phosphatase, and normal flocculation tests.

A plain x-ray film of the abdomen may show the presence of stones. A cholecystogram cannot be successfully carried out in the presence of jaundice.

If the stone does not cause obstruction, the symptoms will be simply those of the associated gall bladder disease without jaundice.

Differential Diagnosis.—The disease must be differentiated from the following.

- 1 Acute cholecystitis, acute pancreatitis, perforated peptic ulcer, renal colic, and coronary thrombosis.

- 2 Other causes of obstructive jaundice such as carcinoma of the head of the pancreas and chronic pancreatitis.

- 3 Intrahepatic jaundice.

Treatment.—While antispasmodics, e.g., atropine, may bring relief in the acute attack, the treatment of ductal stone is surgical. The preoperative preparation is of prime importance. The liver must be protected by a diet high in protein and carbohydrate. The bleed-

BILIARY SYSTEM

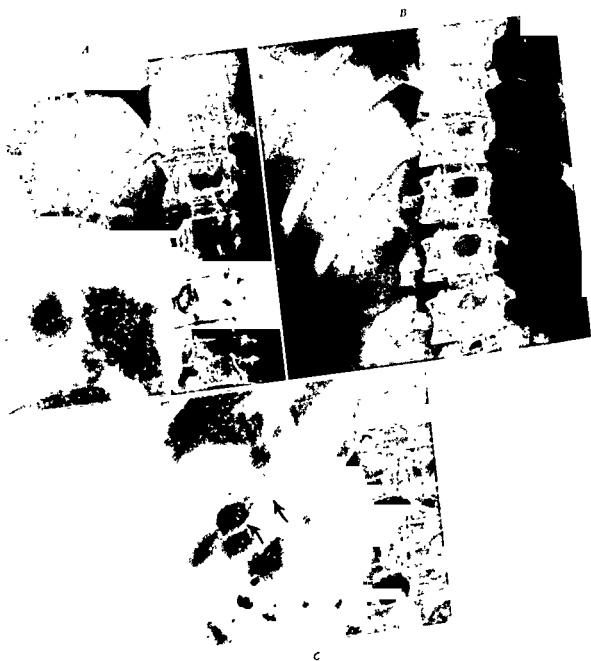


Fig 310—Intravenous cholangiograms
 A, Normal ductal system, postcholecystectomy
 B, Common duct stone
 C, Cystic duct remnant

ing tendency must be counteracted by parenteral vitamin K and whole blood transfusions. The biliary tract must be decompressed in order to relieve the jaundice and the back pressure on the liver. When the patient's condition permits, the ducts must be explored thoroughly and all stones and debris removed. The ampulla of Vater should be dilated carefully to ensure free passage of bile into the duodenum. The gall bladder, if present, should then be removed and the common duct closed over an indwelling T tube.



Fig 311—Cholangiogram through an indwelling common duct T tube, showing the hepatic ducts, the common hepatic duct, and the common bile duct. The distal end of the common bile duct is blocked by a calculus.

Prior to the removal of the T tube a cholangiogram must be made to ensure that no stones remain in the common duct and that the ampulla of Vater is patent.

Stricture

Etiology.—In the majority of cases, acquired stricture of the common duct is the result of injury during operations on the biliary tract. However, it is occasionally associated with ulceration of the duct due to gallstones, with cholangitis and periductal abscess, with the spread of infection from the pancreas, and with tumors.

Clinical Picture.—If jaundice follows immediately after an operation, the common bile duct has been ligated or there is a residual stone. If, however, an external biliary fistula occurs, one may assume that the duct has been partially or completely divided. If the duct has been only partially divided, late stricture will occur with subsequent jaundice and cholangitis.

Differential Diagnosis.—Postoperative stricture must be differentiated from a retained common duct stone. This is frequently impossible without operation. However, cholangiography is of value if a T tube remains in the common duct.

Treatment.—Injury to the common duct can be prevented. Anomalies of the ducts and vessels must be recognized, and all structures must be carefully visualized before any ligation or division is carried out. The treatment of acquired stricture is a most difficult surgical procedure. An effort must be made to reconstruct the ducts and re-establish continuity, and a mucosa-to-mucosa junction should be obtained whenever possible in order to prevent the occurrence of another stricture. If the gall bladder is present and functioning and the stricture is distal to the entrance of the cystic duct, an anastomosis between the gall bladder and the intestinal tract may be satisfactory.

Tumors of the Bile Ducts

Tumors of the extrahepatic biliary tract are uncommon. They may arise anywhere in the ducts, but the commonest site is the ampulla of Vater.

Pathology.—Benign growths are exceedingly rare. Carcinoma is the important tumor. It is usually a diffusely infiltrating adenocarcinoma. In contrast to neoplasm of the gall bladder, these tumors of the ducts are rarely associated with stone.

Clinical Picture.—The onset is insidious with jaundice as the first symptom. The jaundice is obstructive in type. Dull upper abdominal pain resulting from raised intraductal pressure may accompany the icterus. The growth may ulcerate and produce gross or microscopic blood in the stool and a marked secondary anemia. The symptoms tend to be steadily

progressive in contrast to those associated with stone in the common duct.

Physical findings aside from the jaundice are not remarkable. However, if the tumor lies in the lower end of the common duct distal to the cystic duct, the gall bladder will become distended and palpable.

Differential Diagnosis.—Carcinoma of the head of the pancreas, chronic pancreatitis with obstruction, and common duct stone give a similar picture.

Treatment.—In the case of a malignant tumor, a resection of the duodenum, head of the pancreas, and the involved portion of the duct should be carried out. Local resection may be done for benign growths and malignant tumors that are confined to the ampulla. In the majority of cases local spread makes these procedures impossible, and a palliative operation, such as a cholecystojejunostomy, must be done to relieve the jaundice.

INFECTION

Cholangitis

Etiology.—Cholangitis is an inflammatory process which involves the walls of the biliary passages and is usually associated with some obstruction to the free flow of bile. The obstruction is usually the result of stone or stricture of the common duct which predisposes to bacterial invasion. However, it may result from regurgitation of intestinal contents into the common duct.

Pathology.—The degree of cholangitis varies from a simple catarrhal inflammation to frank suppuration with resultant fibrosis and thickening. The inflammatory process may spread through the wall to involve the periductal tissues or extend upward into the tributaries of the bile ducts to involve the liver parenchyma. It may extend until all liver ducts are filled with pus.

Clinical Picture.—Acute suppurative cholangitis presents a picture of repeated episodes of chills, fever, and deepening jaundice. The liver is usually enlarged and tender. The more chronic forms do not present a definite clinical picture, although they tend to flare up in the presence of obstruction, leading to further damage to the liver and ductal system.

Treatment.—The treatment is mainly prophylactic, with relief of biliary tract obstruction. In the acute suppurative phase, the treatment is drainage of the common duct and adequate doses of antibiotics.

FUNCTIONAL MOTOR DISTURBANCES

Biliary Dyskinesia

Biliary dyskinesia is an obstruction to the flow of bile without jaundice, due to a neuromuscular dysfunction of the sphincter of Oddi. Such a disturbance is rare in the absence of other biliary tract symptoms, in whom no evidence of organic disease can be demonstrated, the possibility of dyskinesia should be kept in mind.

Treatment.—The encouragement of bile flow by diet, bile salts, and antispasmodics may bring relief. Dilatation or cutting of the sphincter of Oddi has been reported to be of value.

Postcholecystectomy Syndrome

The symptoms of biliary tract disease may persist or recur following cholecystectomy or choledochostomy. Most commonly this occurs following removal of a noncalculous gall bladder and is found in almost inverse proportion to the degree of pathologic change in the gall bladder wall. Symptoms, however, can recur after cholecystectomy in properly selected cases and are usually due to residual disease. In such cases retained cystic or common duct stone, infection in the cystic duct stump, and neuromas of the periductal nerve plexus are probable etiologic factors. Unfortunately, only too frequently, abdominal exploration reveals no abnormality.

Treatment.—Elimination of the residual organic disease by removal of stones, amputation of the remains of the cystic duct, periductal stripping of the nerve plexus may give relief.

OPERATIVE PROCEDURES

Cholecystostomy

The indications for cholecystostomy are limited and include the following:

1. In acute cholecystitis in the poor operative risk patient or when cholecystectomy presents unusual technical difficulties

2. In certain cases of acute necrosing pancreatitis

3. As a first-stage procedure in certain cases of jaundice due to carcinoma of the head of the pancreas or to chronic pancreatitis

Anesthesia.—Either local, regional, or general anesthesia may be employed

Operation.—A high paramedian or a right subcostal incision is used. Care must be taken

to avoid spilling infected bile into the peritoneal cavity, and the area should be packed off before proceeding. When the gall bladder is exposed, its contents are aspirated with a wide bore needle and syringe or with a trochar and suction. The fundus of the gall bladder should be supported with two Allis forceps or sutures and an incision made of sufficient length to permit entrance of the special scoops which are used to remove the stones. Care should be taken to remove all the stones present and ensure patency of the cystic duct.

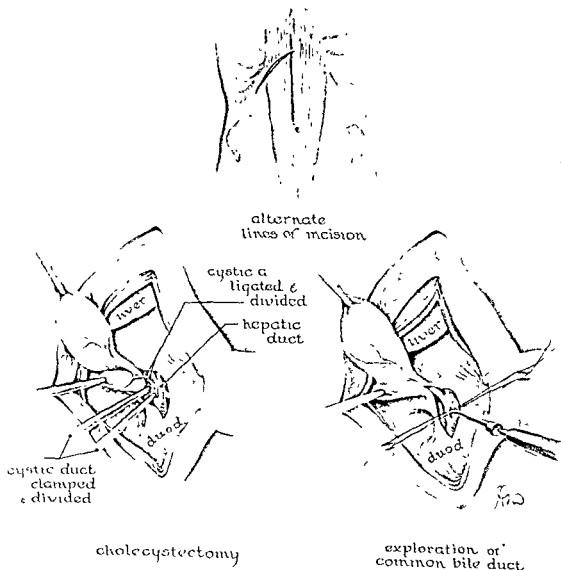


Fig 312—Technique of cholecystectomy and choledochostomy

A good-sized rubber tube is passed into the gall bladder and securely anchored. The opening of the gall bladder is closed about the tube, which is then brought out through the abdominal incision and connected to a receptacle.

Complications.—Approximately 40% of the patients having a cholecystostomy for gall bladder disease will have recurrent symptoms which require future operation

Cholecystectomy

Indications for cholecystectomy include the following

- 1 Cholecystitis and cholelithiasis
- 2 Traumatic rupture of the gall bladder
- 3 Tumors of the gall bladder
- 4 Internal or external biliary fistulas

Anesthesia.—Adequate relaxation is a prerequisite to good biliary surgery. Either spinal or inhalation anesthesia is used.

Operation.—The commonly used incisions are right paramedian or right subcostal.

The gall bladder may be removed (1) by starting at the fundus and dissecting down to the cystic artery and cystic duct or (2) by preliminary ligation of the artery and duct with the subsequent removal of the gall bladder. The latter procedure is usually preferable, and the former should be reserved for those cases where a large stone or adhesions tend to make clear visualization of the duct system difficult.

The fundus of the gall bladder is grasped with a pair of nontoothed artery forceps and drawn up over the edge of the liver. A second forceps is placed on the gall bladder in the region of Hartmann's pouch. When moderate traction is exerted on these forceps, the cystic duct is placed on the stretch. A small incision is made in the peritoneum over the neck of the gall bladder, and by careful dissection the whole length of the cystic duct is exposed. The next step is to display the cystic artery which lies above and slightly posterior to the duct. The various anomalies must be kept in mind, and great care must be taken to display and identify each structure before ligation or division.

When the dissection is complete, the artery is divided between ligatures. The cystic duct

is then divided between forceps $\frac{1}{8}$ " from the common duct and doubly ligated. Care must be taken to avoid inclusion of the common duct in the ligature. The gall bladder is then removed from the liver bed, preserving sufficient peritoneum to cover the raw area on the liver. Soft rubber drains should always be placed in the gall bladder fossa and should not be removed for several days.

Choledochostomy

The indications for choledochostomy include the following:

1. The presence of jaundice or a previous history of icterus
2. Dilated or thickened common duct
3. Palpable stones in the common duct
4. Small stones in the gall bladder with a patent cystic duct
5. In some cases of acute pancreatitis and in those cases of chronic pancreatitis causing common duct obstruction

Operation.—The cystic and common duct are placed on the stretch by traction on the gall bladder. The common duct is isolated, aspirated, and the character of the bile noted. Two stay sutures of fine silk are placed on its anterior surface, and a small incision is made in the duct parallel to its long axis between the two stay sutures. The duct should be irrigated to remove all sand and small stones. A probe is then passed down the duct into the duodenum to ascertain that the ampulla is patent. The ampulla should be dilated with graduated probes. The proximal portions of the ducts must be explored and irrigated to obviate leaving small stones. A T tube is then inserted into the common duct, sutured in place, and the long limb is brought out through the incision and securely fixed to the abdominal wall. The operation is completed by cholecystectomy. Occasionally the stone is so impacted in the ampulla that the duodenum must be opened and the ampulla of Vater sectioned to release it.

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Diseases of the Gall Bladder (A photographic supplement for clinics or conferences) (1949) (By Hilger P. Jenkins, M.D., Rudolph Janda, M.D., and Douglas Packard, M.D., Chicago)	19 min	Silent	American Cyanamid Co. Surgical Products Division Danbury, Conn.
Cholecystectomy for Acute Cholecystitis With Cholelithiasis (Shows surgical removal of a tensely distended, acutely inflamed and partially gangrenous gall bladder containing a single stone obstructing the cystic duct at its junction with the ampulla) (1952) (By Frank Glenn, M.D., New York)	23 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.
Anomalies of the Bile Ducts and Blood Vessels (Part I); Structures of the Common Duct (Part II) (1947) (Directed by Warren H. Cole, M.D., Chicago)	45 min	Silent Color	Ethicon, Inc. New Brunswick, N. J.
Technic for Cholecystectomy (Depicts a standard operative technique in a typical case in which a single combination-type cholesterol stone was removed) (1953) (By John L. Madden, M.D., New York)	24 min	Sound Color	Sturgis Grant Productions, Inc. 322 E. 44th St. New York 17, N. Y.

Chapter 21

Pancreas

Harry S. Dolan, MD, and James F. Hopkirk, MD.

ANATOMY

The pancreas is a retroperitoneal gland which lies in the upper part of the abdomen at the level of the bodies of the 1st and 2nd lumbar vertebrae. It extends from the duodenum on the right to the spleen on the left and is divided into four parts: head, neck, body, and tail. The head lies in the concavity of the duodenum, and its posterior relations are the inferior vena cava and tributaries of the portal vein. The posterior relations of the body are the aorta, superior mesenteric artery, left renal vein, left adrenal gland, and left kidney. The anterior relations of the entire gland are the stomach and the lesser peritoneal sac. The transverse colon is an inferior relation of the pancreas.

The main pancreatic duct (of Wirsung) traverses the complete length of the gland from the tail to the head and usually opens into the duodenum through the ampulla of Vater. In 70% of cases it joins the common bile duct at the ampulla. Occasionally the pancreatic duct joins the common bile duct some distance from the duodenum, or the two ducts open separately into the duodenum. The accessory pancreatic duct (Santorini) enters into the duodenum 2 cm. above the ampulla and rarely joins the common duct.

The blood supply of the pancreas is derived from the superior and inferior pancreaticoduodenal arteries, the splenic artery, and branches from the hepatic artery. The lym-

phatic distribution is rich and drains into the pancreaticoduodenal, preaortic, and celiac nodes. There is also some lymphatic connection between the pancreas, gall bladder, stomach, and spleen.

The nerve supply of the pancreas is via the vagus and sympathetic systems. The vagus is the secretory nerve of the pancreas and controls one phase of its external secretion. The sympathetic nerves (splanchnics) carry the afferent pain fibers.

HISTOLOGY

There are two different types of cells: i.e., those concerned with the external secretion which are very numerous and are arranged in acini that empty into branches of the ductal system, and those concerned with the internal secretion which are relatively few in number and are arranged in clusters or islands known as the islets of Langerhans. These islets form but a small part of the pancreas as a whole and are chiefly found in the tail.

EMBRYOLOGY

The pancreas develops as two endodermal evaginations, dorsal and ventral, from that part of the primitive gut which goes to form the duodenum. The dorsal evagination makes up the body, tail, part of the head of the pancreas, and the accessory pancreatic duct. The remainder of the pancreatic head and the main pancreatic duct arise from the ventral evagination.

The common bile duct originates from the ventral evagination which accounts for the close association of this duct with the main pancreatic duct

PHYSIOLOGY

The pancreas secretes 1,000-2,000 ml./24 hours. It is alkaline in reaction with a pH of 8.4-8.6. The chief salts are sodium, potassium, calcium, and magnesium

The pancreas has two main functions:

1. The manufacture of an internal secretion (insulin), which is one of the main controlling factors in carbohydrate metabolism
2. The manufacture of an external secretion containing a number of digestive ferments,

the chief of which are lipase, trypsin, amylase, and, in addition, maltase, lactase, and rennin.

The insulin controls the level of the blood sugar in the body. The pancreas and the liver are the main means of control of the mobilization and utilization of glucose, and the formation of glycogen from carbohydrate sources. Secretion of insulin occurs after taking food, particularly that of a carbohydrate nature.

Removal of the pancreas results in diabetes, which is, however, less severe in its manifestations than true primary diabetes. An increase in the secretion of insulin results in hyperinsulinism which is found in certain diseases of the pancreas, such as adenoma or hyperplasia of the islet tissue.

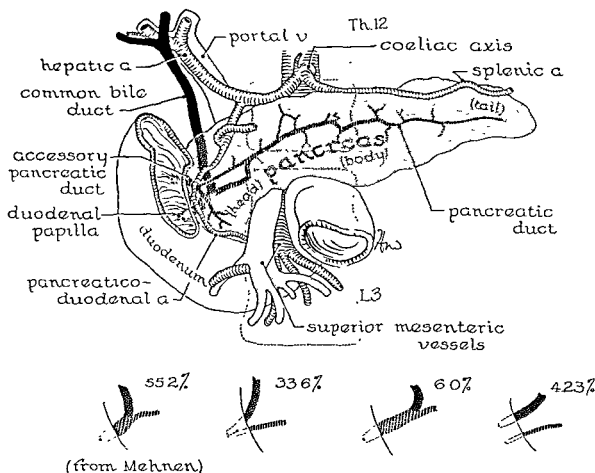


Fig 313—The normal anatomy of the region of the pancreas. Note the intraglandular portion of the common bile duct and how easily it may be occluded by disease in the pancreatic head to cause jaundice. Note, too, the variations in the common bile and pancreatic duct junctions. From an anatomic viewpoint the common channel theory of the etiology of acute pancreatitis is tenable.

The external secretion is stimulated by the vagus nerve, causing a secretion of a thick viscid juice which is rich in ferments, and by a hormone elaborated in the duodenum and upper jejunum, secretin, which passes via the blood stream to the pancreas, where it causes a copious watery secretion of low enzyme content, containing chiefly inorganic alkaline salts.

Secretin is produced by the action of hydrochloric acid and certain foodstuffs, chiefly those of a fatty nature. It is probable that the hormone causes its response through the mediation of the vagus nerve. In addition to its effect on the flow of pancreatic juice, it also increases the flow of bile and duodenal juice.

The action of the sympathetic system on pancreatic secretion is not as yet entirely defined, as either sympathetic stimulation or section will at times cause an increase in secretory flow.

A knowledge of the control of the internal and external secretions of the pancreas is most important when planning the therapy of patients with either acute or chronic pancreatic disease.

DEVELOPMENTAL ANOMALIES

The chief abnormalities encountered are variations of the pancreatic ductal systems and ectopic or aberrant pancreatic tissue which is found in the mesentery, omentum, gall bladder, biliary system, liver, and mediastinum.

Aberrant Pancreatic Tissue

Isolated masses of pancreatic tissue, which are completely separate from the main gland, occur in various parts of the gastrointestinal tract. Most commonly these are found in the region of the stomach, duodenum, and upper jejunum, and are located in the submucosa.

Gross Appearance.—Aberrant tissue is found as small nodules which measure 1-3 cm in diameter and have the color and appearance of normal pancreatic tissue.

Microscopically these have the characteristics of normal pancreatic tissue. They occasionally give rise to symptoms, the nature of which chiefly depends on their site, size, and physiologic activity. For example, when pancreatic tissue is found in the pyloric region of the

stomach, the symptoms may mimic duodenal ulcer or new growth.

Intussusception of the small bowel may result from the presence of aberrant pancreatic tissue in the bowel wall. Hyperinsulinism may be caused by aberrant tissue.

Treatment.—The treatment is local excision, and a careful search must be made in all the possible sites. Occasionally, resection of the bowel or stomach is necessary.

Annular Pancreas

This is a rare abnormality in which the head of the pancreas encircles the duodenum, causing duodenal obstruction of varying degree. The symptoms are those of chronic pyloric obstruction with gastric distention, nausea, and vomiting. The ideal treatment is division and resection of part of the head of the pancreas. In children this has resulted in fistula formation, and therefore duodenojejunostomy has become the accepted treatment. In some cases a gastroenterostomy or partial gastrectomy may be required.

LABORATORY AIDS IN DIAGNOSIS

Pancreatic Enzymes in the Blood.—The pancreatic enzymes, *amylase*, and *lipase*, are found in the blood serum of normal individuals. The normal serum amylase is 80-200 units (Somogyi), and lipase is 85-205 units. The blood amylase level is markedly raised in acute pancreatitis, particularly in the early stages of the disease, and it continues to remain high, usually for a period of 24-48 hours, when it generally returns to normal or subnormal levels, but it may remain elevated for several days. After that time has elapsed a normal serum amylase is of little diagnostic value in ruling out the presence of this condition. The serum lipase is also increased in acute pancreatitis; it, too, becomes elevated early, but it remains at a high level for several days longer than does the amylase. In our experience the serum amylase level has proved to give the more reliable diagnostic information. The serum amylase level of the blood should be estimated daily in any cases suspected of pancreatitis in order to note any transient elevation, should it occur. It should also be estimated daily in proved cases of acute

pancreatitis, as it helps to follow the course of the disease.

Pancreatic Enzymes in the Urine.—The levels of *urinary amylase* and *lipase* are usually increased in acute pancreatic disease, but the estimation of the urinary enzymes lacks much of the accuracy and specificity of the blood serum findings.

Pancreatic Enzymes in the Peritoneal Exudate.—The amylase level of the peritoneal exudate closely parallels that of the serum. Abnormal values, however, can sometimes be obtained somewhat earlier than in the case of the blood serum. A sample of peritoneal exudate is quite readily obtained by needle aspiration.

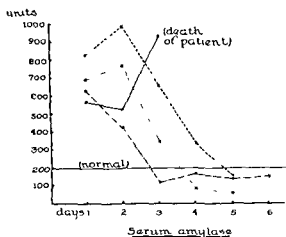


Fig 314—The serum amylase levels in four cases of acute pancreatitis. The drop to a normal range in three of the patients was coincident with an improvement in their clinical condition.

Serum Calcium.—The estimation of the blood *serum calcium level* is desirable in cases of acute pancreatitis, as the deposition of calcium, which forms, with fatty acids, the calcium soap of fat necrosis, causes a fall in the serum calcium. A serum calcium level below 7 mg/100 ml is indicative of a major degree of pancreatic damage and fat necrosis, and, for the most part, gives evidence of a very serious prognosis. The serum calcium should be followed daily during the course of the disease, so that measures can be taken to correct a calcium deficiency, should it occur.

Serum Bilirubin.—A slightly elevated *serum bilirubin* is not uncommon during the early

phases of acute pancreatitis. However, clinical jaundice is unusual.

Blood Sugar.—*Hyperglycemia* is frequently found in acute hemorrhagic and acute edematous pancreatitis. This derangement in carbohydrate metabolism is usually transient in nature and is found early in the acute phase of the disease. However, it may persist following recovery, particularly in those cases in

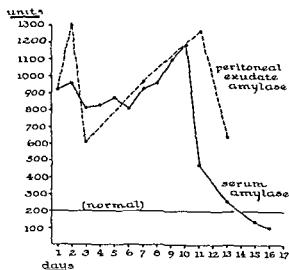


Fig 315—Graph illustrating the comparative levels of amylase in the peritoneal exudate and serum in a case of acute pancreatitis.

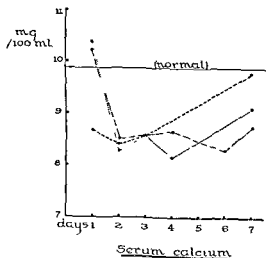


Fig 316—The serum calcium levels in four cases of acute pancreatitis. The values tended to become normal in 6-7 days following the onset of the disease. No extra calcium was administered in these cases.

which marked pancreatic destruction has occurred. It is estimated that 10-15% of patients who recover from severe pancreatitis will be left with an impairment of their carbohydrate metabolism. As in the case of the serum calcium, the blood sugar levels should be estimated daily and insulin should be administered if a high blood sugar persists.

Glycosuria commonly accompanies the hyperglycemia

Hematologic Findings.—The white blood cell count tends to be elevated in acute pancreatitis (15,000-18,000). This elevation is partly due to the pancreatic disease and partly due to the accompanying hemoconcentration, which so frequently occurs in this condition. Marked leukocytosis does not as a rule occur, except in the case of suppuration which may follow acute pancreatitis or pancreatic injury. If hemorrhage into the pancreas is severe, the

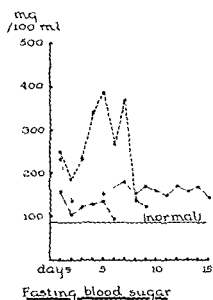


Fig 317.—The fasting blood sugar levels in three cases of acute pancreatitis. The values remained high for some days. No insulin was administered in these cases.

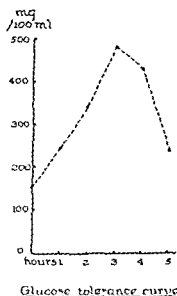


Fig 318.—The glucose tolerance curve of a patient who had marked hyperglycemia. This observation was taken after the patient had recovered from the acute phase of the disease.

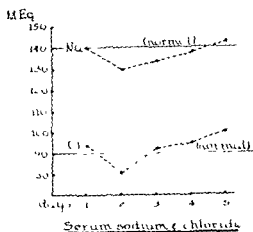


Fig 319.—The daily serum levels of sodium and chloride in a case of acute pancreatitis. The fall is not great, as this patient was treated with sodium chloride solutions, which undoubtedly prevented serious depletion of these ions.

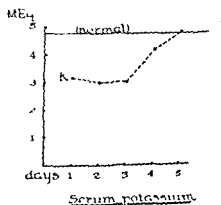


Fig 320.—The daily serum potassium levels in a case of acute pancreatitis. Note the return to a normal range following the administration of parenteral potassium.

red cell count and hemoglobin levels will fall; however, the hemoconcentration due to the accompanying shock usually masks these signs of anemia. The packed cell volume may give evidence of this anemia and is a more accurate and useful test than the estimation of the hemoglobin level.

Serum Electrolytes.—The prolonged and copious vomiting of acute pancreatitis leads to rapid dehydration of the patient. A low level of serum sodium, chloride, and potassium is commonly found in the severe case. The serum levels of these ions should be measured daily and even more frequently in the early and acute phase of the disease, so that any electrolyte deficit may be prevented or corrected.

Duodenal Drainage.—Examination of the aspirated duodenal contents for the presence of pancreatic enzymes, particularly following hormonal or vagal stimulation, will give information regarding the external secretion of the pancreas. The administration of secretin in a patient with pancreatitis should result in a decreased output of the enzymes amylase, lipase, and bicarbonate ion. In contradistinction to the decreased enzyme output of the pancreas, the flow of bile is increased by the secretin injection.

The site of pancreatic lesions in *jaundiced patients* may at times be localized by the use of secretin. If after the injection of secretin both the pancreatic and the bile responses are absent or reduced, this is evidence of obstruction of the biliary and pancreatic ducts which is due to a lesion in the head of the pancreas. If, on the other hand, the pancreatic flow is normal with a decreased biliary response, the lesion is in the extrahepatic biliary system and not in the pancreas. If the pancreatic and biliary flows are normal following secretin injection, the probability is that the cause of jaundice is intrahepatic or hepatocellular. Absence of these enzymes is indicative of decreased pancreatic function and occurs in such diseases as chronic pancreatitis. Cytologic studies of aspirated duodenal contents may help in the diagnosis of carcinoma of the pancreas.

Examination of the Stool.—This is of value in diagnosis of chronic pancreatic in-

sufficiency. In such cases the stools are bulky, pale, and contain large amounts of *undigested fat*, starch, protein derivatives, and carbohydrate, due to lack of pancreatic enzyme digestion. The measurement of undigested fat in the feces is carried out after the patient has been placed on a fixed high fat diet for 3 days (Schmidt diet). The normal values of undigested fat in the stool is 7.3-27.6% of dry weight, and anything over that amount is significant.

The stool fat content may also be measured by radioactive isotope studies, using fat labelled with I^{131} . The fecal excretion of I^{131} is higher in patients with pancreatic disease. Excretion can be reduced by the oral administration of pancreatin. Occult blood may occur in carcinoma.



Fig. 321.—Sentinel loop in a case of acute pancreatitis 8 hours after onset of symptoms.

Urine.—The estimation of urinary enzymes, bile, and sugar is helpful in the diagnosis of pancreatic disease and has been previously discussed.

X-ray Examination.—In *acute pancreatitis*, a plain film of the abdomen will frequently show isolated distended loops of small bowel, usually containing fluid levels and most commonly situated in the upper midportion of the abdomen. As the disease progresses, the x-ray of bowel characteristic of a paralytic ileus. examination shows many large distended loops

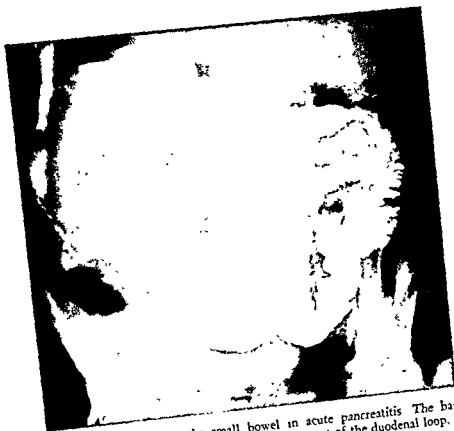


Fig 322—Gas and fluid levels in the small bowel in acute pancreatitis. The barium was administered in an attempt to show the enlargement of the duodenal loop.



Fig 323—Diffuse pancreatic lithiasis. Note the location of the pancreas at the level of L1 and L2.

There may be a haziness or obliteration of the psoas shadow, separation of the stomach from the colon by an opaque mass, and occasionally a pleural effusion.

Pancreatic lithiasis and *calcification* in the gland parenchyma can be seen in the plain film. This calcification lies at the level of the bodies of the 1st and 2nd lumbar vertebrae and is best demonstrated by an oblique rather than an anteroposterior view.

The *pancreatic ductal system* may be visualized during laparotomy by the injection of

the duodenum. The development of a pancreatic abscess or pseudocyst of the pancreas can also be demonstrated by this method.

The x-ray diagnosis of *carcinoma* of the pancreas is difficult. It is accomplished by the use of contrast media introduced into the stomach and duodenum. The following points should be observed in barium studies of the stomach and duodenum:

1. The duodenal folds may be flattened where the tumor presses on the duodenal wall. At times there may be gross evidence of extrinsic pressure



Fig. 324—Carcinoma of the head of the pancreas. Note the widening of the duodenal loop, the partial occlusion of the duodenum, and the upward displacement and compression of the stomach.

radiopaque media into the pancreatic ducts and by subsequent x-ray examination. Occasionally the pancreatic ductal system will be visualized during routine cholangiography.

A barium meal may give confirmatory diagnostic evidence of acute pancreatitis if the patient is not too ill for this examination. There may be upward displacement of the stomach, widening of the duodenal loop, and obliteration of the normal mucosal pattern of

2. The reverse or inverted 3 sign is seen in growth of the head of the pancreas in the region of the common bile and main pancreatic ducts.
3. There may be widening of the duodenal loop by an enlargement of the head of the pancreas.
4. Obstruction of the duodenum occasionally occurs.
5. Obstruction of the common bile duct may be visualized by the use of intravenous cholangiographic methods which will demonstrate a block in the distal portion of the common bile duct. The use of this method is not successful in the case of the jaundiced patient.

Pancreatic cysts may be demonstrated by x-ray by the marked displacement of the stomach and transverse colon that frequently occurs.

damage to other abdominal viscera. The management of associated pancreatic necrosis or fistula formation will be taken up under the individual sections.

Pancreatic Biopsy.—At times it is impossible to make a positive preoperative diagnosis of pancreatic disease. The main problem is that of the differentiation between chronic pancreatitis and carcinoma. Exploratory laparotomy and pancreatic biopsy is, under these circumstances, a necessary procedure. It is important to obtain a representative piece of pancreas, and care must be taken that tissue from deep within the gland substance be included in the sample as the surface cells of the pancreas may not reveal the nature of the pathologic change. Such a deep biopsy may be taken with relatively little risk of fistula development in cases of pancreatic fibrosis or carcinoma as there is very little functioning glandular tissue and there is marked ductal occlusion. It must, of course, be realized that the tissue interpretation is difficult and a negative report for cancer does not always exclude malignancy.

INJURIES OF THE PANCREAS

Serious injuries of the pancreas are rare. When they occur, they are commonly associated with trauma to other intra-abdominal organs. They are caused by stab or gunshot wounds, underwater blasts, and severe contusions of the abdominal wall. Not infrequently minor injuries to the pancreas occur during the course of operative procedures in the upper abdomen, such as gastrectomy for duodenal or gastric ulcer, operations on the common bile duct, and splenectomy.

Occasionally pancreatic necrosis will follow a severe pancreatic injury. Pancreatic abscess, fistula, or cyst formation not infrequently develop following such injuries.

The clinical picture is that of a patient with an abdominal wound or other injury that manifests an unusually severe degree of shock. However, the diagnosis is seldom made prior to surgical exploration.

Treatment.—The treatment of serious pancreatic trauma includes the control of the associated shock and blood loss. Early operation is necessary in order to assess and repair any

ACUTE PANCREATITIS

There are two commonly recognized types of acute pancreatitis: (1) acute pancreatic necrosis or hemorrhagic pancreatitis; and (2) acute edematous pancreatitis. It is not definitely known whether or not these are separate diseases or whether they are different degrees of the same condition, but it seems most probable that the latter concept is the more correct. Acute pancreatic necrosis, although less common than the edematous variety, constitutes the more serious problem as regards treatment, morbidity, and mortality.

Etiology.—Although great progress has been made in the diagnosis and treatment of acute pancreatitis, the etiology of this disease is still somewhat obscure. The following classification indicates the numerous factors which may be the cause of this condition.

Etiology and Pathogenesis

- A Infectious Factors
 - 1 Viral, e.g., mumps pancreatitis
 - 2 Bacterial
 - a By direct spread from a contiguous infected gall bladder
 - b By lymphatic spread from an infected appendix
 - c By hematogenous spread
- B Noninfectious Factors
 - 1 Reflux of
 - a Bile—the "common channel" theory, as a result of
 - stone impacted at the ampulla
 - or spasm of the sphincter of Oddi
 - 2 Obstruction of pancreatic ducts due to
 - a Squamous metaplasia of the duct lining
 - b Pancreatic calculi
 - c Edema of the duct wall (often associated with the consumption of alcohol)
 - d Tumor
 - e Extrinsic pressure, e.g., duodenal diverticulum
 - 3 Trauma (known to give rise to certain cases of pancreatitis)
 - 4 Local vascular factors
 - a Rupture of a vessel
 - b Occlusion of arterial supply from thrombosis or embolism (including the possibility of fat embolism) or capillary thrombosis as a result of a sensitization reaction (Arthus phenomenon)

- 5 Nutritional and metabolic factors supported by experimental work of
 - a Coffey—patients with hyperlipemia and pancreatitis
 - b Kahser—d,l-methionine production of pancreatitis
 - c Hsieh—importance of collagenase, an enzyme present in inactivated pancreatic juice
 - d Alcohol
 - e Schwartzman reaction
- 6 Associated gall bladder disease

Pancreatitis of Infectious Origin

Occasionally the disease may be due to bacteria carried by the blood or lymph stream or in regurgitated bile. However, in most cases it would appear that any degree of infection follows only because of the secondary invasion of bacteria in the devitalized tissue of pancreatic necrosis.

There is an increased incidence of acute pancreatitis associated with typhoid fever and scarlet fever, and it has been reported that the Coxsackie virus has been isolated from patients with acute pancreatitis.

Pancreatitis of Noninfectious Origin

Although the etiology of acute pancreatitis is obscure, one factor seems to be present in all cases of the disease, whether occurring clinically or produced in the experimental animal. This essential factor is the escape of activated pancreatic enzymes from the ducts and acini into the pancreatic stroma and thence into the adjacent tissues. The release of these activated enzymes causes the local inflammation and necrosis, and their subsequent absorption into the blood stream probably plays a part in the profound systemic effect associated with the disease. It is probable that there is more than one mechanism enabling the escape of these activated enzymes into the unprotected gland tissue, the most likely being a blockage of the pancreatic ductal system which may be due to a stone in or edema of the ampulla of Vater, hyperplasia of the ductal epithelium, edema or tumors of the pancreatic duct or ampulla, or an extrinsic pressure factor. Experimental pancreatic duct occlusion will cause pancreatitis following the administration of secretin. The most likely sequence of events is that a regurgitation of activated enzymes

causes a mild inflammatory change. This is similar to the picture encountered in edematous pancreatitis.

Pancreatic necrosis then occurs as the result of a superimposed etiologic factor, probably vascular occlusion. The occlusion of vessels in the presence of inflammation results in necrosis. This vascular occlusion may be due to venous stasis caused by pressure on the vessels by the swollen and edematous pancreas, from vessel spasm, or from fat embolism. The site of the occlusion is probably capillary. The capillary wall breaks down, allowing a mixture of blood with pancreatic juice which produces a toxic substance capable of pancreatic digestion.

The role of the common channel has still not been proved. This anatomic abnormality frequently occurs in patients with pancreatitis and often has been found in autopsy specimens. However, it seems that there must be another factor in addition to the common channel required to produce the disease complex, namely, the factor of obstruction.

The pressure in the pancreatic ducts is 300-500 ml. of water, and it would be necessary to produce a pressure of 500 ml. of water in the biliary tree to cause a bile reflux; even if this does occur, it is unlikely that the pressure in the ducts is sufficient to cause exudation of pancreatic juice.

New impetus to the common channel theory has been given by the work of Elliott et al., who have shown that equal parts of bile and pancreatic juice, incubated for 14-48 hours at 37° C. and injected at low pressures into the pancreatic ductal system, will cause acute hemorrhagic pancreatitis.

Pancreatitis is frequently associated with biliary tract disease. In a series at the Royal Victoria Hospital this was found in 90% of cases. The diseased gall bladder may cause reflux of infected bile into the pancreatic duct, but this has not been proved. Nor has it been proved that gall bladder disease is infectious in origin. Whatever the real relationship, a high incidence of gall bladder disease with pancreatitis has been repeatedly shown.

Pancreatitis has also been found to be associated with lipid metabolism derangements

essential jaundice hyperlipoidemia can be found to precede pancreatitis in some cases.

Pancreatitis can also be produced by the administration of ethionine, which is an amino acid antagonist of methionine. This amino acid interferes with protein metabolism, causing a degeneration of the pancreatic cells.

Alcohol.—Consumption of alcohol is frequently associated with pancreatitis, either acute or chronic. Just how alcohol is involved in this disturbance is problematic. It may act through an associated dietary insufficiency, chiefly protein, which causes pancreatic cell degeneration; it may cause edema of the ampulla of Vater, leading to pancreatic duct obstruction; it may be that the vomiting of acute alcoholism causes a pressure reflux of duodenal contents into the pancreatic ducts, it may also be that alcohol has a specific toxic effect on the pancreatic cells; or it may cause the liberation of large amounts of secretin which increases the pancreatic flow against a partially obstructed duct.

Acute Hemorrhagic Pancreatitis

Pathology.—The pancreas is enlarged and is usually soft and friable, particularly in the fulminating types of the disease, although the gland may be firm in the milder forms. There are hemorrhage, marked edema, and necrosis of the pancreatic tissue. These changes may be confined to one part, e.g., the head or tail, or may involve the entire gland. It is our impression that when the tail is involved, the pain in the left hypochondrium is more pronounced. The yellowish white spots of *fat necrosis* occur on the surface of the pancreas or on the adjacent peritoneum and viscera. Usually these areas are pinhead or match-head in size but can be much larger and very widespread in the severe case, having been seen in the pericardium and mediastinum in some cases which have come to autopsy. This condition of fat necrosis is not a necrotic process at all but is the result of a chemical change caused by action of the pancreatic enzyme lipase, which acts on the neutral fat in the body, splitting it into glycerol and fatty acids. These fatty acids combine with the ionizable body calcium to form an insoluble calcium soap, which, when precipitated, constitutes the characteristic lesions.

A varying amount of blood-stained fluid is found in the peritoneal cavity; the lesser peritoneal sac may be completely filled.

Clinical Features.—Acute pancreatitis usually occurs in middle-aged patients, most commonly in the obese and overindulgent male, although it does occur in young and otherwise healthy adults. There is a frequent history of gall bladder dyspepsia and previous acute attacks of similar nature to the present one. There is a frequent history of a recent heavy meal or a large intake of alcoholic beverage. The disease has an acute onset and is accompanied by pain, which is most marked in the mid or left epigastric region but may extend to either the left or right subcostal regions or may even be referred to the left subscapular, left lumbar, or left shoulder regions. Rarely, the pain may be in the right shoulder and the right subcostal region.

In the severe cases of hemorrhagic necrosis the patient exhibits all the signs of shock, with a rapid pulse, cold clammy skin, cyanosis, and low blood pressure. There is evidence of marked physical and mental distress. Vomiting is severe and persistent, and the vomitus may be blood-tinged. Dehydration is early and severe. Abdominal tenderness is present, but it is not marked and is usually maximum in the left epigastric region. There is rigidity of the abdomen, most perceptible in the epigastric region, but it is not so pronounced as, for example, the abdominal rigidity in perforated peptic ulcer. Rectal examination reveals no tenderness. This fact helps to differentiate this condition from perforated peptic ulcer, where rectal tenderness is usual, because of the acid peptic juice which runs down into the pelvis along the paracolic gutters. There may be areas of bluish discoloration in the flanks and in the umbilical region, due to extravasated blood and pancreatic enzymes. This discoloration is evidence of a very severe form of the disease but, as it occurs late, it is of little or no help in diagnosing either the mild varieties or the early stages of the severe form. Aspirated fluid from this discolored area has a high amylase content.

Acute Edematous Pancreatitis

Pathology.—As is evidenced by the name, the pancreas is swollen and edematous. There

may be small areas of hemorrhage, but there is no widespread gangrene or necrosis of the gland itself. Fat necrosis of moderate degree occurs in the usual areas

Clinical Features.—The clinical features of acute edematous pancreatitis are similar in nature, but are less severe than those of pancreatic necrosis. Again, as in the hemorrhagic variety, there is a frequent history of gall bladder dyspepsia and previous acute attacks of a similar nature to the present one. The pain and the rigidity are much less pronounced, and shock is not common. The condition is frequently confused with acute cholecystitis.

Diagnosis.—The signs and symptoms of the severe form of the disease are typical. However, the diagnosis of the edematous variety frequently requires laboratory confirmation. The serum amylase and lipase are elevated, particularly in the early stages of the disease. The level of the blood sugar is raised, while that of the serum calcium is depressed. The serum bilirubin is slightly elevated. There is a moderate leukocytosis and a raised hematocrit. Survey films of the abdomen often show dilated loops of bowel. Glycosuria may be present (See Laboratory Aids in Diagnosis.)

Differential Diagnosis.—Acute pancreatitis must be differentiated from acute cholecystitis, perforated peptic ulcer, early intestinal obstruction, acute appendicitis, mesenteric thrombosis, acute gastritis, and coronary artery occlusion.

Treatment of Acute Pancreatitis

The plan of treatment of acute pancreatitis is directed toward the support of the patient and the prevention of spread of the disease process. Surgical intervention is not indicated in the uncomplicated case of acute pancreatitis.

The treatment of edematous pancreatitis is the same as that of the acute hemorrhagic type. However, heroic supportive measures are rarely necessary in the milder form of the disease. There are eight aspects of the plan of treatment:

- 1 The relief of pain
- 2 The treatment of shock
- 3 The treatment of dehydration
- 4 The prevention of further autodigestion of the pancreas

5. The correction of secondary metabolic derangements

6 The prevention of secondary infection

7. The removal of the gall bladder

8 Dietary control

The Relief of Pain.—Very severe pain must be relieved. Occasionally it is so severe that analgesics such as morphine or Demerol must be administered in full therapeutic dosage. However, as both these drugs cause an increase in duodenal irritability and spasm of the sphincter of Oddi, they are theoretically contraindicated as this sphincter spasm may contribute to the disease process by promoting bile reflux into the pancreatic ducts.

Milder analgesics such as codeine or acetylsalicylic acid may be sufficient in some cases. The barbiturates and the nitrates are useful in the relief of pain because of their smooth muscle-relaxing properties.

Paravertebral procaine nerve block from T4 to T10 or an epidural or a splanchnic block will relieve pain, even in the most severe cases. This method of pain relief has a second virtue in that it will tend to relieve any pancreatic ischemia by blocking out the vasoconstrictor nerve fibers of the pancreatic vessels.

The administration of analgesics and the employment of paravertebral or splanchnic block should be continued for as long as the presence of pain requires their use. As a rule, one procaine injection is sufficient and need not be repeated. The milder forms of pain-relieving drugs are generally all that are required once the acute phase of the disease has passed.

The Treatment of Shock.—The occasional severe shock in acute pancreatitis is treated by the usually accepted antishock measures, the mainstays of which are intravenous infusions of whole blood and blood plasma. Plasma substitutes may be used as an initial step or if the blood and plasma are not available. Tissue anoxia should be minimized by the administration of oxygen, either by nasal catheter or by an oxygen tent.

ACTH, cortisone, and compound F are useful in those cases of fulminating hemorrhagic pancreatitis which develop severe shock and which do not respond to antishock measures. In such patients we have found that marked

improvement may follow the use of those drugs.

The following dosage schedule should be used: The quick-acting ACTH is given in amounts of 10-25 mg. dissolved in a liter of intravenous fluid. This quantity is given over a period of 12 hours, or intravenous cortisone at the rate of 10 mg./hour. This dose should be repeated every 12 hours for the ensuing 48 hours, after which it can be supplanted by intramuscular cortisone. At the same time as the initial dose of ACTH, the slower-acting cortisone, in a dose of 300-400 mg., should be given intramuscularly. This dose is followed in the second 24-hour period by another injection of 200-300 mg. The cortisone is then repeated daily, reducing the dose somewhat each day, until it is finally discontinued after 5-6 days. Oral cortisone is not practical in these cases because of the persistent vomiting and the therapeutic use of continuous gastric suction.

Repeated eosinophil counts should be done when using ACTH and cortisone therapy, as it has been found that when the eosinophil count does not fall following the use of these drugs it is an indication that the dosage level is inadequate. The dose should then be increased until an eosinophil response is obtained.

Once the immediate shock is controlled, further administration of blood plasma or plasma substitutes is not necessary unless an exacerbation of the acute pancreatitis occurs.

The Treatment of Dehydration.—Dehydration occurs early and is usually very severe. It is due to the prolonged and copious vomiting, the absence of fluid intake, the intraperitoneal loss of pancreatic juice, and the marked peritoneal exudate which occurs because of the irritation caused by released enzymes. In the later stages the developing paralytic ileus, with its large loops of adynamic bowel, which frequently contain many liters of fluid, contributes still further to the state of dehydration. There is, in all these cases, a combined depletion of both electrolytes and water, as large quantities of the acid chloride ion are lost in the vomitus and large quantities of the alkaline sodium ion are lost in the released pancreatic juices. Replacement therapy must therefore be planned and balanced, so that the correct pro-

portions of electrolytes are administered to make up these deficits. In the later stages of the disease the potassium ion should also be given to replace the intracellular ion deficit, which closely follows the original extracellular dehydration.

An indwelling urethral catheter is an important part of the fluid replacement program, as an accurate measure of the urine output is at all times most essential.

Once the marked dehydration has been corrected by vigorous treatment with a balanced electrolyte solution, the day-to-day fluid and electrolyte requirements of the patient who is taking no fluid by mouth and who is on continuous gastric suction must be maintained by a carefully calculated daily amount. This fluid must be given by parenteral and never by oral means during the period of gastric suction. Accurate fluid intake and output measurements must be continued throughout the entire course of the disease.

The Prevention of Autodigestion.—In order to prevent further outpouring of pancreatic enzymes from the damaged ductal system, certain measures can be taken to minimize pancreatic secretion. The neurogenic phase of the pancreatic secretion can be reduced by the administration of cholinergic blocking drugs such as atropine or Banthine which should be given in full therapeutic dosage until symptoms of overdosage of these drugs appear. Atropine and Banthine must be given by hypodermic or intravenous injection, as drugs are not absorbed from the gastrointestinal tract of a patient with acute pancreatitis, and in any event the therapeutic gastric suction prevents successful oral administration of any drug.

The hormonal phase of pancreatic secretion can also be minimized by measures which prevent the gastric contents from entering the duodenum. The best method of accomplishing this is by continuous gastric suction.

Oral fluids should be prohibited during the period of suction as they will only serve to increase gastric secretion, which will increase the production of secretin which, in turn, will further increase the liberation of pancreatic enzymes, thus adding to the neurogenic addition to the direct effect on the neurogenic secretion, the cholinergic blocking drugs, via their vagus effect, also decrease the flow of

gastric juice, thus helping, in this manner, to reduce the hormonal phase of pancreatic secretion.

Antienzymes have been used experimentally: quinine as an antilipase factor, soybean and S.F.S. (sodium formaldehyde Sulfoxylate) as antitypsin agents. A carbonic anhydrase inhibitor (compound 6063), has also been used. The results have not been particularly good, and these substances are not recommended for clinical use at present.

The continuous gastric suction should be maintained until all the biochemical abnormalities have returned to normal. On no account should this important aspect of the treatment be discontinued simply because the serum amylase level becomes normal. The use of the cholinergic blocking drugs should be continued well into the convalescent period. If the gall bladder is to be removed, these drugs should be continued until the patient finally recovers from the operation, which may be 6-8 weeks after the acute attack.

The Correction of Secondary Metabolic Derangements.—

Calcium Deficiency.—A low serum calcium level should be corrected by the administration of intravenous calcium salts. It is estimated that approximately 150 mg. of calcium ion should be given for every 1 mg. drop in the serum calcium level. Due regard, however, must be paid to the errors inherent in serum electrolyte estimations, e.g., hemoconcentration. This dose will not have to be repeated unless there is an exacerbation of the disease process leading to a second formation of fat necrotic areas. The precipitation of the areas of fat necrosis is an early and, so far as we know, a nonprogressive phenomenon, so that there is no necessity for the continued feeding of a calcium-hungry area, such as is found in the bones of a patient suffering from the post-operative tetany of hyperparathyroidism. In these cases calcium must be given daily for a considerable period of time, until the bone depletion has been corrected.

Careful attention must be paid to the fact that different salts of calcium contain very different quantities of the calcium ion, for example, calcium gluconate contains about 9% calcium, calcium lactate about 18%, and

calcium chloride about 36%. If, therefore, it is desired to administer 150 mg. of calcium ion, it is necessary to give the patient approximately 1.5 Gm. of calcium gluconate. The chosen quantity of calcium should be administered by a slow intravenous drip over a period of 3-4 hours. Those salts of calcium which contain a relatively large proportion of ionizable calcium, e.g., calcium chloride, are not recommended.

The continued use of parenteral calcium should not be necessary after the first 48 hours, unless there is an exacerbation of the disease. Once the patient takes food by mouth, the average normal diet contains sufficient calcium for his needs, so that it need not be added to his diet in the form of calcium salts or high calcium foods.

Hyperglycemia.—It is important that any severe and prolonged hyperglycemia be prevented by the administration of insulin. The insulin-producing Beta cells of the pancreas are susceptible to high blood sugar levels, and those islet cells which escape the initial injury of the pancreatic destruction may be permanently damaged should an uncontrolled hyperglycemia be allowed to persist. The blood sugar level should be controlled by small repeated doses of the quick response, short action crystalline insulin. Repeated estimations of the blood sugar are necessary in order to regulate the insulin dosage. Estimation of the urinary sugar is of little value in these cases as the vigorous intravenous therapy with glucose-containing solutions causes a glycosuria, even in the absence of impaired carbohydrate metabolism.

Insulin overdosage should be scrupulously avoided, as the vagus nerve, when stimulated by hypoglycemia, will cause an increase in the external pancreatic secretion.

It is believed that the conservation of islet cell tissue by the careful administration of insulin in these cases of acute pancreatitis, which show more than a transient hyperglycemia, will reduce the number of patients who develop diabetes subsequent to the pancreatitis.

If the acute pancreatitis has been severe, with marked tissue damage, the resultant hyperglycemia may be prolonged and even permanent. Careful dietary and insulin control

should be continued until the abnormal carbohydrate metabolic state becomes stabilized. This may take 2-3 weeks. A glucose tolerance curve should be carried out on all patients after they have recovered from the acute disease in order to diagnose any insipient diabetes.

The Prevention of Secondary Infection.—The development of early infection is not a problem in acute pancreatitis. However, later in the course of the disease, particularly if laparotomy and drainage have been carried out, infection with abscess formation may occur. This abscess formation may also arise via lymphatic drainage from the gastrointestinal tract, or it is remotely possible from regurgitated infected bile. Infection can be prevented by the avoidance of operation and by the program outlined above. A wide-spectrum chemotherapeutic agent should be used. Some of these agents have a theoretic advantage in that when given by mouth, they are secreted by the liver with the bile. This factor may help to prevent reactivation of the pancreatitis from infected bile if such a situation obtains.

The administration of the chosen chemotherapeutic agent should be continued for several days after the patient has a normal temperature and has otherwise recovered from the acute pancreatitis.

Removal of the Gall Bladder.—As chronic cholelithiasis and chronic cholecystitis so frequently accompany or precede acute pancreatitis, it is recommended that once the patient recovers from the acute attack, the diseased gall bladder should be removed. The optimum time for this procedure is 6-8 weeks following recovery. During this intervening time the patient should be kept on a modified acute pancreatitis regime, which consists of a low fat bland diet with atropine or Banthine and the prohibition of alcohol.

In patients in whom there is no demonstrable gall bladder disease or common duct obstruction, the gall bladder should not be removed. However, the patient should be carefully observed over a prolonged period so that if such disease does become detectable, suitable operative treatment may be planned.

Although the treatment of acute pancreatitis is not operative, occasions do arise when a

laparotomy is performed. Perhaps the diagnosis may be obscure or the abdomen may require opening for an associated disease, commonly acute cholecystitis. If the patient is not severely ill, a chronically diseased gall bladder may be removed at this time with perfect justification. If an acutely inflamed gall bladder is discovered, it is desirable that it be removed if at all possible. However, if the patient is extremely ill, drainage of the gall bladder may be all that can be safely performed. If a patient with acute hemorrhagic pancreatitis is subjected to laparotomy, it is doubtful whether any surgical procedure should be attempted except that an acutely inflamed gall bladder occurring in association with the disease may be drained.

So far as the common duct is concerned it should be explored in nearly all cases of elective cholecystitis associated with pancreatic disease. It may at times be explored in patients during an acute attack of edematous pancreatitis if the patient's condition is satisfactory. The common duct, however, should not be touched in cases of acute gall bladder disease associated with edematous pancreatitis or in the presence of acute hemorrhagic necrosis.

Dietary Control.—Once the intravenous feeding has been discontinued, the patient should be placed on a high protein, high caloric, and low fat bland diet. This food should be given in 5-6 small meals a day, which tends to prevent overloading of the stomach and resultant undue stimulation of the biliary and pancreatic systems. Alcoholic beverages should be prohibited.

Complications of Acute Pancreatitis

Complications are more usually found following the acute hemorrhagic type and include abscess and fistula.

The treatment of pancreatic abscess is surgical drainage and appropriate chemotherapy. The treatment of pancreatic cysts and fistula is discussed later.

As a sequel to the disease, impaired glucose tolerance or frank diabetes may occur which will require dietary or insulin control. There may also be an impairment in the production of pancreatic enzymes, leading to the bulky, pale stools of pancreatic insufficiency.

CHRONIC PANCREATITIS

It is still a matter of debate as to whether this condition is a disease in itself or a sequel of acute pancreatitis. The abnormality takes several forms, the most common of which is seen in the indurated pancreas which is frequently found at laparotomy, particularly when the conditions of chronic cholecystitis and cholelithiasis are present. This pancreatic induration probably does not cause symptoms which can be differentiated from those of the associated gall bladder disease. Specific treatment is not necessary, and removal of the gall bladder, in nearly all cases, ends the problem. More advanced chronic pancreatitis is much more serious. The chief symptom is severe and unremitting pain which frequently drives the patient to drug addiction. There is also evidence of chronic malnutrition, pancreatic insufficiency, and at times jaundice.

The third type is the so-called chronic relapsing pancreatitis, the usual pattern of which takes the form of irregularly recurring attacks of acute pancreatitis. The attacks increase in severity and frequency as the disease continues, until the picture is identical with the above-mentioned type of advanced chronic pancreatitis.

Pathology.—The pathologic picture varies from a minimal degree of fibrosis, which may be limited to one area of this gland, to a diffuse fibrous change of all the gland, with an almost complete destruction of the pancreatic parenchyma and obliteration of the ductal system. In addition to the fibrosis there may be calcification of the parenchyma, stones in the pancreatic ducts, or both.

Clinical Features—The diagnosis of chronic pancreatitis is difficult and can be proved only by operation and biopsy or at autopsy. Pain is a very common symptom; it is epigastric in position and tends to radiate to the back. This pain, however, is not pathognomonic of chronic pancreatitis and is not in any way a reliable diagnostic feature. When jaundice occurs it is of the typical obstructive type, and although pancreatic disease may be suspected to be the underlying cause, carcinoma of the pancreas cannot be excluded on clinical grounds alone. Diabetes or low sugar tolerance is not uncommon, and there may be signs of deficiency

in the external pancreatic secretions which is manifested by pale, bulky stools. There may be a decrease or absence of the pancreatic enzymes in aspirated duodenal contents. The levels of serum amylase and lipase may occasionally be elevated. Loss of weight is common if the disease has been established for a period of time. A plain x-ray of the abdomen may show pancreatic calcification.

Treatment.—This is both medical and surgical. Measures should be taken to ensure prohibition of food and drink which promotes a marked pancreatic response, e.g., fat, large meals, and alcohol.

Pancreatic ferments and insulin may be required if there has been marked glandular destruction. Anticholinergic drugs and alkalies may also be helpful.

The main form of treatment, however, is surgical. Selection of the type of procedure varies with the pathologic process, and the choice can frequently be determined by clinical and laboratory means.

There are six forms of surgical therapy.

1 Cholecystectomy for a diseased gall bladder and removal of stones from the common duct.

2 Relief of jaundice in those patients with severe pancreatic fibrosis producing occlusion of the common bile duct. This is accomplished by dilatation of the common duct and subsequent prolonged drainage or by biliary tract diversionary measures such as cholecystenterostomy, choledochenterostomy, or choledochoduodenostomy.

3. Operations which promote common duct drainage such as transductal or transduodenal division of the sphincter of Oddi or choledochoduodenostomy.

4 Operations designed to decrease pancreatic secretion, such as partial gastrectomy, vagotomy, and gastroenterostomy.

5 Operations on the pancreas itself which may be

a Removal of stones from the pancreatic ducts.

b Excision of the tail of the pancreas with subsequent retrograde drainage by anastomosis of the body of the pancreas and the pancreatic duct to a defunctioned loop of jejunum by a Roux-Y anastomosis.

c. Resection of the pancreatic head, i.e., pancreaticoduodenal resection

d. Total pancreatectomy

6 Procedures designed for the relief of pain such as splanchnicectomy, alcohol injection of the splanchnic nerves, or epidural injection with local anesthetic, followed by a subsequent injection of alcohol

Selection of Cases for Therapy.—

1. Cholecystectomy should be performed in every case of chronic pancreatitis if there is any evidence of gall bladder disease. The common duct should be explored at the time of the cholecystectomy.

2. The presence of jaundice always poses the problem as to whether or not cancer is present. A biopsy and frozen section is often very helpful in deciding this point

Chronic fibrosing pancreatitis causing common duct occlusion and jaundice is treated by diversion of the biliary stream by either cholecystenterostomy, choledochenterostomy, choledochoduodenostomy, or prolonged common duct drainage.

3. Division of the sphincter of Oddi is of value only when the pancreatic duct obstruction is at or near the sphincter. If the block is in the head of the pancreas, division of the sphincter will not benefit the condition. This form of treatment is best suited to chronic relapsing pancreatitis.

4 Gastric resection and vagotomy tend to minimize duodenal stimulation by acid chyme and thus prevent vagal stimulation of the pancreas. These procedures are of value only in chronic relapsing pancreatitis.

5 The choice of operation on the pancreas itself varies with the pathologic process. Pancreatic ductal stones are removed when palpable or when visualized in plain films of the abdomen or by pancreatic duct injection with contrast media

Retrograde drainage is feasible when there is stricture, metaplasia, or calcification in the head of the pancreas which can be decompressed by pancreatic intestinal anastomosis

Partial or total pancreatectomy may be carried out for marked fibrosis or calcification. This is a formidable procedure which should not be undertaken lightly.

6. As the nutritional problem following total pancreatectomy is difficult and the mortality of the operation is high, marked fibrosis causing intractable pain is probably best treated by the indirect approach of splanchnicectomy or epidural nerve blockage.

Specific Chronic Pancreatitis

Specific inflammatory conditions of the pancreas, such as those due to syphilis and tuberculosis, are rare. The treatment is that of syphilitic syphilis and tuberculosis.

Pancreatic Lithiasis

The etiology of this condition is unknown, but it is probably a sequel of acute or chronic pancreatitis. Pancreatic stones are similar to salivary calculi. They are usually multiple and lie in the larger ducts. Calcification of the pancreatic parenchyma also occurs, either in conjunction with pancreatic stone or by itself.

Clinical Features.—The clinical picture is almost identical with that of chronic pancreatitis. The pain, which may be very severe and debilitating, is deep epigastric in position and radiates to the back. Nausea and vomiting are fairly common. The patient may show signs of pancreatic insufficiency with sometimes diabetes or jaundice. X-ray of the abdomen will frequently demonstrate the calculi which are usually radiopaque. Calcification of the pancreatic parenchyma is also shown by this method

Treatment.—The treatment is surgical and consists of removal of the stone in cases where this is possible or pancreatic resection in those cases with diffuse calcification. As in ordinary chronic pancreatitis, bilateral splanchnicectomy will give relief of pain in some cases. Attention must be paid to insufficiencies of the pancreatic secretions.

PANCREATIC CYSTS

Pancreatic cysts are not common; the most frequently occurring variety is pseudocyst of the pancreas, which develops as a complication of acute pancreatitis or pancreatic injury. The following classification indicates the types which have been reported (Mahorner and Mattson).

- I. Cysts resulting from defective development
 - (a) cysts in infants
 - (b) cysts associated with polycystic disease of the kidney
 - (c) dermoid cysts
 - (d) inclusion cysts
- II. Cysts resulting from trauma
- III. Retention cysts
- IV. Neoplastic cysts
 - (a) cystadenoma
 - (b) cystadenocarcinoma
 - (c) teratomatous cysts
- V. Cysts resulting from parasites

Pathology.—Pancreatic cysts may occur in any part of the gland. They usually present above or below the stomach but may be below the colon, causing displacement of these organs; this can be demonstrated by x-ray.

Following are the three most common and most important cysts of the pancreas. (1) cystadenoma, (2) fibrocystic disease, and (3) pseudocyst

Clinical Features.—The clinical features of pancreatic cysts in adults are the same regardless of the exact nature of the cyst. There is frequently a history of acute pancreatitis or trauma to the abdominal wall or of abdominal operations. The majority of cysts, however, are not noticed until they form a readily palpable mass. In the later stages there is vague pain, loss of weight, nausea, and vomiting. Rarely jaundice occurs due to extrinsic pressure on the biliary system. Impaired glucose tolerance, diabetes, and insufficiency of external secretion may occur.

Cystadenoma of the Pancreas.—This is a large polycystic tumor with papillary projections from its columnar cell lining. It rarely gives rise to symptoms until it becomes large enough to cause pressure on the surrounding structures. Malignant forms occur. The treatment is surgical removal.

Fibrocystic Disease.—Fibrocystic disease of the pancreas occurs in infants and children. It is characterized by widespread fibrocystic changes in the pancreas, which are usually associated with similar changes in other organs, notably the lungs and kidneys. There is complete loss of external pancreatic secretion due to malformation of the ducts, which gives rise to the condition known as meconium ileus.

Treatment.—Laparotomy is usually required to relieve the intestinal obstruction. Pancreatic enzymes should also be given. The prognosis

is unfavorable because of the associated fibrocystic disease in other organs.

Pseudocysts or False Cysts.—These cysts are effusions into the lesser peritoneal sac which become sealed off by adhesions. Occasionally they contain blood because of small hemorrhages from the vessels either in the pancreas or the cyst wall. They occur following acute pancreatitis or trauma.

Treatment is excision of the cyst with anastomosis of the cystic remnant to the jejunum by a Roux-Y anastomosis. Sometimes this is not feasible because of technical difficulties, and a simple marsupialization of the cyst to the anterior abdominal wall is carried out. If this is done, it is recommended that a sphincterotomy be done at the same time and the patient be forbidden alcoholic beverages and placed on a low fat diet until the pancreatic secretion completely disappears.

PANCREATIC FISTULA

These fistulas may be internal or external. The diagnosis of the external variety is obvious, and it is the only one of clinical importance. There is a history of acute pancreatitis or surgical operation. There may be insufficiency of either the internal or external pancreatic secretions. In severe forms of the external variety, dehydration, due to loss of fluid and electrolytes, may occur. There is frequently a digestion of the skin of the abdominal wall in the early stages of external fistulas.

Treatment is excision of the fistula where possible or anastomosis of the tract to the gastrointestinal system.

TUMORS OF THE PANCREAS

The usual benign glandular tumors which occur elsewhere in the body are found in the pancreas, namely, adenoma, fibroma, and fibroadenoma. They rarely cause symptoms and are seen as incidental findings at laparotomy or autopsy. Two forms of tumors of the pancreas are of clinical importance:

1. Islet cell tumors
2. Adenocarcinoma

Islet Cell Tumors

Tumors of the islet cells are frequently associated with hyperinsulinism. They do oc-

cur, however, with no demonstrable change in carbohydrate metabolism and pass unnoticed unless discovered during abdominal exploration for other conditions or at autopsy. When hyperinsulinism is present, the symptoms are those of severe and recurrent hypoglycemia, which is manifested by sweating, flushing, pallor, dizziness, weakness, hunger, nausea, epigastric pain, syncope, and mental changes. A diagnosis of epilepsy or some circulatory abnormality is sometimes erroneously made in these cases. Hyperinsulinism can also be caused by a diffuse hyperplasia or hypertrophy of the islets of Langerhans. The symptoms are indistinguishable from those of islet cell adenoma.

Treatment.—Surgical removal of the hyperfunctioning tumor is essential. Occasionally a diffuse hyperplasia of the islet cells causes hyperinsulinism. In this case the treatment is radical subtotal resection of the pancreas. The results of excision of the pancreatic adenoma are good if irreversible mental changes have not occurred.

(See Chapter 13, *Surgery of the Endocrine Glands*.)

Carcinoma of the Pancreas

The only malignant tumor of clinical importance other than the islet cell carcinoma is adenocarcinoma. It has been more often encountered in recent years because of more accurate diagnosis and the growing awareness of this condition.

Incidence.—Carcinoma of the pancreas constitutes about 1.2% of all carcinomas. It usually occurs in middle or late life and is more commonly found in men than in women.

Pathology.—The tumor is an adenocarcinoma, usually scirrhous in type, which arises from the epithelium of the duct system. Occasionally it is medullary in type. The neoplasm develops in either the head or tail, or it may involve the entire gland. Carcinoma of the pancreas is most commonly found in the head, 81% of cases according to Berk. It may, of course, be found in the tail or invading the entire gland.

Carcinoma of the pancreas invades the adjacent structures early, and involvement of the vena cava and superior mesenteric and

portal veins frequently prevents pancreatic resection. Metastases occur in the liver and in the lymph nodes accompanying the pancreaticoduodenal, gastroduodenal, and celiac vessels. This problem of early invasion and metastatic spread is one that prevents successful surgical treatment, because when the patient begins to complain of sufficient pain and jaundice to warrant a diagnosis, the disease is far advanced.

Clinical Features.—The usual symptoms of carcinoma, namely, loss of weight, loss of strength, and loss of appetite, are present. Abdominal pain, usually epigastric, is frequent and is of boring character, generally radiating to the back. This pain is a common symptom and the old concept of painless jaundice being diagnostic of carcinoma of the pancreas no longer holds. Some degree of pain is present in over 80% of cases. Carcinoma of the tail or body of the pancreas does not produce jaundice unless it is very advanced. Pain comes on early and is typically the first sign. The tumor is rarely palpable. In patients without jaundice the presence of abdominal pain which radiates to the back, loss of weight, and loss of appetite are indications of pancreatic cancer.

Jaundice is present in 60-75% of all cases and is usually accompanied by pain of a dull, aching character which is frequently referred to the back. The patient also complains of a feeling of intra-abdominal pressure. The jaundice is of an obstructive type and is relatively severe. It is progressive and persistent in contradistinction to the jaundice that occurs as the result of carcinoma of the ampulla of Vater, which may be intermittent in character due to the ball valve action of a pedunculated tumor or necrosis of an obstructing tumor. There is occasionally occult blood in the stool, and blood and tumor cells may be found in aspirated duodenal contents. Ascites, when it occurs, is a late complication and is caused by portal vein obstruction due to lymph node involvement. The gall bladder is frequently palpable in carcinoma of the head of the pancreas. In addition to these signs and symptoms, there may be decreased sugar tolerance or diabetes, glycosuria, and diminution of external pancreatic secretion, as well as a slight elevation in the serum lipase.

Diagnosis.—The diagnosis is frequently difficult. It is made from the history, the presence of jaundice, and the employment of x-ray and other laboratory procedures. There may be an impaired carbohydrate metabolism. The typical x-ray finding in carcinoma of the head of the pancreas is a widening of the duodenal loop due to pressure when visualized by means of barium studies.

Treatment.—To date there has been no satisfactory form of treatment. Many techniques for resection of the involved portion of the gland with reconstitution of the gastrointestinal, biliary, and pancreatic flow have been devised. Such procedures are of value in that they relieve jaundice and thereby make the patient more comfortable. However, the operation is one with a significant mortality, and because of the fact that carcinoma of the pancreas infiltrates rapidly and metastasizes early, it is usually impossible to resect the entire area of malignancy. It is possible that a minute tumor could be cured by radical resection, but unfortunately by the time carcinoma of the pancreas is clinically obvious, it is no longer minute and is no longer amenable to curative surgery. Palliative treatment for severe jaundice with all its accompanying discomforts is helpful. This consists of drainage of the biliary system either externally or by biliary-intestinal anastomosis. The operation of choice is choledochointerostomy by the Roux-Y method. It is important that the gall bladder be removed, as its preservation affords a malfunctioning diverticulum of the biliary tree which is prone to infection and the development of lithiasis. The pain may be relieved by analgesics and by epidural alcohol block. It is not recommended that splanchicectomy be carried out for the relief of pain in these cases.

The duration of life is short once the diagnosis has been made (6-12 months).

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Pancreatic Cysts (By Charles B. Puestow, M.D., F.A.C.S., Chicago)	28 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn
Transabdominal Section of the Sphincter of Oddi for Pancreatitis (1934) (By J. H. Mulholland, M.D., and H. Doubilet, M.D., New York)	24 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn

Chapter 22

Surgery of the Spleen

Louis Lowenstein, MD. and Richard C. Long, MD.

INTRODUCTION

With few exceptions splenectomy is the only surgical procedure involved in diseases of the spleen.

It is stated that splenectomy was performed on Marathon contestants of the last century B.C. in order to increase their stamina. It is not improbable that a high percentage of individuals so treated were handicapped by massive malarial spleens and therefore benefited materially, in a purely mechanical sense, from the removal of such awkward abdominal tumors. In what manner and how frequently it was then possible to compass the technical hazards of operation remains to this day a matter for conjecture.

There are few authenticated records of splenectomy during the Middle Ages and Renaissance period. In 1856 Adelman of Berlin reported 15 cases but failed to mention the underlying pathologic conditions. In 1880 Pean is said to have removed the first splenic cyst. By 1900 Bessel-Hagen had operated upon 37 ruptured spleens. Since the turn of the century splenectomy has followed the general trend of technical improvement and has shared in the security of improved supportive treatment. It is performed with ever-increasing frequency and decreasing mortality. To the traditional indications for this operation—rupture, ectopy and primary tumor—the expanding science of hematology has added vari-

ous blood dyscrasias. However, those of broadest experience will admit that removal of the spleen may be at times impossible and that the consequences of ill-advised surgical interference may be rapidly fatal.

Galen spoke prophetically when he described the spleen as *an organ full of mystery* and Stukeley's opinion (1723), that *what formerly was the seat of joy has become a topic of grief to the moderns*, is doubtless shared by many surgeons of our time.

APPLIED ANATOMY

The spleen is several times larger in life than in death. Its normal autopsy weight is about 100 grams. It is almost completely invested by peritoneum, of which various folds constitute suspensory ligaments; gastrosplenic, phrenicocolenal, lienorenal, and phrenicocolonic. The length of these peritoneal reflections determines the mobility of the spleen, which is subject to marked variation. The phrenicocolonic and phrenicocolic ligaments are normally folded of the lienorenal ligament contains an avascular. The gastrosplenic ligament contains the short gastric and left gastroepiploic branches of the splenic artery, and the lienorenal ligament enfolds the true splenic pedicle. In congestive splenomegaly, all the ligaments are highly vascular, and the spleen is often plastered to the diaphragm by tough adhesions rich in collateral vessels. The peritoneal relationships of the true pedicle make easier the

APPLIED ANATOMY

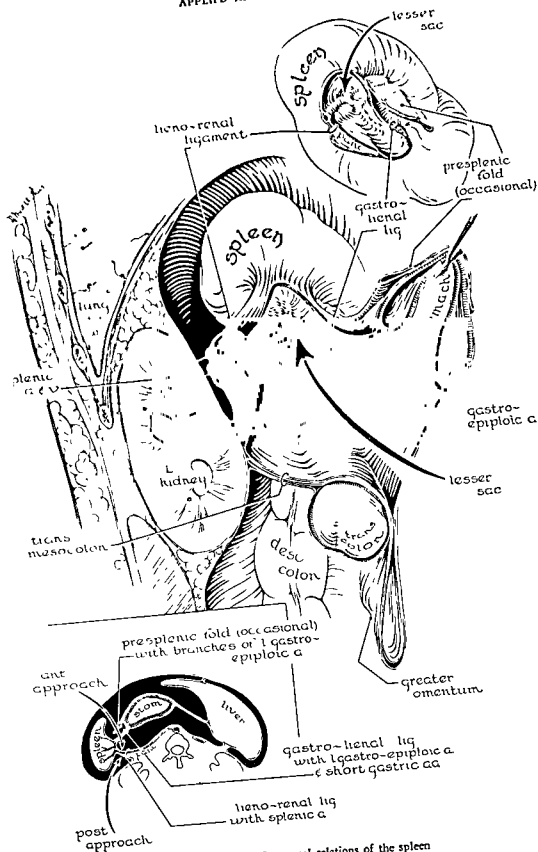


Fig 325—Peritoneal relations of the spleen

posterior approach to the vessels. From behind there is only one intervening layer of peritoneum, from the front, at least three, sometimes five

The spleen lies in the cupola of the left diaphragm. The 9th, 10th, and 11th ribs overlie its convex surface. Because of its situation, friability, and vascular turgidity, it is easily ruptured by direct or indirect violence. Anteromedially, the spleen is closely apposed to the stomach, posteromedially, to the left kidney, the tail of the pancreas, and the colon. Any of these structures is liable to injury during splenectomy.

Microscopic Features.—Besides its peritoneal covering, the spleen possesses a fibro-elastic coat from which trabeculae, penetrating in all directions, form the framework which supports the splenic pulp in its interspaces. The capsule and trabeculae of the human spleen are highly elastic but contain very little nonstriated muscle. Contractility is therefore passive rather than active, in contrast to what is found in laboratory animals. The pulp is a spongy, fibrocellular feltwork of reticulum, of which the meshes contain varying proportions of erythrocytes, lymphocytes, monocytes, reticulum cells, and granulocytes. Anas-

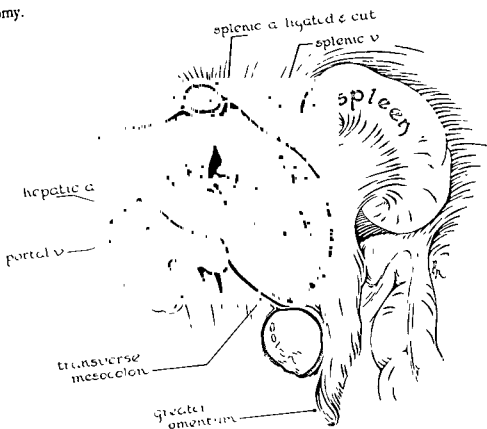


Fig 326—Ligation of splenic artery

The splenic artery lies above the splenic vein, behind the omental bursa, and parallel with the upper border of the pancreas. For purposes of ligation, it may be approached through either the gastrohepatic or the gastrosplenic omentum. Accessory spleens may be present. Their most common sites are the hilum of the spleen, the gastrosplenic omentum, the greater omentum, and the various peritoneal ligaments.

tomosing, cylindrical strands of lymphoid tissue appear in sections as malpighian corpuscles. Each lymphoid cylinder surrounds a lobular branch of the splenic artery. The sharp demarcation of splenic infarcts confirms the frequency of vascular anastomoses. Though it has been described as a giant hemolymph node, the spleen contains no lymphatic vessels.

The microscopic vascular anatomy of the spleen remains a problem. The point of con-

troversty is whether or not the reticuloendothelial components of the pulp are directly exposed to the circulating blood; whether, in other words, the arterial and venous capillaries of the spleen consist of intact endothelial tubes in unbroken continuity, or whether a zone of naked pulp intervenes between them. The former type of circulation has been called *closed*, and the latter *open*. Direct observation of the microscopic circulation of certain mammalian spleens by a number of observers has led us to favor the hypothesis of a morpho-

logically open system which, from the standpoint of function, may be closed rhythmically or in response to those stimuli which cause arterial constriction

APPLIED PHYSIOLOGY

It is generally accepted that the spleen participates in the production, destruction, storage, and filtration of blood. While many of the details of these mechanisms are still obscure, many more have been clarified by recent investigation

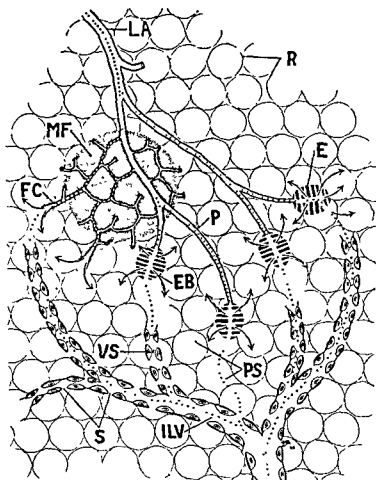


Fig 327—Diagram of the intralobular circulation in certain mammalian spleens (e.g., that of the cat), as suggested, in part, by McNee's representation of a splenic lobule. Arrows radiating from ellipsoids and follicle capillaries suggest a few of the innumerable pathways by which blood may traverse the pulp interstices in relaxed or distended spleens and emphasize the morphologically "open" character of the circulation when the circulation most of the blood, at artery; MF, malpighian follicle, P, ellipsoid, R, red pulp reticulum, PS, pulp spaces, VS, venous sinus, ILV, intralobular vein, S, stigmas in walls of venous sinus and intralobular vein (From MacKenzie, D W, Jr, et al Am J Anat 68: 397-456, 1941.)

Reservoir Function.—Since the classic experiments of Barcroft and his colleagues, it has been thought that the spleen serves as a reservoir of red cells which are released into the general circulation in response either to oxygen lack caused by violent exertion, hemorrhage, asphyxia, diminished atmospheric pressure, or carbon monoxide poisoning, or to any adrenergic stimulus that elicits contraction of the spleen. Anatomically, especially in those animals whose trabeculae contain an abundance of smooth muscle, the spleen is admirably adapted to mobilize its content of blood. In the normal human spleen there is doubt concerning the practical importance of this function, for it has been shown that, following exercise or injection of Adrenalin, hematocrit values increase in splenectomized individuals comparably to those with intact spleens and that in both there is an associated decrease in plasma volume. In certain diseases, however, usually associated with splenomegaly, enhancement of reservoir function may have an important bearing upon the pathologic physiology of the disease, as for example, in spherocytic anemia, hereditary or acquired sickle cell anemia, thalassemia major, and splenic hematopenia. The reservoir function of the spleen has been subdivided in a manner comparable to that of pulmonary function, namely, into complemental, tidal, reserve, and residual blood components.

Hematopoiesis.—Red cells are produced in the spleen during the first two trimesters of fetal life. Following birth, erythropoiesis in the spleen normally ceases. In certain diseases, with impaired hematopoiesis, usually as a result of replacement of normal marrow by abnormal tissue, erythropoiesis, granulocytopoiesis, and/or thrombocytopoiesis may again take place in the spleen, although this compensatory metaplasia is rarely sufficient to adequately replace the marrow hematopoiesis.

Remote Control of Hematopoiesis.—There is evidence that the normal spleen exerts a braking or inhibitory influence upon hematopoiesis in the bone marrow and also controls, to some extent, the maturation and possibly the delivery of red cells to the circulating blood.

To date, most of the evidence for this remote effect of the spleen has been based upon studies of bone marrow and blood before and after splenectomy. In normal persons, removal of the spleen is usually followed by leukocytosis, thrombocytosis, reticulocytosis, the appearance of nucleated red cells, Howell-Jolly bodies, and Cabot rings, basophilia, polychromatophilia, target cells, and increased resistance of erythrocytes to hypotonicity. Anemia may develop and is usually mild and transient. Howell-Jolly bodies have been observed in the red cells many years after splenectomy. The life span of transfused normal red cells is not affected by removal of the spleen. Leukocytosis may persist for years. At first, it is chiefly a neutrophilia. Subsequently the proportion of lymphocytes and monocytes increases. Eosinophilia and basophilia are occasional findings.

Recently, hyposplenism has been observed in nontropical sprue, in sickle cell anemia, and in certain cyanotic congenital heart lesions. In such cases, target cells and Howell-Jolly bodies were found, pointing an analogy to the effects of splenectomy in the normal individual and suggesting the possibility that functional *hyposplenism* as well as *hypersplenism* may occur.

Filtration of Blood.—The capacity of splenic sinusoids and pulp spaces to separate blood cells from blood fluid is readily observed in transilluminated spleens. The term *hematocrit function* aptly describes this activity. The reticuloendothelial components of the pulp, by adsorption and subsequent phagocytosis, immediately remove foreign particulate matter from the fluid which bathes them. This process, which is practically instantaneous, is strong evidence against the presence of a limiting membrane between the circulating blood and splenic pulp.

Blood Destruction.—There is abundant evidence that old or abnormal red cells are selectively trapped in, and destroyed by, the spleen. Though the function of iron storage has been attributed to the spleen, it is not clear that this mineral is retained in excess of the quantity derived from hemolyzed or phagocytosed erythrocytes. In certain diseases associated with excessive blood destruction, abnormal red cells are trapped in the spleen, filtered from the general circulation, and de-

stroyed by increased phagocytic activity. Leucocytes and blood platelets also are phagocytosed in some cases of hypersplenism.

Antibody Production.—Resistance to some infectious diseases is thought to be decreased after splenectomy. Recent evidence indicates that antibodies may be elaborated by and concentrated in the spleen; that, for example, the spleen is the major site of antibody production in some of the acquired hemolytic anemias, thrombocytopenic purpuras, splenic neutropenias, and hematomias. In this group of immunocytopenias the reservoir function, filtration mechanism, hematocrit, stagnating and phagocytic capacities of the spleen enhance the damaging effects of the antibodies. Although the evidence is still incomplete, it seems probable that the spleen is not the sole source of antibody production in the so-called immune types of cytopenias. However, in some instances it may be the major source.

In conclusion, the spleen is not essential to life, nor is its removal necessarily followed by any serious or permanent disturbance.

INDICATIONS FOR SPLENECTOMY

If more were known about the functions of the spleen, the indications for its removal could be stated with greater certainty. In cases of trauma, congenital ectopy, and mechanical exigency, the responsibility is purely surgical. Otherwise, the problem is to interpret the mechanism of imbalance between the hematopoietic functions of the bone marrow and the inhibitory and destructive activities of the spleen. Such interpretation, as has been amply demonstrated, demands the cooperative efforts of a team of hematologists, internists, and surgeons. Adequate preoperative investigation will eliminate most of the pitfalls and disasters of splenectomy. Prolonged follow-up, including frequent hematologic studies, will provide the only accurate assay of its results.

Generally speaking, the indications for splenectomy may be tabulated as follows:

Group I.—As a rule, the following conditions are absolute indications:

1. Rupture of the spleen
2. Torsion of splenic pedicle
3. Hereditary spherocytic anemia
4. Primary splenic hematoma

5. Primary malignant neoplasms of the spleen

6. Aneurysm of the splenic artery

Group II.—Splenectomy may be required for the following:

1. Idiopathic thrombocytopenic purpura
2. Acquired hemolytic anemia
3. Primary splenic hematoma
4. Cystic disease of the spleen
5. Abscess of the spleen
6. Granulomatous infections or parasitic infestations localized to the spleen
7. Hypersplenism secondary to a variety of causes
8. Gaucher's disease, myelofibrosis, kala-azar
9. Splenomegaly of indeterminable etiology
10. Congestive splenomegaly caused by obstruction of the splenic vein

Group III.—Splenectomy may expedite major surgical procedures involving:

1. Esophagus, stomach and pancreas, especially in block resections for malignant disease
2. Tributaries of the portal bed, in shunting operations for the relief of portal hypertension

CONTRAINDICATIONS OF SPLENECTOMY

Splenectomy is contraindicated in the following conditions:

1. Leukemia, lymphatic or myeloid, unless marked hypersplenism present
2. Polycythemia vera
3. Unless marked hypersplenism present, splenic metaplasia, associated with:
 - a. Secondary carcinoma of the bone marrow
 - b. Osteomyelofibrosis
 - c. Marble bone disease
 - d. Refractory anemias resulting from various forms of intoxication (e.g., benzol or radium dial poisoning, or the effects of certain radioactive isotopes)

RUPTURE OF THE SPLEEN

Rupture of the spleen may be caused by direct or indirect violence or it may occur spontaneously. Its effects are either immediate or delayed. Diseased spleens, especially those of malaria, infectious mononucleosis, and acute

generalized infections, are far more liable to traumatic or spontaneous rupture than is the normal organ. It must be remembered, however, that the healthy spleen is more readily lacerated than any other abdominal viscus and that the injury may have been sufficiently trivial to be overlooked by the patient.

Clinical Features.—The signs and symptoms of ruptured spleen are those of internal hemorrhage together with peritoneal irritation. Shock may be immediate. There is abdominal pain, usually more marked in the left hypochondrium and flank. There is often reference of pain to the left shoulder. Muscular resistance is variably increased. Dullness to percussion may be present, shifting on the right but constant on the left side. The picture is frequently complicated by concomitant injury to other organs. In cases where the diagnosis is obscure, abdominal paracentesis with the finding of intraperitoneal blood should make one suspicious of a splenic rupture.

The initial phase may progress rapidly to a fatal termination. Fifty per cent of untreated patients die within an hour of injury. Bleeding may respond promptly to rest, sedation, and one or more blood transfusions, or it may continue slowly and persistently. Delayed hemorrhage is a dangerous complication of untreated rupture of the spleen. It occurs suddenly and without warning hours, days, or even months following the injury. It is due either to a subcapsular hematoma that breaks through the capsule or to a small tear in the spleen with hematoma formation and subsequent delayed hemorrhage.

Treatment.—If massive intra-abdominal hemorrhage is suspected, diagnostic abdominal paracentesis may be performed before laparotomy which must be undertaken as soon as maximum resuscitation has been achieved. Rupture of the spleen will be encountered in over 30% of all severe abdominal injuries. An upper left paramedian incision is preferred. The ruptured spleen, which is usually abnormally mobile, is delivered into the wound, and after its pedicle is secured and tied off, the organ is removed with the least possible delay. A search for other injuries is then carried out; the peritoneal cavity is cleansed and the abdomen closed. The operative mortality in cases

of ruptured spleen is at least 10%; of delayed rupture, 20%. Acute splenic rupture untreated causes death in over 75% of cases.

ECTOPIC SPLEEN

Ectopia of the spleen is a rare condition. It may be congenital, due to anomaly or absence of the supporting peritoneal ligaments, or acquired as a result of trauma which ruptures the phrenicocolic band, or because of splenic enlargement which stretches and abnormally mobilizes the peritoneal folds. A wandering spleen may be found in any part of the abdominal cavity, or it may be contained within a diaphragmatic or external hernial sac.

The important complication of this condition is twisting of the pedicle, which leads to hemorrhagic engorgement, necrosis, rupture, or atrophy. Such a spleen may be palpable and must be kept in mind in the presence of an intra-abdominal tumor.

Treatment.—Complicated or not, the wandering spleen should be removed.

HEREDITARY SPHEROCYTIC ANEMIA

Synonyms.—Chronic acholuric jaundice, congenital or familial hemolytic icterus.

Definition.—A form of hereditary hemolytic anemia transmitted as a simple mendelian dominant, characterized by jaundice, anemia, splenomegaly, spherocytosis, and increased fragility of red cells to hypotonicity. Hemoclastic crises, which are acute exacerbations of the hemolytic process, frequently occur.

Clinical Features.—Symptoms usually develop early in life. The blood of other members of the family may show traits of the disease. Anemia and icterus may be mild or severe; the patient is usually more yellow than sick. The onset may be initiated by an acute hemolytic crisis with pain in the back, abdomen, and limbs, severe malaise, chills, and fever, prostration, and occasionally circulatory collapse, oliguria, and even anuria. If the anemia is severe, dyspnea, palpitation, and other circulatory symptoms are prominent. Hemoglobinemia and hemoglobinuria are very rare. The urine may contain albumin and casts.

Infection, trauma, fatigue, emotional crises,

exposure to cold, and occasionally pregnancy may precipitate an acute hemoclastic crisis.

The indirect plasma bilirubin is elevated, and urobilinogen excretion is increased in the stools and urine. The anemia is only severe during hemoclastic crises. Temporary hypoplasia or aplasia of bone marrow with rapid increase of anemia and decrease of reticulocyte, leukocyte, and platelet counts in the blood without significant increase of jaundice may occur early in an acute hemolytic crisis, especially if the crisis is precipitated by infection. There is a variable reticulocytosis, highest after severe hemolysis. The characteristic red cell is the *spherocyte*, a densely staining cell with decreased diameter and increased thickness as compared with normal erythrocytes. These spherocytes show increased fragility to hypotonic solutions and mechanical tests. The Coombs' antiglobulin test for coating antibodies is usually, but not invariably, negative (see Acquired Hemolytic Anemia). Although usually normal, white cells and platelets may be slightly decreased in number. The bone marrow shows normoblastic hyperplasia. Cholelithiasis occurs in approximately two thirds of cases. Radiologic changes in the bones may be found when anemia and jaundice are of long duration. The spleen is large and firm. Hepatomegaly is frequently observed, and the function of the liver may be impaired. Congenital abnormalities occur with unusual frequency.

Diagnosis.—The various causes of jaundice, fever, anemia, abdominal pain, splenomegaly, and cardiovascular disease must be considered. Cholelithiasis may obscure the primary diagnosis or may precipitate and be hidden by an acute hemolytic crisis.

Complications.—Cholelithiasis and acute hemolytic crises are the principal complications. Infrequently, anuria develops during a hemolytic crisis. Leg ulcers may occur and heal only after splenectomy.

Pathology.—The spleen usually weighs 800-1,500 grams but may be larger. Perisplenic adhesions are common. The cut surface of the spleen is purplish. Malpighian bodies are small and widely separated. Hemosiderosis occurs and, in supravital preparations, increased phagocytosis may be observed. The bone marrow shows a normoblastic hyperplasia.

Pathogenesis.—Most authorities now agree that the pathogenesis of this disease is a hereditary defect in carbohydrate metabolism of the red cells characterized by spherocytosis of adult erythrocytes. Such abnormal red cells are selectively destroyed by the spleen. The mechanism of destruction is unknown. A possible explanation is that the unique structure of the splenic pulp is particularly adapted to the mechanical trapping of spherocytic red cells, in the same manner in which it disposes of aging normal erythrocytes, which tend to become spheroidal.* This specific splenic hemolysis, in which the spleen apparently functions independently of the rest of the reticulo-endothelial system, occurs consistently only in the hereditary variety of spherocytic anemia. Red cells from patients with hereditary spherocytic anemia have a shorter than normal life span, both in their own circulation and when transfused into normal recipients. Their life span is increased both in splenectomized normal recipients and after splenectomy in patients with this disease. Normal red cells transfused into a patient with hereditary spherocytic anemia have a normal life span.

Treatment.—Splenectomy is almost invariably followed by the disappearance of anemia and jaundice. Preferably, splenectomy should precede biliary surgery. Contraction of the spleen prior to its removal may increase the red cell count by half a million or more per cubic millimeter. Leukocytosis occurs but is usually transient. A marked thrombocytosis probably accounts for the high incidence of postoperative thrombotic phenomena. If splenectomy does not achieve the desired result, retention of accessory spleens must be suspected. In some instances, subsequent removal of accessory spleens has resulted in permanent clinical cure. As a rule, spherocytosis and increased fragility are unaffected by splenectomy.

In this disease, preoperative transfusion of whole blood was formerly held to carry with it grave risk of serious reactions. It is our impression, however, that recent advances in

*Whipple and his co-workers have shown, in human subjects suffering from this disease, that the splenic artery contains many times more spherocytes than the splenic vein and that the splenic pulp spaces are packed with these abnormal erythrocytes.

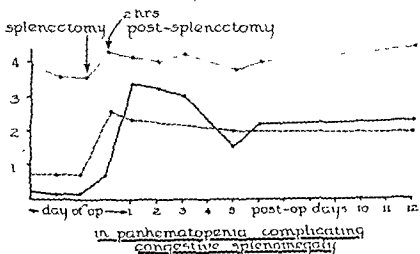
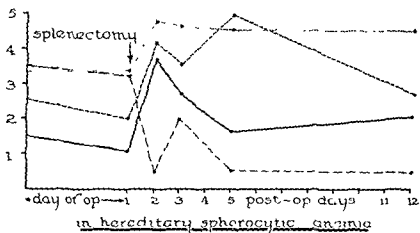
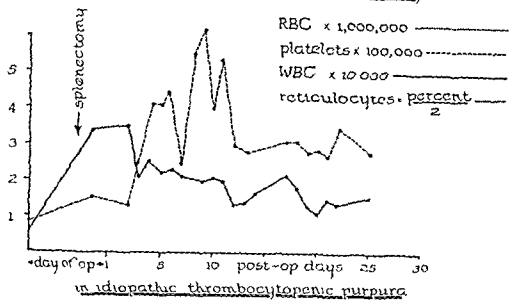
Hematological Effects of Splenectomy

Fig. 128.—Hematologic effects of splenectomy in idiopathic thrombocytopenic purpura, hereditary spherocytic anemia, and splenic panhematopenia in congestive splenomegaly.

blood grouping and transfusion technique have reduced much of this hazard. Few patients with hereditary spherocytic anemia require pre-operative transfusions.

It has been stated that splenectomy is contraindicated during an acute hemoclastic crisis. Obviously, the operative risk is increased during an exacerbation of this disease, but so is the risk of expectant treatment. Current opinion generally supports the view that the results of surgical intervention are considerably better than those of nonsurgical supportive measures. There are occasional cases of congenital hemolytic anemia that are not spherocytic. These patients do not improve following splenectomy and they should be distinguished from those having the spherocytic variety.

IDIOPATHIC THROMBOCYTIC PURPURA (ITP)

Synonyms.—Thrombocytopenic purpura hemorrhagica, Werlhof's disease, thrombocytolytic purpura, hemogenic syndrome

Definition.—A condition of unknown etiology, most commonly occurring in children and in young adult females, characterized by thrombocytopenia and spontaneous bleeding. Spontaneous remissions and exacerbations are characteristic. At times there is a familial tendency. The disease is relatively uncommon in the Negro.

Clinical Features.—The onset may be explosive with gastrointestinal bleeding, cerebral hemorrhage, metrorrhagia, gross hematuria, or bleeding from the site of an extracted tooth. More often, however, it is insidious, with a history of easy bruising, of frequent epistaxis, or a tendency to bleed from the gums. The onset in children is frequently preceded by some acute infectious process. Spontaneous petechiae of the skin and mucous membranes usually occur before massive hemorrhage. Large ecchymotic areas may appear after trivial injuries. Hemarthrosis is rare.

The commonest physical signs are those resulting from hemorrhage. Blood absorption produces fever. The spleen is never markedly enlarged and is palpable in less than one third of cases.

The course of the disease may be fulminating and rapidly fatal, or it may develop in-

sidiously and exist for many years without severe manifestations. Spontaneous remission or recovery is common in childhood, uncommon after the age of 30 years.

Hematology.—Platelets are reduced and bleeding time is prolonged. Coagulation time is normal when determined by the usual methods, but the clot is friable and retracts poorly. However, a coagulation defect may be demonstrated by one of the prolonged clotting-time techniques, by the thromboplastin generation test, or by the prothrombin consumption test. The leukocytes show no consistent abnormality. The degree of thrombocytopenia varies greatly. Bleeding usually occurs if the platelet count is below 60,000. Clinical bleeding, however, does not necessarily parallel the platelet count, especially after splenectomy, and bleeding may be absent with a platelet count of less than 60,000. Prolongation of the bleeding time is usually proportional to the degree of thrombocytopenia. The tourniquet test shows an increased number of petechiae below a blood pressure cuff which has occluded the venous but not the arterial circulation, thus demonstrating the increased capillary fragility. After bleeding into the tissues has taken place, leukocytosis with a neutrophilia and a mild to moderate eosinophilia is not unusual.

The bone marrow findings are of great importance in excluding such conditions as leukemia, hypoplastic anemia, Gaucher's disease, or metastatic carcinoma, in which megakaryocytes are scanty. In ITP megakaryocytes are normal or increased in number. It has been contended that the megakaryocytes exhibit immaturity and impaired platelet proliferation, and that they return to normal, both morphologically and functionally, after splenectomy.

There is now abundant evidence that in many cases of ITP an immunologic mechanism is at work. The serum of a patient with ITP injected into a healthy subject produces a temporary depression of platelets. Some workers have succeeded in demonstrating platelet agglutinins *in vitro*, but the technique is difficult and uncertain and cannot be regarded as a routine laboratory procedure.

In some patients the Coombs' antiglobulin test has been positive, and occasionally there

is evidence also of an autoimmune hemolytic anemia. Thrombocytopenic purpura may be present for some time before an underlying disseminated lupus erythematosus becomes apparent and some apparent cases of ITP probably represent a phase in the course of lupus erythematosus.

Complications.—Bleeding is the chief complication. Its association with pregnancy deserves special mention, for 60 per cent of pregnant women with this disease do not survive. Mortality of the offspring is almost equally high. Over 15% of the children are themselves purpuric. Splenectomy may be performed in pregnancy with resultant survival of the mother.

Diagnosis.—Concealed hemorrhage may be difficult to diagnose. Bleeding into the kidney, the diaphragm, or the gastrointestinal tract may produce the symptoms of an acute surgical abdominal condition. Vaginal bleeding, due to purpura, must be differentiated from other causes. Intracranial hemorrhage may mimic a wide variety of neurologic lesions.

The differential diagnosis of the thrombotic diatheses must be considered. Thrombocytopenic purpura must be distinguished from purpura without thrombocytopenia and also from those conditions in which the thrombocytopenia is a secondary manifestation. A rare and fatal cause of thrombocytopenic purpura is the widespread formation of platelet thrombi in capillaries and arterioles of females (thrombotic thrombocytopenic purpura). Various drugs such as Sedormid, quinidine, arspenamine, and the sulfonamides may produce thrombocytopenia. An antigen-antibody mechanism has been shown to cause the thrombocytopenia produced by Sedormid and quinidine. If the condition is associated with leukopenia or anemia, out of proportion to the amount of blood loss, aplastic or hypoplastic anemia must be considered. If there is a significant degree of anemia, lymphadenopathy, splenomegaly, or hepatomegaly, the possibility of leukemia should be entertained. Liver disease and congestive splenomegaly should be excluded. Carcinomatosis, Gaucher's disease, Hodgkin's disease, and lymphosarcoma involving the bone marrow may induce secondary thrombocytopenic purpura. Allergic thrombocytopenic purpura rarely occurs.

Treatment.—Blood transfusion has often proved lifesaving during the acute phase of the disease. Spontaneous remission may follow this phase, particularly in infancy and childhood. Transfusion is often essential in preoperative and postoperative management. Platelets disintegrate rapidly in ordinary preserved bank blood. If the early phases of coagulation are prevented by careful drawing of blood and by prompt mixing with the anti-coagulant, platelets are preserved for a longer period. Fresh whole blood, platelet-rich plasma, and concentrated platelet suspension prepared by taking blood through plastic tubing into bottles coated with silicone or into plastic bags constitute the most effectual sources of platelets for transfusion in the order given.

ACTH and cortisone have resulted in clinical and hematologic remission for variable periods in this disease. In some instances remission has occurred and has been maintained after withdrawal of the steroid, in others the thrombocytopenia has relapsed after withdrawal, and in others steroid therapy has had no beneficial effect. ACTH and cortisone are often useful in presplenectomy and postsplenectomy management. Their beneficial effect is thought to be due to depression of antibody production and increase of platelet production.

During the first episode of thrombocytopenia, splenectomy should be undertaken only if medical management fails and if severe purpuric manifestations continue after intensive transfusion of platelet-rich blood or plasma. Medical management should be initiated with transfusion of platelet-rich blood or plasma and with the administration of 300 mg of cortisone daily in divided doses, or an equivalent amount of Meticorten. Intravenous ACTH may be used initially in the hope of obtaining more rapid action. Steroid therapy is usually required for a minimum of 2 weeks; as the platelets begin to rise the dosage may be gradually decreased. In mild attacks transfusion alone and careful observation may result in satisfactory remission.

In recurrent or chronic idiopathic thrombocytopenic purpura, splenectomy is indicated if medical management fails to produce improvement within a reasonable period of time. In an acute recurrence the waiting period should be at least 3-4 weeks, whereas in a patient

with mild symptoms the trial period of medical management may be prolonged for 4-6 months. Because idiopathic thrombocytopenic purpura in pregnancy results in a high maternal mortality, pregnancy is a definite indication for splenectomy in the presence of this disease. After splenectomy permanent remission develops in about two thirds of the patients; some of the remaining one third will show a temporary remission which may last weeks or months, and others will develop no remission. Accessory spleens are responsible for no more than one third of the failures. In some instances, clinical bleeding will decrease markedly or disappear after splenectomy, although the thrombocytopenia may persist.

SPLENIC HEMATOPENIA

Definition.—An acute, chronic, or recurrent disease syndrome characterized principally by splenomegaly, normal or overactive bone marrow, and varying degrees of anemia, neutropenia and thrombocytopenia.

The cause of these manifestations is said to be *hypersplenism*. It is thought that the spleen secretes a hormone which depresses maturation of normal cell components of the bone marrow and inhibits their delivery to the circulating blood and that, in addition, excessive destruction of the various cells occurs in the spleen. The syndrome, whatever its cause, is often cured by splenectomy.

Splenic hematopenia has been classified as primary or secondary. In the primary type, no cause is detectable. In the secondary variety, such conditions as Hodgkin's disease, Gaucher's disease, and cirrhosis of the liver with congestive splenomegaly may be etiologically significant.

Clinical Features.—These vary greatly and are largely dependent upon the relative degrees of anemia, neutropenia, and thrombocytopenia. The cellular components of the blood may be decreased singly or in any combination. For example, anemia and neutropenia with normal thrombocytes may occur in one case, in another, thrombocytopenia and neutropenia may be present without anemia. Most commonly, all three components are decreased.

The course of the disease may be acute with high fever, agranulocytic angina, stomati-

tis, oral ulcerations, and occasionally ulcers of the lower extremities. Anemia and purpura may dominate the picture. Lassitude, weakness, low grades of fever, and lowered resistance to infection are characteristic of chronic neutropenia. In some instances, neutropenia may recur in cycles. Occasionally, thrombocytopenia is associated with the menses. Anemia may or may not be hemolytic in character.

Hematologic Findings.—Blood and bone marrow findings are obviously dependent upon which cell type is predominantly involved. If there is a hemolytic reaction, reticulocytosis, increased fragility, spherocytosis of red cells, elevated indirect serum bilirubin, and increased secretion of urobilinogen in stools and urine are common. If there is no acceleration of red cell destruction or excessive bleeding, the anemia is usually normochromic and normocytic, without reticulocytosis or increased hemolysis. An essential diagnostic criterion is an active bone marrow with no abnormal cells.

Treatment.—Splenectomy is the treatment of choice. Blood transfusion may be required, and antibiotics are used to prevent and treat infection in the presence of neutropenia.

CHRONIC CONGESTIVE SPLENOMEGALY

The term splenic anemia was first introduced to describe anemia with splenomegaly not due to leukemia. In 1883 Banti recorded a syndrome of splenomegaly and anemia not associated with leukemia, Hodgkin's disease, malaria, syphilis, hemolytic icterus, or other recognizable pathologic conditions. He noted cirrhotic changes in the liver, a chronic sclerosing endophlebitis of the splenic vein, and typical changes in the spleen itself. It is now recognized that elevation of splenic vein pressure, due to whatever cause, may produce a symptom complex indistinguishable from that previously known as Banti's disease or syndrome and which is now most commonly referred to as *chronic congestive splenomegaly*. The majority believe that the etiology of this condition is obstruction in the portal circulation, which may be either intrahepatic or extrahepatic (see section on Portal Hypertension).

Clinical Features.—Splenic enlargement is usually marked. As noted by Banti, it is associated with anemia of moderate degree, which is normochromic and normocytic, unless complicated by hemorrhage or advanced hepatitis. A mild to marked leukopenia is more constant than anemia. Curiously, the relative percentages of all white cell types is unaffected by this numerical decrease. Thrombocytopenia is usually mild or moderate, but occasionally the platelets drop sufficiently to produce thrombocytopenic purpura. Bleeding from esophageal varices produces a posthemorrhagic anemia and masks typical hematologic findings. If severe liver disease is present, e.g., cirrhosis, a severe disturbance of blood coagulation may either contribute to or be solely responsible for gastrointestinal bleeding, thus the concentrations of prothrombin, proconvertin, proaccelerin, and fibrinogen may be decreased, antithrombin and/or fibrinolysin may be increased, and a variable thrombocytopenia may be present. In the presence of hepatitis the anemia becomes macrocytic. The disease usually appears before the age of 35, although it may develop either in infancy or old age.

PRIMARY NEOPLASMS OF THE SPLEEN

Neoplasms of the spleen are rare. Tumors may arise from the capsule and trabeculae, the lymphoid elements, endothelium, or various reticuloendothelial components. Primary carcinoma of the spleen is unknown. Metastatic carcinoma, especially from the lung and the breast, is present in about 2% of cases. Benign lesions, apart from their mechanical effects, are of little interest or importance. Twenty per cent of primary malignant tumors are lymphosarcomas.

Clinical Features.—The symptoms are those of a tumor in the left hypochondrium, inseparable from the spleen, associated with anemia, cachexia, local pressure effects, and radiologic evidence of displacement of the stomach, colon, and occasionally kidney. There may be ascites and pleural effusion.

Treatment.—Because of the rapid growth and metastasis of such lesions, early splenectomy is the only hopeful treatment. At best, survival seldom exceeds 5 years.

ANEURYSM OF THE SPLENIC ARTERY

Aneurysm of the splenic artery is rarely encountered. Operation has been recorded in about 60 cases, of which 8% were correctly diagnosed before operation. The commonest causes are arteriosclerosis and embolism. Symptoms are usually vague and inconstant. There may be dyspepsia and epigastric pain. A pulsating tumor is rarely palpable. Warning hemorrhage may produce an upper abdominal crisis requiring emergency surgery. Otherwise, fatal rupture is almost invariable. Removal of the spleen together with the involved vessel is generally successful, if undertaken before rupture has occurred. After rupture, the operative mortality thus far approaches 80%.

CYSTIC DISEASE OF THE SPLEEN

Cysts of the spleen may be congenital or acquired. True cysts are dermoid, epidermoid, or endotheloid. False cysts are caused by trauma, infection, or parasitic (hydatid) infestation. They may develop following the degeneration of an infarcted area. The tumor may produce symptoms because of its large size, local pressure effects, or irritation of overlying peritoneum. X-rays aid in diagnosis by demonstrating displacement of adjacent organs. The treatment is splenectomy. Incision and drainage or marsupialization are reserved for complicated hydatid disease, when splenectomy is impossible. Enucleation of the cyst is no longer practiced.

ABSCESS OF THE SPLEEN

Abscess of the spleen is infrequent in temperate climates but may complicate the course of any acute specific infection such as typhoid fever, pyemia, bacterial endocarditis, and parasitic infestations (hydatid disease, malaria, and amebic dysentery). It sometimes follows trauma.

Clinical Features.—The signs and symptoms of splenic abscess are those of left subphrenic suppuration, but they may be delayed until a deeply placed focus has involved the peritoneal coat. Pain in the left upper abdomen, lower chest, and at times in the left supraclavicular

region is continuous, severe, and aggravated by respiratory excursions. Fever is high and associated with chills. The patient is acutely ill, suffers from diarrhea and vomiting, and rapidly loses weight. The abscess may penetrate into the general peritoneal cavity, into the subdiaphragmatic space, and thence into the pleura, into any adjacent abdominal viscus, or outward through the abdominal wall.

Treatment.—Splenectomy, in conjunction with appropriate chemotherapy, is the best form of treatment. In the presence of extensive suppuration, however, one must be content with incision and drainage.

GRANULOMATOUS INFECTIONS OF THE SPLEEN

Splenectomy has been performed for granulomatous infections, such as tuberculosis and syphilis, apparently localized to the spleen. In the light of modern medical therapy, such operations should be rarely, if ever, necessary.

Where endemic, *malaria* is the commonest cause of splenic enlargement, and removal of such spleens was formerly practiced in tropical zones. At present, medical therapy controls the situation in the vast majority of cases. Emergency surgery, however, is not infrequently required because of rupture or torsion of a large malarial spleen. The very large spleen frequently encountered in kala-azar may produce the clinical picture of hypersplenism, which is relieved by splenectomy.

Egyptian splenomegaly affords a classic example of Banti's syndrome produced by increased portal venous pressure. In this instance, the obstructive factor is intrahepatic, caused by the ova of *Schistosoma mansoni* and the cirrhotic changes they induce. Splenic enlargement is of the congestive type, and associated blood changes are those of hypersplenism. Though not curative, splenectomy may delay the advance of this disease which, untreated, progresses to liver atrophy, ascites, and death usually within 4 years of onset.

GAUCHER'S DISEASE

Definition.—A chronic, familial disorder characterized by splenomegaly, hepatomegaly, and skeletal defects caused by accumulations

of large pale cells (Gaucher's cells) containing the cerebroside kerosin.

Although familial, the disease rarely affects more than one generation and is most common among Jews. When the onset is within the first 6 months of life, death usually results before the end of the second year. Over half of the adult forms began in childhood.

Clinical Features.—Symptoms are chiefly related to bone involvement and to the mechanical disturbance caused by an enormous spleen. Invasion of the bone marrow by Gaucher's cells may cause such radiologic changes as decalcification, compensatory sclerosis, and pathologic fractures. Femora, vertebrae, and sternum are most frequently involved. The enlarged spleen may fill the abdomen. Being prone to infarction, it is frequently painful. The head, neck, and extremities often develop a brownish pigmentation. Wedge-shaped pingueculae and brownish pigmentation of the sclerae and conjunctivae may suggest the diagnosis.

Although the hematologic findings are to some extent those of secondary hypersplenism, their essential cause is the replacement of normal bone marrow by Gaucher's cells. Unless thrombocytopenia causes hemorrhage, the anemia is normochromic and normocytic. Occasionally, it may be macrocytic due to the presence of nucleated red cells and reticulocytes. Thrombocytopenic purpura develops in over half of the cases. Leukopenia and neutropenia are frequent and are usually associated with a relative lymphocytosis.

The diagnosis is established by marrow or splenic aspiration yielding the large, pale, kerosin-containing reticulum cells of Gaucher which, with Mallory's aniline blue stain, show numerous spiderlike fibrillae.

Treatment.—There is no curative treatment. However, such patients suffer from the effects of hypersplenism as well as from the encumbrance of a massive intra-abdominal tumor, and these symptoms may be alleviated by splenectomy.

ACQUIRED HEMOLYTIC ANEMIA

Definition.—Acquired hemolytic anemia may be idiopathic, or it may occur in association with a variety of *granulomatous*, pyrogenic, or enteric infections, with Hodgkin's

disease, lymphosarcoma, reticulum cell sarcoma, leukemia, Gaucher's disease, schistosomiasis, ovarian cysts, and carcinomatosis.

Clinical Features.—If not idiopathic, the hemolytic anemia may be masked by the symptoms and signs of the primary disease. Its onset may be rapid and quickly fatal, or it may be a chronic affair, with prolonged anemia and jaundice, recurring remissions, and exacerbations. In contrast to hereditary hemolytic icterus, the patient is often more sick than jaundiced. Anemia and jaundice, with elevation of the indirect plasma bilirubin and increased output of fecal and urinary urobilinogen, are present in varying degrees, depending on the rate of hemolysis. These classic signs of hemolytic anemia may be lacking in the presence of fairly brisk hemolysis. Red cell survival measured with radiochromium-tagged red cells will sometimes show the presence of hemolysis when the usual signs are absent. The red cells exhibit increased fragility to hypotonicity and in about one half of the cases may have, in addition, lowered resistance to mechanical agitation. The anemia is usually normocytic but may be macrocytic. Spherocytosis is present in some cases. Reticulocytosis, polychromatophilia, basophilia, and basophilic stippling reflect increased erythropoiesis. Autoagglutinins, pathologic cold agglutinins, and atypical agglutinins, most commonly warm, and incomplete antibodies are often demonstrable. The Coombs' test for coating antibodies is usually positive in idiopathic acquired hemolytic anemia. It is almost always negative in hereditary spherocytic anemia and, consequently, is useful in differentiating the two types. The test usually remains positive after splenectomy. The life span of normal red cells is shortened when they are transfused into a patient with this condition, and red cells from a patient with acquired hemolytic anemia survive normally when transfused into a normal person. These findings confirm the presence of a hemolytic factor in the plasma of patients with acquired hemolytic anemia and exclude the presence of an inherent defect of the red cell.

Treatment.—Treatment of idiopathic acquired hemolytic anemia is usually medical at the outset. Blood transfusions and steroid

hormone therapy are the mainstays. Whether or not the spleen should be removed is a difficult decision, and the criteria for selection of cases are not at present well defined. The results are uncertain. About 50% of splenectomized patients may be expected to respond fairly well to the operation. However, relapse may occur sooner or later. The ultimate decision is usually based on failure of medical treatment. Recently selection of cases for splenectomy has been helped by determining the localization of transfused red cells in the spleen when these cells were tagged with radioactive chromium.

Multiple blood transfusions, so frequently required in these patients, may lead to the development of new isoimmune antibodies and the occurrence of transfusion reactions. Because of this, the blood transfusion laboratory should employ the technique of high protein and indirect Coombs' cross-matching. The use of packed red cells or saline-washed red cells may be necessary to avoid transfusion reactions.

If there is an underlying disease causing the hemolytic anemia, this should be treated in the appropriate way. Occasionally, lymphocytic leukemia or lymphosarcoma may present a severe hemolytic anemia with a huge spleen which makes management difficult because of the requirement for frequent blood transfusions. Splenectomy may be very helpful in such patients.

SPLENOMEGALY OF INDETERMINABLE ETIOLOGY

Enlargement of the spleen, sufficient to be palpated beneath the costal margin, is noted on occasions without demonstrable cause. Blood and bone marrow changes are absent. There is no general or localized lymphadenopathy. Portal venous pressure is normal. Signs of bacterial infection or parasitic infestation are lacking. There is no gross metabolic disturbance.

In such cases, the presence of some obscure pathologic process, localized to the spleen, cannot be excluded. For this reason, it is the opinion of some surgeons that splenectomy for splenomegaly alone deserves serious consideration.

SUPPLEMENTARY SPLENECTOMY

Removal of the normal spleen may greatly facilitate major surgical procedures on the upper abdomen and thorax and at the same time render these operations more effectual, particularly when their purpose is the eradication of malignant disease. Block resection of the spleen, the greater and gastrohepatic omenta, together with the stomach, increases the scope of radical operation for gastric cancer. Esophagoduodenal anastomosis is performed much more easily and safely in the absence of the spleen. The same applies to abdominal repairs of large diaphragmatic hernias, with or without incarceration of the spleen.

TECHNIQUE OF SPLENECTOMY

When the spleen is sufficiently mobile to be lifted out of the abdominal incision, its removal is a technically simple and safe procedure. When it is bound down by dense, vascular adhesions, splenectomy may have to be abandoned and the operation consist of some palliative procedure such as ligation of the splenic artery or division of as much of the vascular pedicle as can be safely reached.

Preoperative Measures—Facilities for blood transfusion should be available. The stomach is intubated and kept deflated throughout the procedure to permit better exposure. Optimum posture is obtained with an air cushion supporting the lower left ribs and flank and the table tilted slightly to the right.

Operative Incision.—Most surgeons prefer a long, left paramedian, epigastric incision. However, in some hands the curved, subcostal approach has been highly satisfactory, especially when the spleen is greatly enlarged and the subcostal angle markedly widened. In cases of suspected rupture, a midline epigastric incision may be advisable. It can be opened and closed more swiftly than the others and affords easier access to the right upper quadrant. The abdomino-thoracic approach, combining a transverse or left paramedian epigastric incision with an extension through the bed of the 8th rib or 8th intercostal space and the diaphragm, has been recommended for difficult splenectomies. Thoracotomy, however, increases the operative risk.

Removal of the Spleen.—As soon as the abdomen has been opened, thorough exploration must be carried out. Injury to other organs must be excluded in accident cases, complicating regional extensions in inflammatory disease, metastases in neoplasms, and calculous biliary disease in spherocytic jaundice. In this last condition and in thrombocytopenic purpura, accessory spleens must be removed. In congestive states, the site of portal obstruction should be accurately determined.

When the spleen is intact, free from adhesions, and not too large, the following procedure is recommended. The lower two thirds of the gastrosplenic ligament is divided between clamps. The spleen is tilted medially, the costal margin retracted, and the lienorenal ligament, together with its underlying areolar tissue, incised. The stomach and spleen are drawn downward and to the right to fully expose the very short upper third of the gastrosplenic ligament, which is then safely clamped and divided. When the avascular phrenicocolic band is cut, the spleen is fully mobilized and may be lifted into the incision. The tail of the pancreas is carefully dissected away from the lower posterior portion of the pedicle. The splenic artery is divided between heavy ligatures. After a brief interval, to permit collapse of the spleen, the veins are individually ligated and divided and the specimen is removed. If the splenic reservoir is large, its evacuation may be hastened by the injection of a few minims of adrenalin into the artery just before it is tied. The pedicle stump is peritonealized and the roof of the lesser sac is reconstructed with a few interrupted sutures. The abdominal incision is closed in layers. Drainage is indicated in the presence of persistent bleeding, injury to the pancreas or established infection.

Perisplenic adhesions may be delicate and relatively bloodless, or dense and vascular. The former yield, as a rule safely, to manual exploration, but the latter have called a halt to many splenectomies. To cope with this type, the use of high-frequency current has been recommended.

As a prelude to difficult splenectomies, it is sound policy to ligate the *splenic artery*. The vessel may be approached via the gastrohepatic omentum and tied close to its origin, or

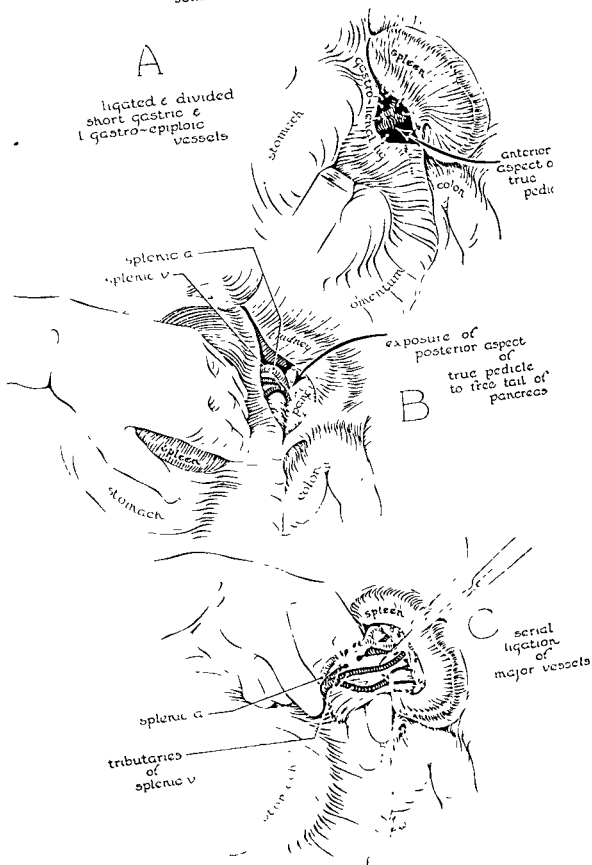


Fig 329—Technique of splenectomy

through the gastrocolic omentum and secured where it parallels the superior border of the pancreas, several centimeters from the pedicle. Such a step will lessen blood loss and permit the patient to transfuse himself with the reservoir content of his spleen. Some feel that splenic artery ligation per se affords considerable palliation in cases of advanced cirrhosis that will not tolerate major vascular shunts. This we doubt (see Portal Hypertension).

Complications of Splenectomy

The complications of splenectomy may be the result of errors in technique, errors in judgment, and other causes relevant to certain types of splenopathy.

Technical errors may lead to postoperative hemorrhage, damage to adjacent viscera with subsequent perforation, peritonitis, and fistula formation, infection consequent to local contamination and inadequate hemostasis, and wound disruption because of infection or faulty suture technique. The operation may be useless because of failure to remove accessory spleens. Expert hematologic advice is the only safeguard against errors in the selection of cases for splenectomy.

In certain splenopathies, various postoperative complications are more commonly recorded. The removal of large spleens predisposes to pulmonary collapse and pneumonia. Temporary paralysis of the diaphragm occurs frequently. While it is often stated that there is an increased incidence of postoperative thrombosis following splenectomy, it is our feeling that this risk has been overrated. This is true in spite of the thrombocytosis which occurs. Debilitated individuals with congested, adherent spleens are particularly liable to develop left subdiaphragmatic suppuration. Hemorrhagic diatheses and malnutrition delay wound healing and pave the way for disruption. Closure therefore must be especially meticulous. Nonabsorbable suture material is preferable to catgut.

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SURGERY OF THE SPLEEN

Title	Film References		Sound or Silent	Procureable From
	Running Time			
Splenectomy for Congenital Hemolytic Anemia (Illustrates the technique of removing a large spleen and an accessory spleen by the conventional route) (1951) (By C Stuart Welch, M.D., Albany)	28 min		Silent Color	American Cyanamid Co. Surgical Products Division Danbury, Conn
Splenectomy in the Treatment of Hypersplenism (Preliminary classification and descriptions of the disease entities of hypersplenism are followed by a clearly visualized presentation of the splenectomy operation) (1951) (By Robert M Zollinger, M.D., and Edwin H Ellison, M.D., Columbus)	20 min		Silent	Robert M. Zollinger, M.D. University Hospital Columbus 10, Ohio

Chapter 23

Peritoneum, Omenta, and Mesenteries

James R. McCorriston, MD.

INTRODUCTION

The peritoneum is the lining of the abdominal cavity. It is a serous membrane, derived from the mesenchyme, with a surface area approximately that of the skin. Since the peritoneum is in intimate contact with the intra-abdominal organs, the retroperitoneal tissues, the diaphragm, and the abdominal parietes, it is not surprising that it is involved in a wide variety of disease processes, the majority of which have their origin in other tissues and organs.

EMBRYOLOGY

In the embryo, after the somatopleure and the splanchnopleure develop, the mesoderm splits laterally into the dorsal somatic mesoderm and the ventral splanchnic mesoderm. The cavity thus formed is the body cavity or celom which eventually is divided into the pericardial, pleural, and peritoneal cavities. Variations may occur in the development of the peritoneum and mesenteries, but these are so numerous that no attempt will be made to describe each one separately. It is sufficient to realize that variations can and do occur with surprising frequency. Certain variations may be present throughout life without causing symptoms, whereas others may lead to serious conditions such as internal hernia, volvulus, or intestinal obstruction.

ANATOMY

In the male the peritoneum forms a closed sac, but in the female the free ends of the fallopian tubes open into the peritoneal cavity. The mesothelial cells which form the peritoneal surface are flattened and polygonal, separated by cement substance. This layer of cells rests on a thin layer of fibrous tissue which is connected to underlying fascia or viscera by areolar tissue.

The continuous peritoneal membrane is invaginated by the hollow and solid viscera. This portion constitutes the visceral peritoneum, while the remainder is called the parietal peritoneum. The mesenteries of the bowel are formed by the two layers of invaginated peritoneum, and the greater omentum is an extensive fold hanging from the greater curvature of the stomach. The peritoneal cavity is between the parietal and visceral peritoneum and contains, normally, only a small amount of serous fluid which lubricates its lining. The heavy folds of peritoneum attached to solid organs constitute their ligaments. The blood vessels, nerves, and lymphatic vessels are important additional supports, especially in the mesenteries of the bowels.

Anatomically, the peritoneal cavity is divided into two major compartments, the greater and lesser sacs, which communicate by means of the epiploic foramen (foramen of Winslow).

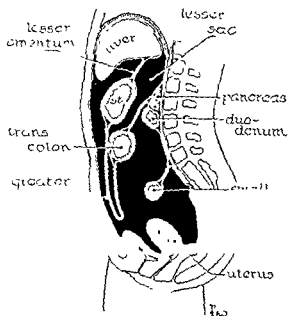


Fig. 330—The peritoneum (median sagittal section). This drawing illustrates diagrammatically the relationships of the omenta, transverse mesocolon, and mesentery of the small intestine.

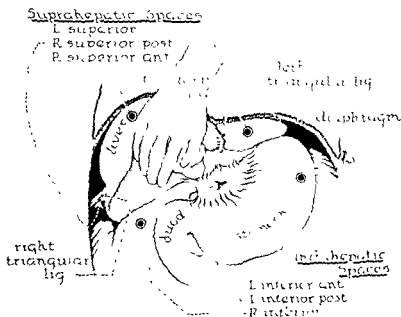


Fig. 331—The subphrenic spaces. The six subphrenic spaces are shown from the anterior aspect. The right lobe of the liver is drawn medially and rotated upward.

From a surgical point of view, there are four main divisions: (1) supracolic, (2) right infracolic, (3) left infracolic, and (4) pelvic.

Surgically, the supracolic subdivision may be looked upon as the *subphrenic space*, as it lies between the diaphragm above and the transverse colon and its mesocolon below. This region is divided by the liver into suprahepatic and infrahepatic portions. Three suprahepatic and three infrahepatic spaces exist. The falciform ligament divides the suprahepatic space into right and left parts. On the right side the right triangular ligament of the liver separates a large anterior space from a small posterior space. The left triangular ligament extends along the posterior border of the left lobe of the liver, separating the superior from the inferior surface. Therefore only one space exists to the left of the falciform ligament.

The three suprahepatic spaces are named: (1) right superior anterior subphrenic space, (2) right superior posterior subphrenic space, and (3) left superior subphrenic space.

The infrahepatic region is divided into right and left parts by the second (descending) part of the duodenum. To the right of the duodenum and below the liver is the space often called Morison's pouch. To the left of the duodenum the space is divided into anterior and posterior parts by the stomach and lesser omentum.

The three infrahepatic spaces are named: (1) right inferior subphrenic space (Morison's pouch), (2) left inferior anterior subphrenic space, and (3) left inferior posterior subphrenic space (lesser sac).

These six spaces are all intraperitoneal, but an extraperitoneal space exists which corresponds to the bare area of the liver.

The right infracolic is separated from the left infracolic subdivision of the peritoneal cavity by the mesentery of the small bowel. The right and left paracolic gutters are important because they provide routes for the ascent and descent of exudate or pus. The pelvic subdivision is within the confines of the true bony pelvis.

glide freely over each other and parietal peritoneum. The peritoneum marked capacity to absorb both small particles. Fluids that are not with body fluids are usually rendered before absorption takes place; then of absorption may be rapid. The capillary portal system are extremely permeable to protein molecules. Absorption of α -globulins and of crystalloids by capillaries is rapid and complete. Macrophages, blood cells and particulate matter, the absorptive power of the greater omentum is enhanced by its mobility.

Under conditions of stress the cells are probably capable of giving rise to macrophage cells. In any event the peritoneum has, at times, enormous powers of resistance to infection, which is exemplified by the fact that pure cultures of pathogenic bacteria introduced into the peritoneal cavity usually produce peritonitis, unless there is some traumatization or the introduction of some material. It has been shown that the capacity of absorption and resistance are approximately equal in all parts of the peritoneum. It is probable that the peritoneum reacts in the presence of an infective process. Although, grossly, there may be localization of infection, diffuse or generalized peritonitis, one area of the peritoneum is involved. The whole peritoneum pours forth an exuberant defensive effort.

The sensitivity of the peritoneum to the disease process is clinically important. The peritoneum has few or no nerves, the bowel wall is supplied by the parasympathetic (Meissner and Auerbach) (of the autonomic nervous system), while the parietal peritoneum is supplied by fibers of the somatic nervous system. The wall of the intestine, when burned, crushed, or cut without injury, is painless, but traction or tension on many parts of the peritoneum is painful. The parietal peritoneum reacts to trauma as do other parts of the body supplied by cerebrospinal nerve fibers. The peritoneum which does not have such

forms in the wound it is covered with mesothelium, and scarring is minimal. In the presence of severe trauma or infection, repair is by second intention; scar tissue is greater in amount and adhesions or bands, which are often dangerous, may form. Such adhesions are primarily beneficial since they splint the parts, prevent or control leakage of exudate, and permit complete healing. Once the acute phase is past, these adhesions tend to stretch, ultimately lose their blood supply, and are absorbed. Adhesions which develop as a consequence of inflammatory processes have frequently disappeared by the time of a later abdominal operation. Although adhesions are necessary for repair, if there is much tissue trauma or extensive peritonitis they may become dense, are not absorbed, and may lead to intestinal obstruction or lesser degrees of interference with function.

Mechanical and chemical injuries to the peritoneum are the commonest types. The injury may also initiate the reaction known as inflammation. If there is a peritoneal wound or tissue death, the process of repair will also be initiated.

The peritoneal fluid is normally small in amount, clear, and viscid. It contains cells, over 90% of which are large mononuclear, polymorphonuclear, and basophilic cells.

When an irritant is placed in the peritoneal cavity, polymorphonuclear leukocytes flood the region and become the predominant cells. Within a few hours mononuclear macrophages appear in large numbers in the increasing volume of peritoneal fluid which also contains fibrin. Foreign particles or bacteria are caught in the meshes of clotted fibrin, and phagocytosis occurs. As the days pass, the cells degenerate and, together with their phagocytosed contents, disappear. After several days, when the process subsides, the peritoneal fluid returns to normal. The preceding description is that of mild peritonitis which results in resolution, but all grades of severity of the reaction can occur, and if the infection is very intense, death may result from toxic absorption, dehydration, electrolyte imbalance, and other secondary factors. The inflammatory response of the peritoneum is shared by the underlying and surrounding tissues.

When the peritoneum is divided or devitalized, repair follows. The process of inflammation is set up, and fibrinous tissue fluid causes adhesions to form, which are at first fibrinous but which may later become fibrous. The degree of development of adhesions depends upon many factors, not the least of which is the inherent tendency of certain individuals to form adhesions. The factors that influence the formation and disappearance of adhesions are not understood, so that various methods for their prevention have rarely proved successful. Gentle operative technique, removal of blood and foreign materials, and reduction of bacterial contamination to a minimum are the best means of avoiding persistent adhesions.

ACUTE PERITONITIS

Peritonitis is the term that denotes any inflammatory process in the peritoneum, regardless of its extent or severity. Peritonitis may be *bacterial* or *chemical*, and it varies in extent from localized to generalized. Non-bacterial peritonitis follows the introduction of sterile traumatizing or irritating substances into the peritoneal cavity. Body fluids such as bile or gastric juice, clotted blood, gauze, sulfonamide crystals, surgical instruments, talcum powder, and antiseptics all produce peritonitis with signs and symptoms indistinguishable from those of bacterial infection. More severe grades of peritonitis are caused by bacteria which are usually of intestinal origin. Pyogenic peritonitis is generally caused by one or a combination of two or more of the following bacteria: coliform bacteria, staphylococci, streptococci. Other organisms are frequently found, particularly anaerobes (e.g., gas-forming pathogens). Many of these organisms produce powerful toxins which seem to be responsible for the severe toxemia often found and which are more important as a cause of death than bacteremia.

SECONDARY PERITONITIS

Acute bacterial peritonitis follows direct contamination when bacteria reach the peritoneum secondarily from an intra-abdominal viscus, some adjacent tissue or organ, or through an abdominal wound.

Locally, in bacterial peritonitis, the peritoneum soon becomes reddened and edematous, loses its normal sheen, and is covered with fibrinopurulent exudate and later pus. Such an exudate, bathing the bowel wall, causes decrease or loss of peristalsis, leading to distention. The greater omentum may be successful in limiting the process to a localized peritonitis (or abscess), but if localization does not result, diffuse or generalized peritonitis ensues. When a relatively large focus of peritonitis is suddenly set up, absorption of toxic materials occurs over an extensive surface. The route of spread depends upon local anatomic structures such as the paracolic gutters, gravitational spread into the pelvis, or the watershed effect of the mesentery of the small bowel.

It has been pointed out that the surface area of the peritoneum is approximately that of the skin, and widespread peritonitis is, therefore, comparable to a very extensive cutaneous burn. Many general effects result from severe widespread peritonitis.

1. Edema of the inflamed tissues pools body fluid which is initially at the expense of the extracellular fluid and later depletes the intracellular fluid. This is actually the creation of a third fluid space in addition to the intracellular and extracellular spaces. As a result there are hemoconcentration, hypoproteinemia, hypochloremia, and reduction of circulating blood volume. This, in turn, produces a degree of stagnant anoxemia.

2. Bacteremia, toxemia, and hemoconcentration all destroy erythrocytes, producing anemia.

3. Peritoneal exudate and edema of the bowel wall inhibit peristalsis and so cause paralytic ileus. The concomitant distention exerts upward pressure on the diaphragm and secondarily interferes with pulmonary function.

4. When obstruction of the bowel is caused by paralytic ileus, vomiting occurs, accelerating the process of dehydration.

5. Dehydration and associated factors interfere with renal function, resulting in azotemia and oliguria or anuria.

All of these effects vary in degree of severity according to the suddenness of the inflammation and the treatment provided.

Diagnosis.—In the diagnosis of peritonitis, its presence, and then its source, must be determined. Initially, the symptoms and signs of peritonitis may not be distinguishable from those of the intra-abdominal lesion causing it. The fever is usually high, particularly so in children. Only in overwhelming infections is shock present and the temperature subnormal. The white blood cell count is high (e.g., 20,000-30,000/c.mm.) with over 80% polymorphonuclear leukocytes present. The patient is distressed and concerned about his illness. He adopts a position in bed which reduces intra-abdominal tension to a minimum, i.e., his knees and hips are flexed and he breathes rapidly and shallowly. He tends to lie quite still, for any movement causes an increase in discomfort owing to stretching or moving of the inflamed peritoneum and mesenteries. Examination of the abdomen will show some degree of distention, increased tone of abdominal muscles, and local or generalized tenderness with or without rebound pain. In the presence of acute peritonitis, especially when it is localized, the tenderness is usually pronounced. Tenderness, elicited by means of rectal or vaginal examination, is acute in cases of pelvic and generalized peritonitis. In the later stages of severe peritonitis marked abdominal distention, vomiting, oliguria, coated tongue, foul breath, spiking fever, restlessness with picking at the bed coverings, delirium, and finally coma occur. X-ray films of the abdomen will reveal dilated loops of bowel with fluid levels.

Conditions which may be confused with peritonitis, particularly early acute peritonitis, include pneumonia, pleurisy, influenza, renal colic, retroperitoneal hemorrhage, and retroperitoneal tumors. Once the presence of peritonitis is diagnosed, there follows the task of determining its source. Sources of peritonitis are discussed elsewhere under the various intra-abdominal lesions.

Treatment.—The treatment of peritonitis includes the following:

1. Prevention by means of prompt attention to its cause.
2. Elimination of the focus of infection.
3. Prevention and treatment of complications.

4. Inhibition or destruction of the bacteria with chemotherapeutic and antibiotic agents when possible

5. Provision of early drainage of abscesses Although it is at times advisable to postpone operative treatment until the general condition of a very ill patient is improved, removal or drainage of the focus of infection is very important. Closure of a perforated peptic ulcer and removal of an inflamed appendix are examples of procedures used in this connection which should be carried out with minimal delay

Complications of peritonitis may be both numerous and dangerous and may lead to the death of the patient besides prolonging the disease. Toxemia, ileus, pneumonia, actual organic bowel obstruction, and septicemia are examples of grave complications of peritonitis

Chemotherapeutic agents are employed to great advantage in the prevention and treatment of peritonitis, and their use does not interfere with other methods of treatment. When peritonitis is caused by an organism or organisms sensitive to these agents, they become indispensable. Vaccines are of no practical value

In all cases of peritonitis close attention must be paid to the maintenance of fluid and electrolyte balance and the provision of blood transfusions when indicated. The intestines are kept at rest by means of continuous nasogastric or intestinal tube suction; morphine is used to allay fear, anxiety, and pain, and nothing is permitted by mouth. Morphine diminishes peristalsis of the gut and simultaneously increases the tone of its muscle. Patency of the suction tube may be assured by irrigating it every 2 or 4 hours with 30 ml of physiologic saline solution, although this is not always necessary. Because production and absorption of vitamins in the alimentary tract are seriously interfered with in peritonitis, and because ordinary parenteral fluids contain no vitamins, parenteral vitamins in high dosage must be given daily

PRIMARY PERITONITIS

Primary peritonitis is rare and may be looked upon as metastatic peritonitis, since the bacteria reach the peritoneum indirectly from an outside focus of infection. It may begin after

pathogenic organisms reach the peritoneum via the fallopian tubes or via the lymphatics, but the common route is the blood stream. Organisms can usually be found in the blood in these cases, and often a respiratory infection is present beforehand. The lesions are typical of generalized peritonitis, the type of exudate present depending upon the causative organism. Primary peritonitis is usually caused by the streptococcus or pneumococcus. A less common causative organism is Friedländer's bacillus.

Pneumococcal Peritonitis.—Pneumococci reach the peritoneum via the blood stream from a focus in the respiratory tract, the accessory nasal sinuses, or the middle ear, or from the genital tract of female infants or children through the fallopian tubes. The pneumococcus causes acute peritonitis, and a thick, whitish exudate is formed. Cultures of peritoneal exudate yield pure pneumococcus. Characteristically the fever is quite high and may rise to 104° F. The leukocyte count is usually very high, often in the neighborhood of 25,000-30,000/c mm

Streptococcal Peritonitis.—This form of metastatic peritonitis leads to the production of a serosanguineous exudate which, on culture, yields pure streptococcus.

Acute primary peritonitis produces signs and symptoms of generalized peritonitis without localization. Foci of infection leading to primary peritonitis are hard to differentiate and may coexist. For this reason surgical exploration is not infrequently indicated as soon as the patient's general condition permits. When the peritoneal cavity is opened, a culture and direct smear of the exudate should be made. If pure streptococcus or pneumococcus is found on examination of the smear, the abdominal wound should be closed without further traumatizing exploration. Treatment thereafter is conservative, consisting of general supportive and symptomatic measures combined with chemotherapy guided by sensitivity tests performed on the cultured causative organism.

LOCALIZED ABSCESSES

Acute bacterial peritonitis tends either to resolve completely or to localize and form one

or more abscesses. An abscess, in turn, may undergo resolution and may point toward the surface of the body and discharge its contents or toward an abdominal viscus (e.g., rectum). At times such an abscess may become dormant for long periods of time, later resolving or flaring up to produce further tissue destruction. Not infrequently diffuse or generalized peritonitis becomes loculated so that multiple abscesses develop.

Abscesses develop in certain sites which warrant separate consideration since the conditions leading to them and their treatment differ.

Subphrenic Abscess.—In the section on Anatomy, the subphrenic spaces were considered in detail because of their importance in respect to abscesses which may develop within them. Subphrenic abscess is often difficult to diagnose because of the paucity of local



Fig 332—Subphrenic abscess in right superior anterior space. This is a single x ray film of the abdomen, with the patient erect, after the introduction of 1,500 ml of oxygen into the peritoneal cavity. The right half of the diaphragm is elevated, inferior to it can be seen the gas shadow and fluid of inflammatory pneumoperitoneum to a lower level.

physical signs, although general signs and symptoms of severe infection may be present, with spiking fever, severe malaise, high leukocyte count, and secondary anemia. When the superior subphrenic spaces and the bare area of the liver are sites of abscess formation, signs and symptoms of pleural and pulmonary involvement are usually present, whereas, if the inferior subphrenic spaces are involved, abdominal signs and symptoms predominate. A right superior anterior subphrenic space abscess, for example, often leads to pleural irritation, with the development of a pleural exudate and secondary lower lobe atelectasis. The abscess may penetrate the diaphragm and pleura, producing empyema and lung abscess. A most important diagnostic aid in detecting or confirming the presence of a superior subphrenic space abscess is the use of fluoroscopy and x-ray films, with or without induced pneumoperitoneum. Owing to the presence of the subphrenic abscess, the involved hemidiaphragm is elevated and relatively motionless on respiration, and the abscess may throw a soft-tissue shadow or displace the liver downward. When gas-forming organisms are present in the abscess, a fluid level will be seen with an overlying bubble of gas. When gas cannot be seen on x-ray examination, the failure of oxygen in an induced pneumoperitoneum to enter the superior subphrenic space is evidence of abnormal adherence of the serosa of the liver to the parietal peritoneum over the diaphragm. When doubt as to the diagnosis and site of a subphrenic abscess exists, it is at times helpful to attempt aspiration of pus by means of a syringe and wide-bore needle. Great care must be taken to avoid contamination of the pleural space or perforation of bowel during this maneuver.

Inferior subphrenic abscesses, if large, may be detected as abdominal masses, and x-ray examination (particularly with the aid of barium) may demonstrate displacement of the stomach or duodenum from its normal position.

Although the subphrenic spaces may become infected by organisms carried by the blood stream, the usual means of infection is the introduction of infected material from an

abdominal viscus (e.g., perforated peptic ulcer, gall bladder, or appendix; bowel surgery; suppurative pancreatitis).

Pelvic Abscess.—Any type of bacterial pelvic peritonitis may lead to the formation of a pelvic abscess, although it may also result from the gravitational spread of infected material from any site in the abdomen. Pelvic abscess develops most commonly following pelvic inflammatory disease in the female, suppurative appendicitis, and infective lesions of the large bowel. In addition to the general signs and symptoms of infection, there are definite local signs and symptoms which permit the diagnosis to be made with certainty. Owing to irritation of the pelvic colon and rectum there is usually diarrhea, often accompanied by the passage of mucus in large amounts. Rectal examination will reveal the presence of a boggy, tender mass pressing against the rectum. If it is large, abdominal examination may reveal a tender mass rising out of the pelvis. Often a pelvic mass causes irritation of the bladder, with symptoms of dysuria and frequency. In the female, pelvic examination will add to the information gained by rectal examination.

Paracolic Abscesses.—Abscesses develop frequently enough in the paracolic gutters and in the iliac fossae to warrant separate mention. On the right side, lesions of the appendix and right colon and, on the left, lesions of the pelvic colon are the common starting points of paracolic abscesses.

Treatment of Localized Abscesses.—In addition to conservative supportive measures and chemotherapy, localized abscesses require operative drainage in most instances. The indications for operative drainage include progression of the abscess, spread, pressure effects (e.g., pressure on the common bile duct by a right inferior subphrenic abscess), septicemia, intestinal obstruction, and failure to respond to conservative measures. In certain instances concomitant operative treatment of a causative lesion (e.g., previously undiagnosed perforated peptic ulcer) is necessary.

The surgical approach for drainage of right superior anterior subphrenic abscess is via a right subcostal incision with care to avoid opening the uninvolved peritoneal cavity. The

right superior posterior subphrenic space may be drained by means of an extrapleural approach, through the bed of the 11th or 12th rib. The left superior and inferior anterior subphrenic spaces may be drained through a left subcostal incision. The right inferior subphrenic space may be approached through a high right paramedian abdominal or subcostal incision, taking advantage, when possible, of previous postoperative adhesions to avoid contaminating the remainder of the peritoneal cavity. The left inferior posterior space (lesser sac) may be drained transperitoneally or retroperitoneally by means of an incision through the bed of the left 12th rib.

A pelvic abscess can usually be drained through the rectum or vagina, although it may be necessary to employ an abdominal route.

Right and left paracolic abscesses may be drained by means of incisions directly over them, with care to avoid opening the uninvolved peritoneal cavity.

The contents of abscesses should always be cultured and chemotherapy guided by the type of organism present and its sensitivity to particular antibiotic agents. At times it is of great value to irrigate an abscess cavity at intervals with a suitable antibiotic in solution. When it is not feasible to provide dependent drainage, it is desirable to posture the patient to attain this end.

SPECIAL FORMS OF PERITONITIS

Tuberculous Peritonitis

Tuberculous peritonitis is more frequent in females than in males and in the young than in the aged. Frequently it is one manifestation of miliary disease but may take origin from an abdominal organ. It often originates in the fallopian tubes and in the appendix. There are two chief forms of tuberculous peritonitis.

1. The *exudative type* is a slowly developing disease which leads to the formation of ascites and small, gray tubercles studding the peritoneum.

2. The *proliferative or dry type* often goes on to the formation of areas of caseation and fistulous connections between loops of bowel. External fistulas are likely to follow surgical procedures. Much granulation tissue develops, forming matted masses in the abdomen, par-

ticularly in the pelvis. If tuberculous peritonitis is not associated with widespread tuberculosis elsewhere, the prognosis is fairly good.

Diagnosis.—In tuberculous peritonitis the abdomen is distended but not markedly tender on palpation. Frequently the abdomen has a doughlike consistency, and a mass can be palpated owing to the rolling up of the greater omentum which often occurs. The onset is usually gradual or it may be abrupt, with signs and symptoms very similar to those of acute pyogenic peritonitis (see above). There are usually low-grade fever, general malaise, loss of weight, and some abdominal discomfort with a feeling of fullness. The presence of tuberculosis elsewhere in the body is a common finding. The white blood cell count tends to be normal or only slightly elevated, with a relative lymphocytosis and monocytosis.

Treatment.—The basis of treatment is general supportive measures and rest as for tuberculosis elsewhere. Streptomycin, para-aminosalicylic acid and isoniazid compounds have been used with success. Foci of infection, such as the appendix or fallopian tube, should be removed surgically. Strangely enough, simple laparotomy often benefits the patient.

Gonococcal Peritonitis

Gonococcal peritonitis arises secondary to infection of the female genitalia. It is rarely fatal but is important because of the necessity to differentiate it from diffuse peritonitis of other origin. When the diagnosis is not clear, it is safer to explore the right lower quadrant and remove the appendix than to risk delay should the lesion prove to be acute appendicitis.

Treatment.—The treatment of acute gonococcal peritonitis is general supportive measures, bed rest, and penicillin in high dosage. Operative treatment is used only to drain persistent abscesses or to remove a chronic tubo-ovarian abscess (See also Chapter 28, The Female Genital Tract.)

Miscellaneous Types

In acute rheumatic fever the peritoneum may become inflamed. Actinomyces may cause peritonitis, as may brucellae and lymphogranuloma venereum.

CHRONIC PERITONITIS

It must be remembered that any type of acute peritonitis may become chronic, with thickening of the peritoneum and the formation of widespread, dense adhesions. When peritonitis lasts more than a few days, peritoneal reaction cells appear, which are probably phagocytic and which resemble large mononuclear cells. Sections of such peritoneum show that these cells project from the surface. Quantities of cloudy peritoneal fluid often persist in chronic peritonitis, which, on culture, prove to be sterile. Such collections are often found in the vicinity of walled-off intraperitoneal abscesses.

Granulomas

In addition to chronic infective granulomas, such as tuberculosis and actinomycosis, the peritoneum may be involved in granulomatous processes caused by irritating foreign material.

Talc Granuloma.—Talcum powder as a surgical glove and instrument powder is now being replaced by other substances but is still in fairly widespread use. Talc crystals in contact with the peritoneum act as an irritant, causing a foreign body reaction. Ordinarily this reaction subsides after a time and no harmful effects are noted clinically. However, in certain individuals, talc in the peritoneal cavity leads to a very pronounced reaction which is proliferative, producing widespread adhesions and masses of granulation tissue which resemble a slowly growing neoplasm. Harmful effects may appear when such masses become bulky and widespread adhesions interfere with bowel function.

Prophylaxis is secured by using other types of surgical glove powder or by careful washing of the gloves and instruments before introducing them into the peritoneal cavity during an operation. **Treatment** of the established granuloma consists of relieving complications, such as intestinal obstruction due to adhesions, by appropriate surgical procedures. On a few occasions it has been possible to excise granulomatous masses.

Sulfonamide Crystal Granuloma.—Sulfonamide crystals, placed in the peritoneal cavity for the prevention and treatment of infection, occasionally cause a granulomatous reaction

with widespread vicious adhesion formation. Crystalline sulfathiazole has been the worst offender because of its low solubility.

Granulomas Due to Nonabsorbable Suture Material.—A small foreign body granuloma forms about every nonabsorbable suture placed in the peritoneum as in other tissues. These reactions seldom reach harmful proportions unless secondary bacterial infection supervenes. If infection persists, sinuses often develop and persist until the suture material is discharged or removed surgically.

TUMORS AND CYSTS

Primary tumors of the peritoneum, omenta, and mesenteries are extremely rare. There is only one true primary tumor of the peritoneum, the so-called *mesothelioma (endothelioma)* which is diffusely infiltrating and malignant.

Cysts of the omenta and mesenteries are rare. *Lymphatic cysts* of the mesentery are considered to be congenital lymphangiomas. They are often single and vary from a few centimeters in diameter to a very large size. They have milky contents and a flat endothelial lining. *Gas cysts* are very rare, occurring in clusters along the mesentery of the small bowel. Such a cyst may rupture spontaneously, leading to pneumoperitoneum and abdominal pain. The diagnosis of pneumoperitoneum can be made roentgenographically, but laparotomy may be necessary to exclude perforation of a hollow viscus. *Hydatid cysts* of the mesentery form multiple masses, sometimes of large size.

Metastatic malignant tumors (usually carcinomas) frequently involve the peritoneum. The three most common intra-abdominal primary sites are the stomach, large bowel, and ovary. Spread is direct by penetration of the serosa and dissemination over the serous surface. Implantations may appear in isolated places at a distance from the parent tumor. When metastasis is widespread throughout the peritoneal cavity, it is called abdominal carcinomatosis. Irritation of the peritoneum causes ascites to develop and the fluid is often sanguineous. Metastasis from primary neoplasms at distant sites may occur in the peritoneum, carried by lymphatics, or, more commonly, in the blood stream (e.g., malignant melanoma).

Tumors arising in the retroperitoneal tissues, whether benign or malignant, are similar to tumors of connective tissue origin elsewhere. Retroperitoneal lipoma and sarcoma are the most common types.

It is noteworthy that peritoneal tumors are often very difficult to diagnose by the usual means of examination, including x-ray methods. If there is free peritoneal fluid present, it should be aspirated and studied carefully, with attention to the type of cells present, bacterial culture, guinea pig inoculation for tubercle bacilli, direct smear, specific gravity, protein content, and microscopic appearance of sections of the sediment.

Treatment.—There is no curative treatment for primary mesothelioma of the peritoneum. Paracentesis abdominis may be performed at intervals if a large volume of peritoneal fluid collects.

Cysts of the omenta and mesenteries require no treatment unless they become very large and cause symptoms. Benign cysts may be carefully dissected out of the mesentery or, if in the greater omentum, may be removed along with surrounding omentum.

Treatment of metastatic peritoneal tumors is limited to palliative operations to relieve symptoms and interference with bowel function except in cases of carcinomatosis peritonei with ascites, where the introduction of radioactive gold seems to be beneficial by reducing the ascites in certain instances. X-ray therapy is of questionable value except, perhaps, in the case of certain radiosensitive sarcomas.

Pseudomyxoma Peritonei

This curious condition may follow rupture of a pseudomucinous cyst of the ovary or of a mucocele of the appendix. Sometimes the entire peritoneal surface is studded with small tumor nodules which secrete an enormous amount of jellylike material that accumulates in the peritoneal cavity. This material acts as an irritant, producing a low-grade peritonitis with the formation of adhesions. The signs and symptoms include abdominal distention, pain, and interference with bowel function.

The treatment is removal of the primary focus, which occasionally results in the disappearance of the whole lesion, but the prog-

nosis is usually poor. In a limited number of cases x-ray therapy has apparently caused arrest or regression of the lesion. Radioactive gold may prove to be useful. This condition must be differentiated microscopically from metastatic colloid carcinoma.

THE APPENDICES EPILOICAE

Significant lesions of the appendices epiploicae rarely occur, but torsion of an appendix epiploica may produce the clinical picture of an acute abdominal emergency. Occasionally the blood supply of an appendix epiploica is interfered with and it undergoes calcification, appearing then as a calcified shadow in x-ray films.

ASCITES

Ascites is an accumulation of serous fluid in the peritoneal cavity. The fluid may be a transudate or an exudate.

An *exudate* is a collection of fluid secondary to an inflammatory process and may be clear, turbid, or purulent, depending on the number of cells present. Blood may be present. The specific gravity is over 1.018 and the protein content over 30 Gm/L.

A *transudate* is a noninflammatory collection of fluid. Such fluid is clear, of light straw color, with a specific gravity below 1.018 and usually below 1.015. The cell count is low and the protein content under 30 Gm/L.

There are many causes of ascites. General causes include cardiac failure, renal disease, constrictive pericarditis, and increased mediastinal venous pressure. Depletion of the plasma protein results in a decrease in plasma osmotic pressure and ascites. Locally, an increase in portal vein pressure from hepatic disease or obstruction of the portal system, an increase in lymphatic pressure from neoplastic or inflammatory obstruction of lymphatic vessels, or rarely an ovarian neoplasm (Meig's syndrome) can cause ascites.

Treatment of ascites may be divided into two broad categories: (1) conservative and (2) operative treatment.

Conservative treatment is of prime importance and is directed toward raising the serum albumin concentration, restriction of salt intake, use of mercurial diuretic agents, and cor-

such as pinworms, seeds, and indigestible food are an unusual and almost accidental cause of appendicitis. Anaerobic microorganisms may also play a part in the development of gangrene.

There is no evidence that the state of health of the patient plays a part in the etiology, as many healthy youngsters come to operation with all degrees of appendicitis and recover immediately when the offending organ is removed, unless complications delay the progress.

Recurrent attacks of appendicitis result in fibrosis with a narrowing of the lumen, and this may give rise to symptoms of partial obstruction. At times the obstruction may be complete, leading to obliterative appendicitis and occasionally to empyema of the appendix, or mucocoele.

Bacteriology and Pathology

The commonest organism cultured in cases of appendicitis is the colon bacillus. However, infections are mostly mixed and the streptococcus plays an important role. In fact it may be the primary cause. The improvement obtained in many cases by penicillin would appear to support this view. In gangrenous appendicitis one of the anaerobic organisms, such as Welch's bacillus, may be a factor, as suggested by occasional postoperative infection in the abdominal wall.

Pathologically, the changes in appendicitis range from simple catarrhal inflammation to gangrene. A small area may show edema, round-celled infiltration, ulceration of the mucosa, suppurative inflammation, or necrosis. On the other hand, the whole appendix may be involved in an acute inflammatory process, and be red, edematous, friable, and gangrenous. An abscess may occur in the wall, or in the periappendicular tissue. A spreading peritonitis may be present with turbid fluid, or the infection may spread to the entire abdomen, causing general peritonitis. The cecum may be involved and become infected, edematous, and friable, a point to be remembered when one considers inverting the base of the appendix in such cases. In an effort to wall off the infection, the omentum may wrap itself around the appendix and form a mass readily palpable on examination but indistinguishable

from a localized abscess. The small bowel may become adherent and give rise to symptoms of partial obstruction.

Clinical Picture

In considering the clinical picture of appendicitis, it will be easier to classify it into acute, subacute, and chronic forms. The acute form should be divided into two fundamentally different types, the first of which is acute inflammation of the wall, and the second, acute obstruction of the lumen, or closed loop obstruction, which was originally described by the late Professor Wilkie of Edinburgh.

ACUTE APPENDICITIS

Acute Inflammation of the Wall (Acute Appendicitis).—Although diagnosis is very simple in a typical case of appendicitis, few diseases present so many diagnostic variants. The typical picture is one in which recent abdominal discomfort centers around the umbilicus and is accompanied by nausea and frequently vomiting. Later the pain becomes localized in the right lower quadrant, with rebound tenderness, increased resistance and tenderness in the right iliac fossa, and tenderness on rectal examination, especially on the right side. The temperature is usually 99°–100° F, with a leukocytosis of 10,000–14,000. If examination of the chest and the urine is negative, the diagnosis is established. The history is frequently of short duration, although there may have been previous attacks.

Diagnostic difficulties increase if the appendix is retrocecal, if it lies along the lateral wall of the cecum, hangs over the brim of the pelvis, or lies adjacent to the ureter. Difficulties are further increased by the type of pathologic change—catarrhal, acute fulminating, perforating, abscess forming, gangrenous or obstructive.

In children, the picture of appendicitis is considerably different from that of the adult. In apparently healthy children, crying and vague tenderness in the right iliac fossa may be the only signs of an acute appendicitis, so that extreme care in examination is necessary. On the other hand, this is the age of exanthemas, many of which have transitory



Plate 28.—The Pathologic Appendix.

abdominal pain. Gastroenteritis is also common during this period, and the best differential method is to administer sweetened fruit juices which act as a mild intestinal sedative in gastroenteritis but do not affect the pain of appendicitis. Fever may be unusually high, or the temperature may be normal. The leukocyte count is not of much help as it may be extremely high—20,000 or as low as 3,000-4,000.

Acute appendicitis occurring during pregnancy is most common around the 3rd month. It presents no special difficulty at this time. Later, as the uterus enlarges, the position of the appendix is higher and the problems increase until just before and immediately after term, when diagnosis is much more difficult. Fortunately it is a rare occurrence at this stage. If strict attention is paid to the usual signs, the diagnosis can be made. Removal must be carried out immediately. Gentleness and care will preclude interference with the pregnancy. Antibiotics will control the infective process. Cesarean section, if contemplated, should not be performed until approximately one month later. The diagnosis of appendicitis during the puerperium is the most difficult of all, and it requires extremely careful study before it can be established. The treatment is appendectomy.

In the aged, appendicitis is less common because of the gradual obliterative process that takes place during the years, but this may predispose to a closed loop obstruction and be all the more serious in elderly patients with low resistance. Here, the only safeguard is to remember appendicitis as a possible diagnosis.

The original pain in the epigastrium or around the umbilicus is usually of a cramplike nature, but when it settles in the right lower quadrant, it is steady and is increased by movement or blowing out of the abdominal wall. Sudden release of pressure on the right side of the abdomen causes pain in the right iliac fossa, so-called rebound pain. This test should not be carried out too vigorously as it could conceivably break down recently formed adhesions and spread the infection. Rovsing's sign is frequently present—pressure over the large bowel causes pain in the right

iliac fossa. Hyperesthesia is often found over the appendix. If the lumen of the appendix is obstructed, the pain is usually crampy, or colicky in nature, and in the early stages muscular resistance is unusual. When the appendix is in the retrocecal position all signs are less marked, because it is shielded by the cecum. When the appendix lies in the lateral paracolic gutter, the pain and tenderness may be localized in the flank. If it is adjacent to the ureter, a few red blood cells may be found in the urine on microscopic examination.

Though the point of maximum tenderness is usually in the neighborhood of McBurney's point, this depends on the site of the appendix. The degree of tenderness and muscular spasm also depends on the position of the appendix, being much more marked when the organ lies against the parietal peritoneum of the right iliac fossa. Tenderness on rectal examination is more marked if the appendix is situated in the pelvis. If a mass is felt, it is due either to the presence of an abscess or to the omentum walling off the appendix as a protective mechanism. The temperature will increase as the infection spreads, with a rise in the pulse rate. When the patient is seen early in an attack, it is unwise to delay treatment because of the absence of fever or tachycardia.

Psoas spasm is a common sign, especially in retrocecal appendicitis. This is elicited by hyperextending the thigh or by flexing and adducting it against resistance. Obturator spasm may be present, especially when the appendix lies in the pelvis, and can be noted by flexing the thigh and internally rotating the leg, which gives pain through spasm of the internal obturator muscle. While a history of constipation is frequently elicited, diarrhea may occur in appendicitis. As a rule, however, this suggests enteritis, and great care should be taken to establish the diagnosis before surgical intervention is undertaken.

Acute Obstruction of the Lumen (Closed Loop Obstruction).—The main characteristic of the obstructive type is an intermittent, crampy pain which is very severe and made worse by movement. The cessation of this crampy pain is indicative of perforation, relieving the tension of the closed loop obstruction, but pain will recur in a few hours as a

generalized peritonitis develops. In the early case of appendicular colic, which, as Wilkie states, so often goes on to perforation, it is usual to find a normal pulse and leukocyte count, but both of these will rise as soon as inflammatory changes develop, either in the wall or surrounding tissue. When leukocytosis is present, the differential count shows a marked increase in the polymorphonuclear cells, with a shift to the left or to the more primitive forms.

Differential Diagnosis

A surgeon should consider the patient with an acute abdomen from two points of view: first, what is the most serious diagnosis, and, second, are the signs and symptoms of such a nature that a laparotomy is advisable in spite of a failure to make a definite diagnosis. The latter observation applies especially to young children and to the aged. Many of the patients in these groups are left until perforation occurs because, until then, the signs are indefinite. In children, early pneumonia may simulate appendicitis, but the tenderness and spasm are less marked, the pain is frequently higher, and the respiratory rate is increased and is frequently accompanied by movement of the abdominal wall and alae nasae. The fever, pulse rate, and leukocyte count are higher than in appendicitis.

In considering the differential diagnosis, it seems easier to classify the conditions into three groups:

1. Those associated with pain and protective muscular resistance
2. Those with pain but without muscular resistance
3. Those associated with a mass in the right iliac fossa

With Pain and Protective Muscular Resistance

Acute Salpingitis.—In the female, acute salpingitis frequently presents diagnostic difficulties. The most important diagnostic points are the history of exposure, a burning sensation on urination, a vaginal discharge, obtaining a drop of pus by milking the urethra, and marked tenderness on moving the cervix.

The sedimentation rate usually shows a greater increase than in appendicitis. If the tenderness is bilateral on bimanual pelvic examination, a waiting policy with the use of antibiotics is advisable.

Ruptured Ectopic Gestation.—Internal hemorrhage and shock predominate, with softening of the cervix and moderate enlargement of the uterus. There is usually a history of a missed period.

Early Pneumonia.—Early pneumonia, especially of the basal type, not only in children but sometimes in adults, may cause confusion. An x-ray of the chest and careful examination of the site of the pain localization is necessary. In pneumonia it is usually higher in the abdomen and is associated with an increased respiratory rate.

Perforated Peptic Ulcer.—A perforated peptic ulcer usually gives a characteristic board-like rigidity in the upper half of the abdomen, but if the gastric contents move down the right paracolic gutter, pain, tenderness, and even a mass may be found in the right iliac fossa. A history suggestive of a chronic ulcer may offer a clue. A plain film of the abdomen showing free gas under the diaphragm, along with an accurate history, should assure a correct diagnosis.

Acute Cholecystitis.—Acute gall bladder conditions may also simulate acute appendicitis. The appendix in cases of nondescent of the cecum may be adjacent to the gall bladder or the liver, and the gall bladder may lie lower than usual. It is important to know whether the patient has had previous attacks of pain in the right hypochondrium. Radiation of pain to the angle of the scapula or to the back, or a history of flatulent indigestion, especially in a middle-aged obese female, suggests gall bladder disease. Further, in cases of acute cholecystitis, the globular tender surface of the gall bladder, moving with respiration, can usually be palpated, and a positive Murphy's sign is pathognomonic.

Regional Ileitis.—This may be confused with recurrent appendicitis. The long history of cramps and diarrhea with gastrointestinal disturbances suggests the advisability of having a barium series, with follow through of the barium through the ileum, when the character-

istic picture of "string and puddle" confirms the diagnosis. In the acute stage of this condition it may be impossible to differentiate the condition prior to operation.

Mesenteric Lymphadenitis.—The initial centralized abdominal pain and muscle spasm of acute appendicitis are absent, but otherwise the picture is remarkably similar. Most cases are diagnosed correctly only when the abdomen has been opened.

Genitourinary Conditions.—Hydronephrosis and pyelitis frequently mimic acute appendicitis, especially during pregnancy. The presence of blood or pus in the urine, the radiation of pain from the loin to the inguinal region, or genitalia, the palpation of a tender kidney, and the demonstration of a renal or ureteric calculus by a skiagram are important. In pyelitis a catheterized specimen of urine will show gross pus, the fever is higher, and prostration may be present. Tenderness is usually situated in the loin.

Pneumococcal Peritonitis.—This occurs in young girls in the lower socioeconomic group between the ages 5-8 years. It is usually primary but occasionally may be secondary to pneumonia or otitis media. When primary, the infection is thought to ascend by way of the patent fallopian tubes. The symptoms are high fever, with diarrhea and vomiting. On palpation, a doughy, slightly distended abdomen is felt in a thin young individual. The treatment is operative. This confirms the diagnosis and permits the local administration of penicillin.

Pain Without Muscular Resistance

Colic, whether biliary or renal, is usually due to a stone passing down the duct and is frequently severe. The patient moves about in agony but is comfortable between attacks.

Biliary Colic.—The pain radiates to the back and shoulders, and there is usually a history of previous attacks. There is flatulence, indigestion, and occasionally jaundice.

Renal Colic.—There is frequency of micturition and the pain usually radiates from the loin to the genitalia. Urinary investigation revealing blood and pus cells in the urine and the roentgenologic demonstration of a calculus are important.

Appendicular Colic.—Appendicular colic is usually of an entirely different character from the other colics. It is better described as a sudden cramplike abdominal pain of varying intensity, with no relation to meals, and frequently accompanied by nausea, vomiting, and headache. Examination is negative, unless the obstruction continues into obstructive appendicitis.

Mass in Right Iliac Fossa

The presence of a mass in the right lower abdomen may be due to one of the following:

Appendicular Abscess.—This is one of the presenting signs of appendicitis.

Ovarian Cyst.—Small follicular cysts are very common and only cause pain when they rupture. They are frequently too small to be felt on examination. On the other hand, larger ovarian cysts, about the size of a fetal head, can be felt on pelvic examination. They tend to become twisted and thus cause pain which is aggravated by movement.

There are several other chronic conditions which may cause a mass or pain in the right lower abdomen, but the distinguishing features should be brought out by the history, examination, and special investigations.

It should be stressed that acute appendicitis is the commonest surgical abdominal condition and that most of the other conditions which have been mentioned are relatively uncommon. The most important differentiation is that of early pneumonia, when unnecessary operation is hazardous and must be avoided.

Complications

Abscess.—This is the commonest complication of appendicitis and, of necessity, means that the infection has spread through the wall or that the appendix has perforated and the infection has been localized by means of the omentum wrapping itself around the infected organ. The position of the abscess will depend on the position of the appendix. The four situations have already been referred to but will be mentioned again.

Retrocecal. This is associated with psoas spasm and is usually late in appearing. It may point in the groin.

Pelvic. When the appendix is in the pelvic position, the infection spreads down into the pelvis and frequently causes irritation to the bladder and rectum, with frequency of micturition and diarrhea. On rectal examination there is a tender, boggy mass in the pelvis, which may be fluctuant.

Intestinal. When the appendix lies between the coils of the small intestine, the abscess is localized in between these coils and presents as an enteritis. A mass may be present, which, however, may be very difficult to feel because it is masked by the distended intestine.

Juxtaparietal. The appendix lying next to the parietal peritoneum causes early irritation and is therefore usually dealt with before abscess formation takes place. However, the

Paralytic Ileus.—With peritonitis alone, the patient recovers, but should paralytic ileus develop, the prognosis is grave. It is due to severe toxic infection which paralyzes the small bowel and causes increasing distention.

Thrombosis of the Portal Vein.—This is due to the infection spreading into the venous system. When this spreads to the liver, suppurative pyelphlebitis develops, with chills and high fever, and an enlarged, tender liver. Fortunately, with the use of the antibiotic and earlier operative interference, this complication is now rarely encountered.

Pulmonary Embolism.—This may occur after any operation, from thrombi originating in the veins of the legs and pelvis. Small emboli make themselves evident by pain in the



Fig. 336.—Sites of localized peritonitis depending upon position of appendix

localizing adhesions or the omentum may form a mass in the peritoneal cavity near the anterior abdominal wall.

Subphrenic abscess is becoming less common after appendicitis and is usually a residuum of generalized peritonitis.

Generalized, Spreading Peritonitis.—Generalized, spreading peritonitis with failure to localize the perforated or infected appendix is a serious complication. There is widespread abdominal tenderness, increased pulse rate, and boardlike rigidity of the abdominal wall. This is soon followed by the hippocratic facies and abdominal distention.

and the spitting of blood. A large embolus usually occurs suddenly without warning about the 10th day and is commonly fatal.

Treatment

The treatment of acute appendicitis is the removal of the organ as soon as the diagnosis is made. To delay only means to court complications. Perforation may occur within a few hours and may be precipitated by the administration of cathartics. Only when operative removal is impossible is it permissible to temporize. The patient is placed in Fowler's position, nothing is given by mouth, and anti-

iotics are administered. Cold compresses should be applied to the abdomen to slow down the process and relieve the pain.

Operation

For the sake of clarity, the operative treatment will be divided into that for the unruptured and ruptured appendix.

Removal of the appendix is carried out under aseptic conditions; spinal or general anesthetic may be used.

The skin incision may be oblique, or transverse following Langer's lines, with the center of the incision at McBurney's point. The subcutaneous fascia is next incised. The external oblique aponeurosis is opened in the direction of the fibers. The internal oblique and transversalis muscles are separated in the direction of their fibers, and by gentle traction, the peritoneum is exposed. This is carefully lifted and opened, and the cecum usually presents. If the finger is curled around the taenia, the cecum can usually be delivered through the wound. Where the three taeniae converge, the base of the appendix will be found. The appendix is then carefully delivered through the wound, and the mesoappendix, containing the appendicular artery, is clamped, divided, and ligated. A purse-string suture is placed about the base of the appendix, and the surrounding area is covered with moist gauze. The base of the appendix is then clamped with forceps and the crushed portion tied near the base. The appendix is then severed with the actual cautery, and the stump of the appendix is invaginated by means of the purse string which is tied. The raw area of the mesoappendix is carefully covered. The small bowel should be examined for a distance of 3 feet to rule out the presence of a Meckel's diverticulum and enlarged lymphatic nodes in the mesentery of the small bowel. In females, the uterus and appendages are palpated with the two fingers. The wound is closed in layers with interrupted sutures, without drainage.

In some cases this gridiron incision is not adequate, but when necessary it may be extended medially to the sheath of the rectus muscle. When the appendix is adherent and cannot be delivered, it may be advisable to cut the base and dissect the appendix out in

a retrograde fashion. When the diagnosis is in doubt, a lower paramedian incision rather than a gridiron should be used.

The choice of the incision is governed by the type of patient and the certainty of the diagnosis. The McBurney or gridiron incision just described may, if necessary, be extended medially to the rectus muscle. This is in effect a transverse incision, and in adults may be used to explore the peritoneal cavity. In children, it may be further extended into a complete transverse one. The disadvantage of a long transverse incision at this level in the presence of infection is the danger of an incisional hernia which is extremely difficult to repair. The better choice therefore for exploration of the whole of the peritoneal cavity, particularly in females, or for a laparotomy in males, is the paramedian incision centered slightly below and to the right of the umbilicus.

When a localized abscess is encountered and the appendix presents easily, the organ is removed as described above and the abscess is drained through the incision or a separate stab wound. If, however, an abscess is encountered and the appendix is not readily available, then it is wise merely to drain it and leave the appendix to be removed at a later date, usually 3 months afterward. The danger of breaking down a natural barrier and spreading the infection is more serious than leaving the organ where the infection has already spread beyond its limits. If the cecum is edematous and friable, it may be impossible to invert the stump of the appendix, and in such cases the omentum may be gently sutured over the cecum as a further precaution.

Ruptured Appendix.—The perforated gangrenous appendix with a generalized peritonitis is a serious condition. The appendix is therefore removed as quickly and as gently as possible, and second, the peritoneal cavity should be drained. Usually the drains are placed in the most dependent position or where the infection is most likely to localize, such as the pelvis, retrocecal fossa, or toward the ileocecal mesentery.

In localized abscesses, and, in fact, even in generalized peritonitis, topical application of penicillin and streptomycin into the cavity

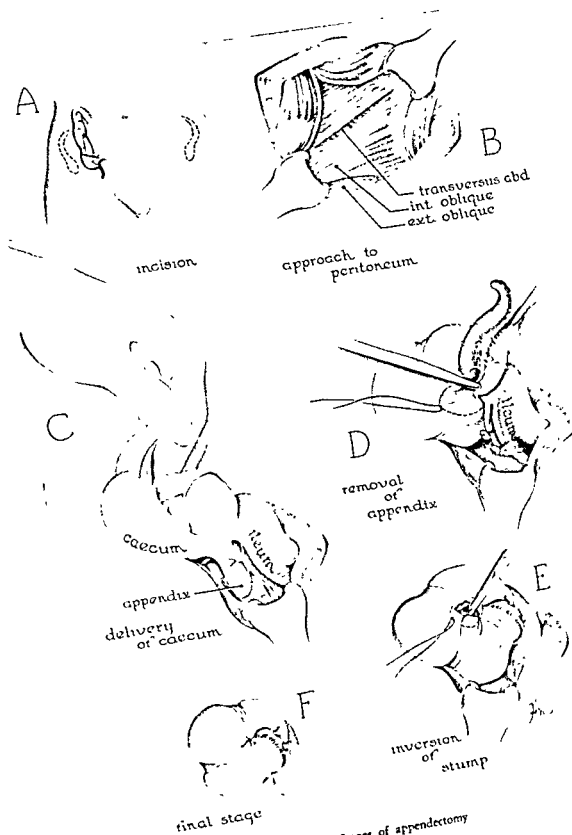


Fig 337—Stages of appendectomy

through a small catheter beside the drain has been of great value. The local concentration is about one thousand times higher than with parenteral administration, and the formation of adhesions has been minimal. This should be continued for 2-3 days, using 500,000 units of penicillin and 0.5 Gm of streptomycin. Other antibiotics such as Aureomycin, Chloromycetin, Terramycin, or neomycin may be added as desired.

Expectant Treatment

The expectant treatment advised by Ochsner and Sherrin is really the treatment of a localized abscess or inflammatory mass of some duration. It should never be used in the treatment of acute appendicitis except in cases occurring in isolated areas where adequate surgical facilities are not available. The patient is placed in the true Fowler's position at an angle of 30 degrees with the floor, hot fomentations are applied to the abdomen in order to assist in the resolution of the mass, nothing is given by mouth, and sedatives are given to relieve pain. If this procedure is followed, the mortality is lower, and subsequent removal 3 months later may be carried out safely.

Postoperative Care

In the uncomplicated cases there is remarkably little postoperative treatment. Patients are encouraged to get up the next day and to lead as normal a life as possible.

In those cases with a localized abscess or generalized peritonitis, the patient is placed in Fowler's position and gastric suction or, better, intestinal drainage by means of a tube is used to prevent distention of the bowel and the development of adynamic ileus. Change of posture, complete abstinence from food and fluids by mouth, and the intravenous administration of fluids and electrolytes also aid in accomplishing this result. Any attempt to stimulate the paralyzed bowel by drugs or surgery is futile.

SUBACUTE APPENDICITIS

Subacute appendicitis is a convenient term to describe those cases which are milder than the acute form and may include recurrent ap-

pendicitis. There is no practical way of correlating these milder forms with etiologic causes or pathologic classifications except to say that they will include catarrhal, exudative, proliferative, and productive inflammation, but exclude suppurative, ulcerative, and obstructive inflammation and necrosis of the appendix.

RECURRENT APPENDICITIS

The term chronic appendicitis, while in wide use in medical circles, should be reserved for the specific granulomas such as tuberculosis or actinomycosis. Vague symptoms of pain in the right lower quadrant, often ascribed to chronic appendicitis, are frequently not relieved by removal of the appendix.

However, the changes that occur in the appendix during an acute attack predispose to recurring infections. In recurrent acute appendicitis the symptoms are similar to those described under the acute form, although milder in degree, and between attacks the patient may be free of symptoms. If this clinical picture presents, the diagnosis is not in doubt and the patient will be cured by removal of the appendix.

More common is recurrent appendicular colic. Fecaliths or other foreign bodies contained within the appendix cause excessive peristalsis in the organ as it attempts to expel the object. Colicky spasm felt in the right lower quadrant of the abdomen, associated with deep tenderness on palpation in this area, will confirm the diagnosis.

ACTINOMYCOSIS OF THE APPENDIX

The second commonest site of actinomycosis is the right iliac fossa, although in comparison with other causes of appendicitis it is a rare disease. The patient usually presents after several days or weeks with a mass and tenderness in the right iliac fossa. At operation an abscess is found with pus containing yellow sulfur granules. If these are squeezed between two microscope slides, the ray fungus can be identified with a microscope in the theater. Removal of the appendix is carried out, the abscess is drained, and large doses of penicillin in combination with streptomycin or other antibiotics are administered.

TUMORS OF THE APPENDIX

Benign

The general incidence of new growths of the appendix is low, consisting of about 0.5% of all intestinal growths; but the appendix may be frequently involved in secondary neoplasms. The simple tumors, all of which are rare, include fibroma, myxoma, lipoma, angioma, and adenoma. This last is more common than the others, is usually associated with the symptoms of mild appendicitis, and is an accidental finding at operation.

Malignant

Primary malignant tumors of the appendix are uncommon. Adenocarcinoma occurs but is rare. Of more importance is the carcinoid tumor. These occur in the appendix and in the small bowel. The location of the tumor is of some importance, for if in the appendix they are of low malignancy and do not metastasize, while those in the small intestine may be multiple, produce obstruction to the bowel, and metastasize to the regional lymph nodes.

Carcinoid tumors of the appendix usually cause no symptoms, and the diagnosis is made only at operation. They produce a firm localized swelling in the appendix and appear light yellow on their cut surface. Microscopically, the tumor consists of masses of spheroidal

cells which are rich in lipid. According to Masson they are chromaffinomas.

The treatment is resection. Recurrence of carcinoid tumors of the appendix is rare, and the prognosis is excellent.

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Chapter 26

Colon

Harry S Morton, MB

HISTORY

The history of colonic surgery began in the 18th century when Littre suggested colostomy for obstruction in 1710, and Pflöze performed it successfully 66 years later.

During the next hundred years the mortality for resection prevented progress, but when surgeons turned to exteriorization, peritonitis was reduced. Mikulicz in 1903 popularized and gave his name to this method which had been independently developed by Bloch and by Paul. During the first part of the century this treatment was mainly used for tumors, and when it was applied to gunshot wounds of the colon during World War II, the extremely high mortality was immediately reduced to approximately 25%.

The postwar development of preoperative colonic preparation helped to make possible resection for tumors and diverticulitis. Intravenous therapy and intraluminal suction are other factors which have assisted in the lowering of mortality.

ANATOMY

The large intestine begins in the right iliac fossa as a blind pouch known as the cecum which extends about 2½" below the ileocecal junction. It then continues as the ascending colon, the hepatic flexure, the transverse colon, the splenic flexure, the descending colon, the sigmoid, and finally ends at the peritoneal reflection in the pelvis.

The ascending and descending colon and the two flexures are fixed posteriorly and covered by the peritoneum, except on their posterior aspect. The transverse colon and the sigmoid, on the other hand, have mesenteries which permit wide mobility. The total length of the large bowel is usually 5', approximately one fifth that of the alimentary canal. The diameter gradually diminishes from about 2½" in the cecum to 1½" in the sigmoid.

The wall of the large intestine consists of five layers. There is a lining mucous membrane of single columnar cells which are arranged into the crypts of Lieberkuhn. Under this is the submucosa containing the vessels, nerves, and lymphatics. Next comes the circular muscle coat. The longitudinal muscle fibers are characteristically collected into three bundles called the taeniae. These are about one sixth shorter than the remainder of the intestine, and therefore the wall is puckered into large sacculations. The outermost layer is the serosa or visceral peritoneum covering the appendices epiploicae which consist of fat and blood vessels. The taeniae and appendices epiploicae are particularly characteristic of the large bowel.

Blood Supply

Blood is supplied to the colon from two sources. The superior mesenteric artery gives off the middle colic artery and the right colic artery. This latter vessel divides into two branches, the descending branch anastomoses

with the terminal branch of the superior mesenteric artery in the ileocecal region; the ascending branch anastomoses with the right branch of the middle colic artery. The left colic artery arises from the inferior mesenteric artery near its origin and passes upward and to the left, where it divides into ascending and descending branches. The ascending branch anastomoses with the left branch of the middle colic artery near the splenic flexure, while the descending branch anastomoses with the first sigmoid artery. This series of anas-

1-6 and anastomose with each other in arcades. There is a second series of smaller arches before the short straight arteries enter the sigmoid in the same manner as in the rest of the colon. The architecture of the vessels in the sigmoid is therefore similar to that of the small bowel.

The length of the sigmoid arcades is of fundamental importance in proctosigmoidectomy because it is only when the arcades are long that it is possible to mobilize the sigmoid down to the anus and preserve the blood supply.

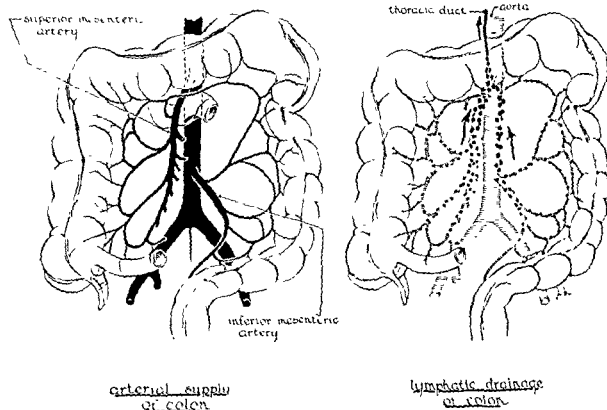


Fig. 338.—Normal arterial supply and lymphatic drainage of colon

tomoses near the colon forms the marginal artery. All these branches give off short straight vessels to supply the colon. As they approach this organ, they divide into two branches which penetrate the muscle wall. Finer branches move around the surface to supply both the muscle and mucosa near the two anterior taeniae. The openings for these vessels through the circular muscle coat frequently cause weakness in this layer, particularly on the left side. The sigmoid arteries vary in number from

The arterial architecture is accompanied by the venous tributaries, the lymphatic drainage, and sympathetic nerves. Many surgeons believe that the blood supply determines the extent of the surgery that can be performed, but it has been found in practice that end-to-end anastomosis can be executed in any part of the large bowel, and the blood supply is better than was previously believed to be the case, provided the patency of the marginal artery is preserved.

Lymphatic Drainage

The lymphatic drainage begins in the sub-mucosal layer with the vessels forming a network which drains into numerous nodes along the posterior aspect of the colon and in the mesentery. These nodes in turn drain by vessels to other nodes in the middle of the mesentery and finally into the para-aortic nodes. The limiting factor to cancer surgery is involvement of the para-aortic nodes by the growth which makes them adherent not only to the abdominal aorta but also to the inferior vena cava. Therefore surgery, to be successful, must be carried out before the disease has spread to these nodes.

PHYSIOLOGY

One function of the colon is to concentrate the feces by removing water. The size of the colon varies with the diet of the individual animal. Meat-eating animals have very little need for a colon because digestion has been completed in the small bowel. It is therefore only necessary for the water to be absorbed from the feces. In herbivora, on the other hand, digestion of cellulose products takes place in the colon, and there is usually a large cecal pouch where digestion may continue for a long time. Man's colon is designed for an omnivorous diet. It is slightly sacculated as in some of the herbivora, and the length is suitable for the digestion of a moderate amount of cellulose.

Movements of the Colon.—It is extremely difficult to observe peristaltic movement of the large intestine either at operation or during radiologic studies. Occasionally a sudden movement may be observed on the fluoroscope, when the contents of the right half of the colon move over to the left half in a matter of seconds. This is called *mass movement* and usually occurs after a meal, in association with the gastrocolic reflex. There is a relaxation of tonic action of the musculature, the haustral markings disappear, and the colon becomes a smooth tube. Then a large peristaltic wave sweeps the whole contents along.

The Nerve Plexuses.—The nerve plexuses are arranged in a somewhat similar manner to those in the stomach. In fact there appears to be a closer association between the central

nervous system and both the stomach and the large bowel than with the small bowel. The autonomic nervous supply consists of the sympathetic and parasympathetic nerves; the sympathetic follow the blood vessels to the large bowel, while the parasympathetic are the vagus, and the sacral outflow from S2 and S3. So far as is known the vagus supplies the right half of the colon almost to the splenic flexure, and the sacral outflow the left half of the colon. The numerous plexuses in the colonic wall are still being debated, as there are more than those described by Auerbach and Meissner.

EMBRYOLOGY AND CONGENITAL ANOMALIES

(See Chapter 30.)

FOREIGN BODIES

Swallowed foreign bodies commonly pass through the alimentary canal; occasionally they may become lodged in the colon, usually the sigmoid; they may also be introduced from below or travel from an adjacent structure. Several factors contribute to the lodgment of foreign bodies in the sigmoid colon since it is sacculated, may contain diverticula, its lumen is narrower, and there is a physiologic sphincter at the rectosigmoid junction. Those introduced from below seldom get beyond the rectum. Penetrating foreign bodies are rare, but occasionally impaling accidents may cause them to break off and remain in the lumen.

The symptoms are extremely variable. There may be pain, hemorrhage, constipation or diarrhea, or the complications of infection or genitourinary tract involvement.

Treatment.—Unless peritonitis has occurred, the treatment is expectant, as most foreign bodies will pass spontaneously. If peritonitis results from perforation of the bowel, laparotomy must be performed. Rectal perforation may result in a pelvic cellulitis which should be drained.

WOUNDS AND INJURIES OF THE COLON

External violence from blunt instruments seldom causes injury to the colon because of its mobility and elasticity, while that from

knife wounds may cause lacerations of the abdominal wall without necessarily injuring the adjacent bowel wall. On the other hand, gunshot and especially high velocity missiles cause jagged perforations with marked contusion of the surrounding bowel wall, which necessitate exteriorization. Such injuries are not suitable for closure by suture.

When an individual is near the blast of an explosion the intestine containing gas is often perforated. This is due to the rapid acceleration of the compression wave at the interface between solid and gas and to the tearing force of the confined gas. In civilian practice, workers using compressed air nozzles may cause damage to the colon, whether by accident or horseplay, when the nozzle is brought near the anus. This is an extremely dangerous accident since the compressed air, passing harmlessly through the clothing, seldom injures the anus and rectum which are well supported, but causes blowout of the splenic flexure or cecum, the seriousness of which may not be recognized for several hours.

The symptoms of perforation of the colon are pain and localized tenderness. Later there are the signs of peritonitis—nausea, vomiting, boardlike rigidity, and shock. The spread is rapid throughout the peritoneal cavity, and the critical time period is approximately 6 hours.

Treatment.—The shock must be treated quickly by raising the foot of the bed and by giving vasopressors and blood transfusions. The keynote of colonic surgery in military perforations is exteriorization of the damaged gut. Lacerations require suture and proximal defunctioning colostomy.

MESENTERIC CYSTS

Mesenteric cysts may be classified into

- 1 Developmental
 - a Enterogenous
 - b Chylous
 - c Lymphatic
- 2 Traumatic, usually hematomas
- 3 Inflammatory, such as
 - a Mesenteric adenitis
 - b Tuberculous adenitis
 - c Hydatid
- 4 Neoplastic
 - a Dermoid cysts
 - b Teratomas

Enterogenous cysts are usually single and attached to the intestine at one point, with a wall consisting of all the layers of the bowel. The cavity contains clear mucoid fluid, usually opalescent, but it may be thick and opaque. These cysts arise as localized duplications of the bowel but cause symptoms only in adult life. They are usually found near the cecum and ascending colon.

Chylous cysts contain thick inspissated material resembling chyle; the wall is of thick fibrous tissue with an endothelial lining and a round cell infiltration with occasional giant cells.

Lymphatic cysts are thin walled, are frequently multiple and multilocular, and have clear contents.

Clinical Picture.—The clinical picture is a vague mobile abdominal mass in a young adult, giving rise to indefinite symptoms. The size varies but may be considerable. The cysts are usually painless unless the complications of infection, adhesions, rupture, hemorrhage, or intestinal obstruction occur.

Treatment.—The treatment is removal, and this can often be done without interfering with the blood supply to the bowel. Should this prove to be impossible, then resection of the affected bowel and its mesentery is indicated.

VOLVULUS

Volvulus is a torsion of an abnormally mobile loop of bowel on its axis, usually resulting in obstruction. This most often occurs in the sigmoid region because of the length of its mesentery. The cecum may be affected, but only if there has been failure of attachment and it is freely mobile with a mesentery, as mentioned in the second anomaly of malrotation (see Fig 395). This condition is commoner in inmates of mental institutions than in the general population.

Clinical Picture.—The picture is one of acute severe intestinal obstruction. The abdominal distention is marked, and copious vomiting occurs, with early dehydration and shock, particularly if strangulation of the involved loop has developed. There is frequently a history of similar but less severe attacks of pain from which the patient has recovered.

A flat film of the abdomen will show greatly dilated loops of bowel, and it is frequently possible to ascertain the area of volvulus by the position of the most dilated loop.

Treatment.—If one or two enemas do not relieve the torsion, laparotomy is indicated with untwisting of the involved loop of bowel. When strangulation has occurred, resection is obligatory. As the volvulus may recur following untwisting, colopexy or resection is frequently advocated. In all cases the accompanying shock and dehydration must be corrected by intravenous fluids, electrolytes, and whole blood transfusions.



Fig 339—Acute intestinal obstruction due to volvulus of the sigmoid. Note the marked unequal distention of the sigmoid loop. The markers indicate the adjacent walls of the distended loop.

DISEASES OF THE APPENDICES EPIPLOICAE

The appendices epiploicae are localized, peritoneal-covered outgrowths of fat from the wall of the large bowel and are one of the main characteristics of the colon. Their number varies enormously and may be in excess of two hundred. They are most numerous in those areas with a mesentery, namely, the

transverse and sigmoid colon, and are arranged in two parallel rows adjacent to the anterior and posterior longitudinal taeniae. One of these appendices may become twisted, causing a sudden sharp abdominal pain which is referred to the right side of the abdomen. Vomiting, tenderness, and rigidity are present, giving the picture of an acute surgical condition which may be mistaken for appendicitis, cholecystitis, or diverticulitis. The treatment is surgical removal. The appendices epiploicae may become adherent to other abdominal contents and cause intestinal obstruction. This is usually a late result of infection. No definite symptomatology can be given.

DIVERTICULOSIS

True and false diverticula occur in the large bowel, the former are rare, amounting to less than 1%, and occur in the proximal half of the colon. Their walls consist of all the layers of the bowel. False diverticula generally occur in the distal portion, are nearly always multiple, and consist of a herniation of the mucosa through the musculature, usually where the vessels penetrate the wall. They are caused by increased intraluminal pressure. They are frequently seen in barium studies of the colon and do not cause any symptoms.

Pathology.—Diverticula are found in 10% of persons over 40 years of age. If the opening of the sac into the intestine is large, symptoms will rarely develop, as the sacs empty. However, when the openings are small, the contents of the bowel enter the false diverticula and may become lodged there, leading to ulceration of the walls and the production of diverticulitis.

DIVERTICULITIS

Inflammation in a false diverticulum is diverticulitis. The signs and symptoms are those of localized intra-abdominal inflammation, pain, tenderness, and rigidity, usually in the left lower quadrant of the abdomen. Perforation of a diverticulum may result in a localized abscess or generalized peritonitis.

If an abscess develops, a tender mass will be palpable. The abscess may rupture, causing generalized peritonitis, or it may communicate with adjacent bowel or bladder, resulting in a

fistula. These fistulas may be internal or external onto the skin of the abdomen or perineal region. Because of the age of the patients, it may be difficult to rule out malignancy, and careful investigation must be carried out. Occasionally the inflammatory process may obstruct the lumen of the bowel.

In establishing the diagnosis, sigmoidoscopic examination, though it gives little positive information, may help to eliminate the possibility of cancer. The barium enema shows the many diverticula, and the narrowing of the lumen in peridiverticulitis is longer and more tapered than in stenosis from cancer. Diverticulosis and carcinoma, however, are both common diseases in the older age group. They not infrequently occur together, and the final determination may be only at laparotomy and by pathologic examination.

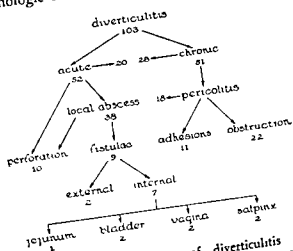


Fig. 340—Complications of diverticulitis. An analysis of 103 cases showing how the various complications arise.

Treatment.—Unless complications arise the treatment is conservative. This includes a low residue bland diet, mineral oil as the only laxative, and the administration of anticholinergic drugs. The treatment of the complications has recently changed from a conservative to a more radical approach. For perforation with generalized peritonitis, drainage of the peritoneal cavity with the administration of local and systemic antibiotics is routine. Obstruction and fistula formation require a proximal transverse colostomy, usually in the transverse colon, later followed by resection and subsequent closure of the colostomy. Some

authors advocate resection even earlier in percolitis, in local abscess, and in the chronic stage, in the hope of preventing the more serious complications.

ULCERATIVE COLITIS

Etiology.—By definition the cause of non-specific ulcerative colitis is unknown. Various theories have been advanced. Some authors have supported an infective etiology—any of the normal flora of the colon may penetrate the mucosa and initiate minute inflammatory foci. Others believe it to be an allergic phenomenon, but this has been confirmed only in about 10% or less of the cases. The psychiatrists state that the patients are usually psychologically dependent on one member of the family group, that they are frustrated and not ambitious. These mental conflicts are transmitted through the vagus nerve to the colon, where it has been demonstrated that the impulses affect the blood supply causing first ischemia, later followed by a marked congestion. On the other hand, most authorities are still in doubt whether this psychologic theory is a cause or an effect. Some have suggested a vitamin deficiency and others, neurovascular imbalance. Another theory involves enzymes, such as lysozyme, while still another concerns the relationship of metabolic derangements of the steroids and related hormones.

To simplify this complicated etiology, it would seem wise to combine these theories into a composite picture. In persons with a certain psychologic pattern impulses may pass down the parasympathetic to the colon, thereby altering the blood supply and allowing the normal bacterial population better access to the mucosa. The congestive phase favors aerobic organisms, the ischemic phase anaerobic, so that infection is a secondary manifestation in an already abnormal bowel. The stimulation of the vagus nerve increases the motility, which fractures the friable mucosa, producing ulceration. The steroids and enzymes further influence the metabolic derangements. With the presence of ulceration there is the loss of absorption, vitamin deficiency, and alteration of nutrition, with resulting further diarrhea and the setting up of a vicious cycle.



Plate 29.—Diverticulosis and Diverticulitis.

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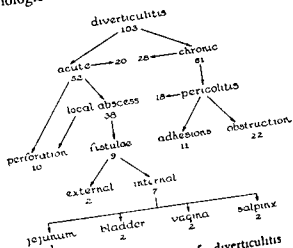


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Plate 29.—Diverticulosis and Diverticulitis.

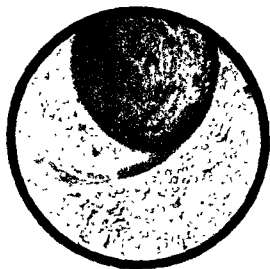


Plate 30.—Ulcerative Colitis.

Clinical Picture.—Nonspecific ulcerative colitis usually presents between the ages of 20-40 years but may occur at any age, and the sexes are almost identically affected.

In the true form of recurrent ulcerative colitis the attacks begin with severe diarrhea, with frequent stools up to 15-20 a day, lasting more than a week. The diarrhea is associated with pain, malaise, weakness, loss of appetite, and loss of weight. The patient is pale, the eyes sunken, the abdomen slightly tender with no palpable masses, and the temperature is elevated. On sigmoidoscopic examination the colonic mucosa is red, granular, and bleeds easily to the touch, and pinpoint ulcers may be found. These pinpoint ulcers gradually enlarge until the muscle coat is exposed. Between the ulcers the remaining mucosa is engorged and edematous, forming pseudopolypi which occasionally may become malignant. The ulcers have even been noted to arrange themselves in three longitudinal bands in extensive cases. Radiologic examination of the large bowel by barium enema shows an irritated, hyperactive colon with loss of normal haustration. In the more advanced cases, it has a "pipe-stem" appearance, and ulcers are seen. Laboratory tests reveal low plasma proteins, and in severe cases the liver function is impaired.

Complications.—The *minor* complications are hemorrhoids, fissures, pruritus, hypertrophic osteoarthropathy, and infantilism. The *major* complications are pseudopolypoidosis, stricture, perirectal abscess, fistula, perforation, and carcinoma.

Differential Diagnosis.—The diagnosis of this disease is one of exclusion. At the time of sigmoidoscopic examination a swab culture is taken from the mucosa in order to exclude *Entamoeba histolytica* and the organisms of Shiga and Flexner which cause bacillary dysentery. Sigmoidoscopic examination will be negative for the 10% of the cases in which the disease is confined to the proximal colon, but this will be demonstrated by a barium enema. This examination will also determine the extent and stage of the disease. Malignant changes can only be excluded by biopsy and supervene in about 5% of cases.

Treatment.—Medical treatment consists of blood transfusions and iron therapy for anemia,

increased fluid intake to overcome dehydration, supplementary feedings of high protein milk-shakes, and vitamins, particularly B and C. Intestinal sedatives should be given, such as atropine, hyoscine, and phenobarbital. Recently 100 mg. of cortisone daily for a week has been used. This therapy is successful in more than half of the cases. A further 20% will be continued on medical treatment but with definite impairment of function. About one quarter of the patients will require surgical intervention.

Indications for Surgery.—The surgical therapy of ulcerative colitis should be restricted to only the most carefully selected cases. For this purpose ulcerative colitis is divided into three groups:

Group I. Those patients with fairly mild symptoms who respond well to medical therapy and those cases in which the disease does not prevent regular attendance at work or interfere with the daily routine of life.

Group II. Those patients with moderately severe symptoms, with only fair response to medical treatment, frequent exacerbations of symptoms, and a reduction of activity. This group is also subject to the complications or sequelae of the disease which are as follows:

- 1 Perforation of the distended colon
- 2 Stricture formation
- 3 Massive hemorrhage
- 4 Fistula formation
- 5 Abscess formation
- 6 Chronic inanition
- 7 Psychologic changes
- 8 Pseudopolypoidosis
- 9 Malignant change

Group III. The fulminating type. These patients develop an extremely severe type of the disease, with almost continuous diarrhea, hemorrhage, abdominal pain, dehydration, and rapid wasting.

Operative treatment of ulcerative colitis should be reserved for patients in Groups II and III. Group III patients should be prepared for operation by concerted efforts to improve the nutritional status, restore the blood volume, and reduce the pain and secondary infection. It may be necessary to operate on a woefully ill patient, but this is frequently the only means of saving life.

Group II patients, of course, require surgery for the complications of the disease. However, some Group II patients without complications

require operative treatment, although great care in selection must be taken. We consider that operation is indicated under the following conditions:

1. If there is loss of time from work
2. If pleasures or social activities are affected
3. If pathologic changes develop

In any case, an ileostomy should not be performed until it becomes a most welcome relief from the tortures of the disease. It is our experience that if the ileostomy is done early in these so-called intractable cases, the patients do not become psychologically adjusted to the ileostomy and are a continual problem for the surgeon. On the other hand, when the escape from a hitherto intolerable situation is given by ileostomy (and colectomy), the relief of the patient is such that he never wishes to go back to his old way of life.

Operative Treatment.—The operative treatment of ulcerative colitis has been unsuccessful in the past. Many different-staged procedures have been recommended. Except from a historic point of view, it would now appear unnecessary to mention them—appendicostomy, cecostomy, and internal defunctioning anastomosis.

Ileostomy.—This alone carries with it a mortality of 15-20%. It does not remove the disease and has been recommended in the past as a primary procedure to be followed in convenient stages by a right, then a left, hemicolectomy, and finally abdominoperineal resection.

Partial or Subtotal Colectomy.—When medical treatment fails, the patients with fever, dehydration, anemia, continuing diarrhea, and blood, pus, and mucus in the stool should be prepared for operation by intensive intravenous therapy, including blood transfusions, supplementary feedings by mouth, sulfonamides, antibiotics, and intestinal sedatives. The hemoglobin should be at least 80% and the plasma proteins within normal limits. Then an ileostomy combined with a subtotal colectomy should be carried out in one stage. This procedure eliminates the toxic absorption from the diseased bowel and the protein and the red cell loss in the bowel exudate. The mortality from this procedure is much less than from ileostomy alone, even in fulminating cases, but it is important that this surgical procedure should be

carried out early enough to be lifesaving. Some surgeons have attempted, in the very severe cases, to do a total colectomy with abdominoperineal resection in one stage. This, however, carries a high mortality and would seem to be too extensive.

A small proportion of cases, where the disease is limited to the proximal colon, may have a subtotal colectomy and an ileosigmoid anastomosis. Otherwise, the patients have to carry a permanent terminal ileostomy. The question is often raised whether it is possible to close the ileostomy and join it to the remaining sigmoid, rectum, or anus. This is possible if the distal portion of the bowel is stretched to an adequate diameter, if the disease is under control, and if the anastomosis is made distal to the sacral parasympathetic outflow.

Vagotomy.—In early cases, vagotomy has been advocated, particularly if the disease is limited to the proximal colon.

Abdominoperineal Resection.—This should be carried out if the disease process persists in the distal remaining segment.

Prognosis.—The patient with an ileostomy and a total colectomy can resume normal activity. There is no difficulty in maintaining satisfactory nutrition on a normal diet. The mortality for this procedure is less than 5%.

INTUSSUSCEPTION IN ADULTS

The year 1751 marks the beginning of our knowledge of adult intussusception. Nuck first made the diagnosis in a female of 50 years; and Velse and Bonetus each operated on adults for this condition. By definition it is an invagination of a proximal portion of the bowel (the intussusceptum) into a distal portion (the intussusciens).

Pathology.—Almost any area in the large or small bowel may be the site of an intussusception. It usually occurs singly, although occasionally compound, multiple, retrograde forms have been reported, particularly in the colon. The following types are usually described:

1. Ileocecal—where the apex of the intussusception is the ileocecal valve
 2. Ileileal
 3. Jejunileal
 4. Ileocolic
 5. Colocolic
 6. Jejunogastric
 7. Gastroduodenal
- These forms constitute a classification of anatomic sites.
- These occur in association with simple tumors of the stomach.

Once the intussusception becomes established, edema of the bowel sets in and there is peritoneal exudate, blood vessel strangulation, and finally gangrene if the condition remains unrelieved. In a few cases, spontaneous reduction may occur, the process may become chronic, or the intussusceptum may slough and be passed per rectum.

Etiology.—In contrast to the unknown etiology in children, the cause in the adult is usually a tumor in the wall, such as a submucous lipoma, a papilloma, or carcinoma of the colon.

typical "cupola" outline at the head of the barium mass

Treatment.—The treatment of choice is laparotomy and reduction of the intussusception by taxis, that is, by compressing the apex backward. If this is at all difficult, then resection with end-to-end anastomosis should be carried out. In the adult, there is nearly always an organic lesion as the underlying cause, which should be excised. If, however, carcinoma is found then the principles of cancer surgery are employed

(See also Chapter 30, Pediatric Surgery.)



Fig 341—Intussusception of carcinoma of the ileum into the colon in a female patient, aged 50 years

A, With barium B, Postevacuation

Clinical Picture.—The clinical picture is usually that of intestinal obstruction with the gradual onset of crampy abdominal pain, nausea, and vomiting. In the enteric group this occurs at an earlier stage than in the colonic cases, where the process is less acute and consists of obscure abdominal pain associated with an ill-defined mass

Investigation should always include a rectal examination and a search for blood, particularly in the colonic group. A barium enema will be helpful in establishing the diagnosis, with the

SPECIFIC INFECTIONS OF THE COLON

Tuberculosis

Tuberculosis is becoming steadily less common. When it occurs it affects the ileum and is due to swallowed tubercle bacilli in a patient with pulmonary tuberculosis. In the region of the cecum it usually causes a large inflammatory mass which is palpable. It can be confused pathologically and clinically with regional ileitis.

The treatment is a short-circuiting operation, or resection with subsequent anastomosis

Actinomycosis

Abdominal actinomycosis commonly affects the cecum, terminal ileum, and appendiceal regions. It cannot be recognized preoperatively unless there are sinuses, when the ray fungus can be demonstrated in the discharge.

The treatment is surgical removal and chemotherapy

Amebiasis

Amebiasis is of importance because of both its local and distant manifestations. There is a chronic thickening of the colonic wall, with dysenteric ulcers of varying size, usually having a ragged undermined edge and a slough covering the floor containing the ameba. The later cicatrizing cases present as chronic intestinal obstruction and must be distinguished from carcinoma. There is a thick edematous swelling of the wall, longer than in malignancy, frequently multiple, and without ulceration, while the malignant lesion is firm, short, single, friable, and ulcerated. When amebiasis is diagnosed, the treatment with emetin gives dramatic results.

The distant manifestations are amebic abscess of the liver with the "anchovy sauce" contents, which must be drained. Rarely is there an abscess of the brain or lung.

BENIGN NEW GROWTHS

All varieties of benign growths may be found in the colon, the most common being lipomas and adenomas. The fibroadenoma is frequently seen as a multiple polyposis, either congenital or acquired. The rarer growths are angiomas, fibromas, and myomas. These new growths may give rise to bleeding, diarrhea, and occasionally pain.

Multiple Polyposis

The pedunculated adenomatous polyp may be either single or multiple, and it usually occurs in the distal portion of the large bowel. Such polyps have a marked tendency to become malignant. In fact, the comparative distribution of carcinoma is so strikingly similar

that the removal of the area containing the polyps amounts to a prophylactic cancer operation.

Treatment.—The small sessile adenomas may be removed by cauterization. Pedunculate polyps are cauterized, including the entire base. Large sessile adenomas must be excised.

Villous Papilloma

Long, thin, delicate branching finger processes projecting from the surface, the pile of a velvet carpet, are much more numerous than the other forms and require radical removal.

Pseudopolyposis

This exuberant inflammatory mucosal proliferation between the ulcers of ulcerative colitis usually occurs in the more chronic forms of the disease, both specific and non-specific. The incidence of carcinoma developing in pseudopolyposis varies from Thorlakson's 6% to Duke's 50% in those cases of over 10 years' duration. The indication for colectomy in such cases is therefore clear.

Familial Polyposis

Familial polyposis is a relatively rare condition characterized by the appearance of multiple adenomatous polyps in the colon and rectum. It is termed familial because it is transmitted from one generation to the next usually as a mendelian dominant, although occasionally as a recessive character. Although polyps may be present at birth, they usually appear later, producing symptoms between 15-40 years of age. Rarely, the condition exists throughout life without causing symptoms.

Diagnosis.—Except when the family history of the condition is known and an apparently well member of the affected family is examined specifically for the presence of polyps, the diagnosis is made after symptoms of complications develop. Common symptoms are (a) bleeding by rectum, (b) intussusception, (c) protrusion of a low-lying polyp from the anus, and (d) bowel obstruction, chronic or acute, as a rule due to a carcinoma developing in a polyp.

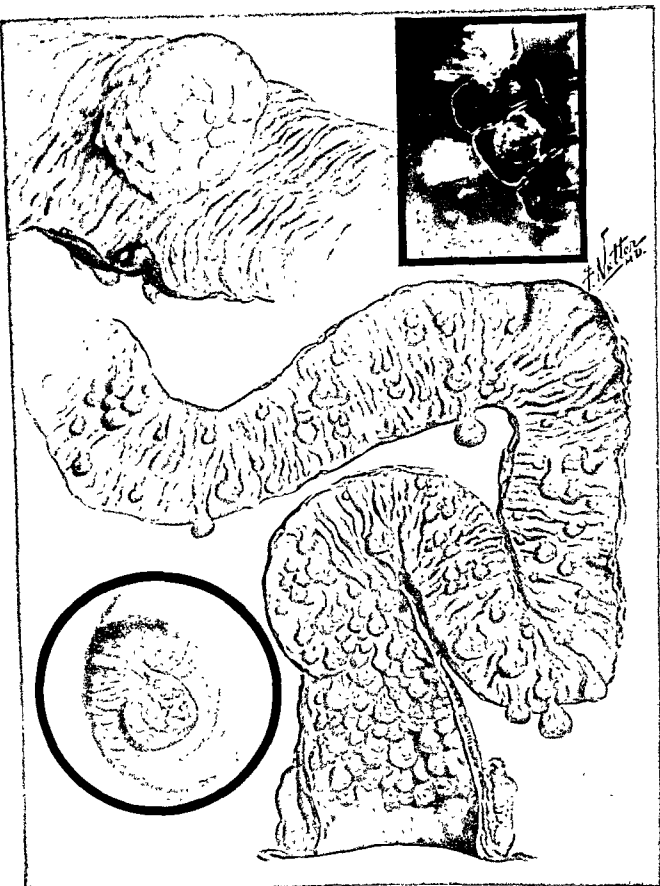


Plate 31.—Benign Tumors Multiple Polyposis



Almost invariably the diagnosis can be made by rectal and sigmoidoscopic examinations. The barium enema x-ray examination, particularly with air-contrast technique, will reveal the presence of the polyps in the bowel above the reach of the sigmoidoscope, provided they have reached sufficient size. When malignant degeneration of a polyp has occurred, the findings will be those of carcinoma of the colon or rectum.

Treatment.—Because each of the multiple polyps bears malignant potentialities, almost every individual with familial polyposis will sooner or later develop carcinoma of the

to the remainder of the rectum; after the latter operation removal of benign lesions in the remainder of the rectum by fulguration through a proctoscope.

The rectum must be resected when the rectal polyps are very large and too numerous for safe removal by fulguration.

MALIGNANT TUMORS OF THE COLON

Carcinoma of the colon is apparently becoming more frequent because of two factors: the increasing accuracy of diagnosis and the lengthening span of life. During the 20th

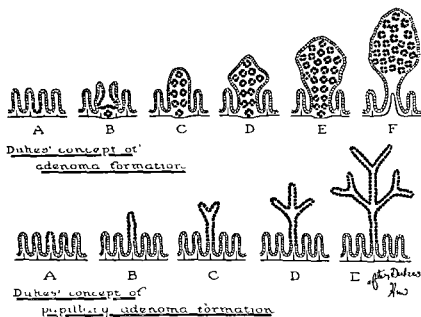


Fig 342—Dukes' concept of adenoma and papillary adenoma formation

bowel. For this reason prophylactic excision of abnormal mucosa is necessary. Not only does the individual concerned have to be treated, but the other members of the family should be thoroughly investigated.

There are two principal methods of treatment of multiple polyposis before malignant degeneration has occurred:

1. Excision of the cecum, appendix, colon, and rectum, with the establishment of a permanent ileostomy.
2. Excision of the colon and upper portion of the rectum, with anastomosis of the ileum

century, this has increased by 15-20 years, so that a larger number of people are entering the cancer age.

Carcinoma of the large bowel is a common disease, accounting for 10% of all cases of cancer.

The site of the growth in the large bowel depends on the accurate definition of the recto-sigmoid. In order to overcome individual interpretation of this area, it is better to consider the colon and rectum as one unit. The following table shows the proportional involvement by site in this hospital.

COLON

DISTRIBUTION BY SITE

Cecum	7%
Ascending colon	10%
Transverse colon	14%
Descending colon	5%
Sigmoid	21%
Rectosigmoid	7%
Rectum	36%

Etiology.—The origin of cancer of the colon is not known, but there are several predisposing conditions to cancer in this site. Of these, the most important is familial multiple polyposis, which invariably becomes malignant. Single isolated polyps may occasionally undergo malignant change, while pseudopolyposis becomes cancerous in approximately 2-3% of cases. Diverticulitis and cancer of the colon coexist in from 3-5% of cases.

Pathology.—There are two main types of carcinoma in the colon. The commoner is the annular stenosing obstructive growth which is more frequent on the left side of the bowel. The proliferative cauliflower-like papillary variety occurs characteristically on the right half of the colon. They are both adenocarcinomas. Colloid or mucoid degeneration occurs in about 5% of cases.

Benign polyps may have a malignant change at their tip which can be called a carcinoma-in-situ. Invasive carcinoma arising in the polyp will show changes in the ground substance, but the growth may still remain outside the lamina propria. If the growth occurs at the base rather than at the tip of the polyp, it is more dangerous for two reasons: it is nearer to the lamina propria and, second, local removal is liable to failure because a portion of the malignant growth may be left. The next boundary line is the muscularis mucosae, then the layer of circular musculature. Malignant cells may be picked up in either the lymphatics or blood vessels from the lamina propria on ward. The grading of pathologic types by Broder's classification or Dukes' stages is of some value. No staging can be accurately done without pathologic assistance. The present convention of staging as applied to the rectum could be conveniently applied to the colon.

Stage A—Confined to the mucosa
1 Carcinoma in-situ
2 Carcinoma invasive

Stage B—Involving circular musculature

Stage C—Local lymphatic glands involved but removed surgically
C—More distant lymphatic glands involved possibly surgically removable
Stage D—Generalized dissemination of malignant disease

The spread of carcinoma of the colon is usually by the lymphatics, but the other routes should not be forgotten; namely, by direct spread, along the lumen, implantation, and by means of the blood stream carrying the cells to the liver. This latter method is not quite as common in the colon as in the rectum. It is earlier in the flat sessile growths than in the papillary type. Metastases may also be found in the peritoneum and in the lung.

The rapid venous route of spread is more common than is generally realized. The figure of 15% given 10 years ago has been increased to 40%, and by recent cytologic techniques it was further raised to 60%. It may even be higher. In the hope of controlling these blood-borne emboli, thio-TEPA is being administered intravenously at the end of the operation in an experimental series in Chicago.

Passage along the lumen can be prevented by careful handling at operation.

Recurrence of the growth occurs at the line of the anastomosis. This is a result either of inadequate removal or of implantation of cells at the time of operation. The latter complication can be avoided by tying the bowel with umbilical tape above and below the tumor, and irrigating the two cut ends for 5 minutes with a suitable antiseptic fluid before performing the end-to-end anastomosis.

Wangensteen has recently been advocating the second look, which is a deliberate re-exploration of the abdomen after 6-9 months in those cases of carcinoma of the colon that showed evidence of lymph node involvement at the time of the first procedure.

Clinical Signs and Symptoms.—Carcinoma of the colon is a relatively silent disease, and the early symptoms are vague and indefinite. Consequently it is frequently diagnosed only in the late stages. Vague complaints, such as weight loss and fatigue, must be followed up thoroughly. There is usually some type of dyspepsia, constipation, or mild alteration in the bowel habit, such as a frequency in the desire to defecate. Diarrhea, either spurious or blood

stained, is always worthy of careful investigation, as is anemia, especially if it does not yield readily to medical treatment. Hemorrhage, whether as bright red blood or melena, is classically the commonest symptom. Patients with early hemorrhage have the best prognosis. Pain is usually late but may be the presenting symptom. When cramplike in nature, it is evidence of early obstruction; when spastic or associated with tenesmus, there may be a growth in the distal sigmoid or rectum. Steady pain in the right iliac fossa may be due to distention of the cecum, with a competent ileocecal valve and distal obstruction. A tumor may be felt by the patient or discovered accidentally on routine examination. Any mass without pain in the course of the large intestine must be regarded with grave suspicion.

Right Half of the Colon.—In this segment of the bowel, large cauliflower-like growths are common. These interfere with the normal function of absorption and give rise to anemia, intoxication, or a mass, which may be felt on examination. There may be a mild alteration in bowel habit, and bleeding may occur which should stimulate further investigation. Carcinoma of the transverse colon usually behaves like that in the left half of the colon.

Left Half of the Colon.—Carcinomas in this portion cause the obstructive syndrome. The scirrhous growth spreads in the wall, causing a gradual narrowing of the lumen with the onset of acute superimposed upon a chronic obstruction. This is characterized by visible peristalsis, borborygmi, distention of the abdomen, and pain in the right iliac fossa due to enlargement of the cecum when the ileocecal valve is competent. Progressive constipation is the rule, while blood in the stool, although less common, is of more dramatic significance.

The stools may be small in caliber or contain blood, either macroscopically or microscopically. Pus in association with blood suggests a carcinoma. This vague pattern of many signs and symptoms should be thoroughly investigated in order to establish, or disprove, the presence of malignancy.

Diagnosis.—The diagnosis is made by a careful history. Any alteration in the bowel habits of a patient of the older age group immediately indicates a clinical examination of the abdomen for the recognition of any pal-

pable masses and the noting of any abdominal distention.

This should be followed by digital and sigmoidoscopic examination, although most colonic growths are above the reach of the examining finger and even the sigmoidoscope. Therefore an accurate diagnosis requires a barium enema.

Treatment.—The treatment is entirely surgical. Radiation therapy has little effect, so the only hope is early and complete removal. The preoperative preparation of patients with carcinoma of the colon has been gradually improved due to control of anemia by blood transfusions, of protein metabolism by concentrated feedings, both by mouth and intravenously, relief of distention by Miller-Abbott tube, and the sterilization of the bowel by chemotherapeutic agents. Castor oil and magnesium sulfate should be avoided in preparation of the bowel in ulcerative colitis, regional enteritis, and intestinal obstruction, as violent purgation increases the mortality. In cases of carcinoma, milk of magnesia, 2 drams, should be given. The nonabsorbable sulfonamides are given for 5-6 days before operation, 3 days before operation one of the antibiotics affecting the gram-negative organisms is added. Streptomycin 2 Gm a day, or neomycin 1 Gm every 4 hours for 1 day only, may be used. These are all given by mouth in powder, capsule, or tablet form.

The synergistic action of the sulfonamides and antibiotics will control the flora and allow healing of the anastomosis. The bacterial content of the feces, both aerobic and anaerobic, can be considerably reduced, the only caution being that this should be limited to a 7-day period.

OUTLINE OF PREOPERATIVE PREPARATION OF THE COLON

1 "Sterilization" of bowel:

- | | |
|---------------------------|---|
| a. Sulfathaladine | 2 Gm q 4 h for 5 days |
| b. Achromycin or neomycin | 2 Gm b.i.d. for 2 days
1 Gm t.i.d. for 1 day |
| c. Menadione | 5 mg b.i.d. for 4 days |

2 Decompression and cleansing:

- | | |
|-----------------------------------|-------------------------------|
| a. Mineral oil | 1 oz b.i.d. for 5 days |
| b. Magnalax | daily p.r.n. |
| Avoid castor oil | |
| c. High protein, low residue diet | for 2 days prior to operation |
| d. Nasogastric suction tube | on day of operation |

dicating a painful fissure; or a thrombosed external hemorrhoid with its blue, swollen appearance may be seen. In this case further examination must be postponed until after the patient has been anesthetized. Apart from these two painful conditions, digital examination should always be performed. The patient may

the notch between the superficial and deep parts of the external sphincter can usually be felt. Crypts, polyps, and fistulas can be detected. Continuing the rectal examination, the prostate and its consistency is determined in the male, while the pelvic organs are examined in the female. It is convenient to complete

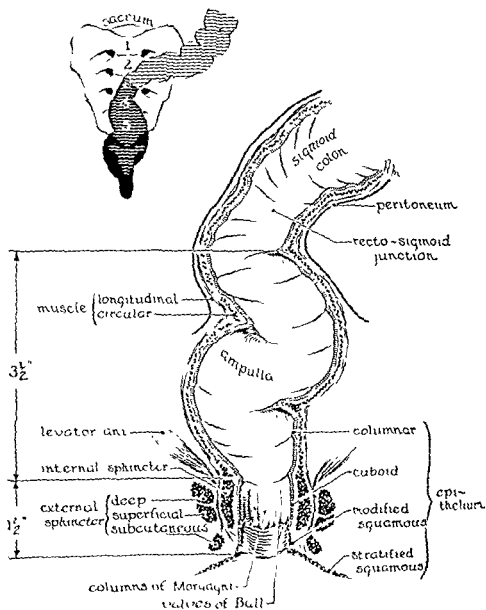


Fig 345B—Diagrammatic anatomy of the rectum

be postured on the left side, or in the knee-chest position. Gentleness and reassurance are necessary at all times. When the finger has been fully introduced, the puborectalis muscle can be palpated posteriorly and laterally and the tone of the sphincters determined. Even

bimanual pelvic examination, especially in children and virgins, by this method. At this time, feces may be found in the rectum; if so, a sample should be removed and examined for color, blood, etc. Next, the finger is swept laterally and posteriorly to examine the coccyx

and lower sacrum for mobility, pain, or other abnormalities. Internal hemorrhoids cannot be felt. *However, more than half of the carcinomas of the colon and rectum lie within reach of the examining finger.*

Examination should be completed by the use of the anoscope, proctoscope, or sigmoidoscope. Before completing this instrumental examination, swabbing of the mucosa may be performed or a specimen of stool collected for culture of organisms. A biopsy may be taken from an ulcer or growth, or fulguration of a polyp may be carried out through the instrument.

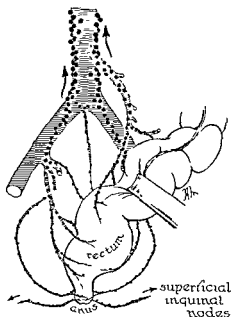


Fig 346—Lymphatic drainage of the rectum

CONGENITAL ABNORMALITIES

(See Chapter 30, Pediatric Surgery)

FISSURE-IN-ANO

This painful condition is usually associated with constipation. The scybalous mass tears the anal valve usually in the midline posteriorly, i.e., 6 o'clock, down to the anal verge, thus leaving a raw area or fissure with the subcutaneous sphincter exposed. The patient complains of an internal sharp, stabbing pain on defecation which is caused by spasm of the sphincter. This is often followed by the

passage of a few drops of blood. On examination there is a hypertrophied tag of anal tissue (sentinel pile) usually posteriorly, but in about 10% of cases anteriorly, in the base of which may be seen the lower end of the fissure.

The treatment consists of softening the stool by liquid paraffin and injecting the patient with a local anesthetic in an oil base which should afford relief of pain for approximately a week. If this fails, then under anesthesia, the sphincter is dilated up to four fingers in size, and the fissure and sentinel pile are excised, usually combined with partial severance of the subcutaneous external sphincter.

HEMORRHOIDS

External Hemorrhoids

External hemorrhoids are varicosities of the external hemorrhoidal veins. These form enlargements around the anal margin, and occasionally become thrombosed. The occurrence of this thrombosis is marked by acute pain and the appearance of a firm lump at the anal margin. On examination the overlying skin is bluish in color and there is a firm tender mass, which is nonfluctuant.

Treatment.—The pain and discomfort can be relieved by the moist heat of hot baths or compresses. An incision under local anesthetic will enucleate the dark blue clot and give immediate and dramatic relief.

Internal Hemorrhoids

Internal hemorrhoids consist of the enlargement of the internal hemorrhoidal venous plexuses, the main sites being at 3, 7, and 11 o'clock. The hemorrhoidal plexus is one of the main connections between the portal and systemic circulation, the others being the lower end of the esophagus, around the umbilicus, and between the renal and lumbar veins. Patients are predisposed to hemorrhoids by the inheritance of weak-walled veins and also by the absence of valves in the internal hemorrhoidal veins. The predisposition to hemorrhoids is aggravated by an increase in intra-abdominal pressure from straining at stool, constipation, pregnancy, and pelvic tumors. Hemorrhoids may also occur in portal hypertension.

Symptoms.—By far the commonest symptom is bleeding, usually at the time of defecation. The patient may also complain of prolapse, that is, the piles protrude and may have to be replaced manually. Discharge and irritation may accompany hemorrhoids, while pain is only associated with the complication of prolapse or thrombosis. They may occur at any time from adolescence onward. On examination the bluish bulging mucosa is readily recognized in the three primary positions, although it may vary in size. The examiner must be careful to distinguish between prolapsing internal hemorrhoids and prolapse of the rectum. This can be done easily, by asking the patient to strain. If a complete ring of mucosa appears outside the anal margin, prolapse of the rectum is obvious.

For convenience in description, first degree hemorrhoids are those that bleed but do not prolapse. Second degree cases prolapse and return again with or without bleeding. Third degree piles prolapse and become irreducible.

Complications.—The commonest complication is anemia, due to both the amount and duration of bleeding. The second complication is strangulation and thrombosis which occurs only with prolapsing hemorrhoids. Thrombosis may be followed by infection, accompanied by a sharp throbbing pain, or ulceration with a persistent discharge. Fortunately portal pyemia is now practically unknown.

Treatment.—This may be considered from three points of view. In mild cases and young individuals, *palliative treatment* may be all that is required. This consists of cold compresses combined with an analgesic ointment or suppositories. In those cases in which operative treatment is contraindicated, or in the milder cases, *injection therapy* may be carried out. The actual selection of cases is of fundamental importance in this treatment, the most suitable being first or second degree piles with no fibrosis and no external hemorrhoids. It is an unsuitable method for prolapsed third degree hemorrhoids, for fibrosis of internal piles, or for the compound external-internal variety. Injection therapy is carried out with a 5 ml Luer-Lok syringe and a long needle with a shoulder less than $\frac{1}{2}$ " from the tip. Three milliliters of 5% phenol and 5% menthol in almond oil are injected into the submucosa

near the base of the internal hemorrhoid, injecting one at a time at weekly intervals.

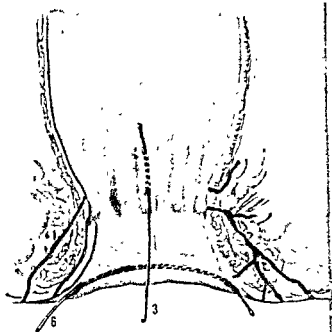
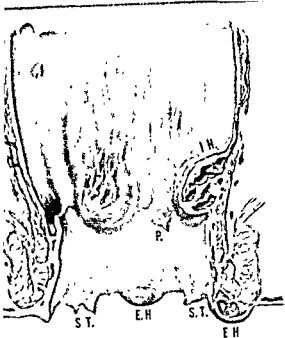
Complications of this treatment are remarkably few, but abscess formation and sloughing are occasionally encountered. If the injection is placed too low, there will be considerable pain from the phenol solution in contact with the anal sensory nerves.

Operative treatment is indicated in second and third degree hemorrhoids. It is contraindicated in pregnancy, in the presence of infection or gangrene, and in such general conditions as portal hypertension. A sigmoidoscopic examination is performed to exclude any other local organic cause. The hemorrhoids are dissected with a small flap of skin, demonstrating the subcutaneous external sphincter, and leaving a bridge of normal mucosa between the hemorrhoids. The base is ligated by transfixion, the bleeding points are carefully tied, and an absorbable hemostatic agent may be used as a pack. Early ambulation, hot baths, and mineral oil by mouth help toward a comfortable convalescence. Digital dilatation may be necessary to avoid a stricture. Postoperative pain may be prevented by injecting a local anesthetic in oil into the area at the time of operation.

Postoperative Complications.—Retention of urine is common, particularly with the use of spinal anesthesia. Secondary hemorrhage occurs occasionally, but careful technique will minimize this complication. Stricture formation may result if too much tissue is removed at the primary operation. This may often be prevented by repeated dilatations in the early postoperative period. Incontinence may occur as a result of damage to the external sphincter by excessive stretching or section or by the removal of too much mucosa, with consequent loss of sensation.

PROLAPSE OF THE RECTUM

Prolapse of the rectum is divided into two types: partial, consisting of the mucous membrane only, and complete, involving the entire thickness of the rectal wall. *Partial prolapse* usually occurs at the extremes of age. In children, it is due to a straight sacrum or to a lack of fat in the hollow of the sacrum and the ischio-rectal fossa. The precipitating causes are usually constipation, straining, or diarrhea. In



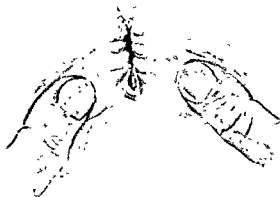
I.H. Internal Hemorrhoids
 E.H. External Hemorrhoids
 S.T. Skin Tags
 C. Cryptitis
 P. Papillitis

1 and 2. External Fistulae
 3. Complete Internal Fistula
 4. Blind Internal Fistula
 5. Branching Type Fistula
 6. Horseshoe Fistula

F. Netter M.D.



RECTAL PROLAPSE



FISSURE-IN-ANO

Plate 33.—Common Anorectal Lesions.

adults, partial prolapse is often associated with hemorrhoids and is always the result of straining efforts common with constipation or enlargement of the prostate. *Complete prolapse* is rare. It is more common in females, occurs in both stout and thin persons, and should be looked upon as a sliding hernia of the pouch of Douglas.

Symptomatology.—There is slight bleeding after straining and discomfort of a mild type. Complete prolapse may be caused at any time by straining. There is a mucous discharge, incontinence, and regulation of bowel habit is difficult, so that the patient usually has to wear some pad or support. On examination, inspection is of the greatest importance. A swab is held over the anus so that there will be no incontinence when the patient is asked to strain. The rectum sometimes protrudes as much as 18". Palpation reveals the loss of sphincter tone. There may be ulceration of the mucosa, and even strangulation of the protruding mass, leading to irreducibility and gangrene. The diagnosis is easy but may be missed unless the patient is asked to strain. It is important to see the full extent of the prolapse, to replace the bowel as soon as it comes out, and then to decide on the line of treatment.

Treatment in Children.—This is conservative. Formal attempts at bowel training, such as leaving the child straining on the toilet for prolonged periods, may precipitate or aggravate the condition. The child's health must be improved. The specific treatment is reduction of the prolapse and maintenance of the reduction by strapping the buttocks together. Bowel training must be postponed until the condition is cured. Surgical treatment is rarely indicated except for those children who have a non-functioning anal sphincter or a defect in the levator ani muscle sling.

Treatment in Adults.—In *partial* prolapse associated with hemorrhoids it is only necessary to do a hemorrhoidectomy. The injection of 5% phenol in almond oil into the submucosa is helpful. In *complete* prolapse of the rectum, operation is advisable. Gabriel advocates the incision of the mucosa around the anal canal with amputation of the prolapsing portion of bowel through a *circular incision* and subsequent anastomosis. The second

method is that of Roscoe Graham: the abdomen is opened, and the floor of the pelvis is reconstructed; the rectum is then sutured to the pelvic floor, thus preventing prolapse from above.

Unfortunately both of these methods have a fairly high incidence of recurrence, because the underlying causes are still present. In order to reduce the recurrence rate, an addition to the method of Gabriel is advisable, namely, suturing the levatores ani to form a tighter sling.

CRYPTITIS

The small crypts behind the valves of Ball and between the bases of the columns of Morgagni frequently collect infected material, and these inflamed pockets are responsible for anal spasm, pruritus, pain on defecation, and even frequent bowel movements. The treatment consists of hot sitz baths and dilatation of the sphincter. The crypts should be exposed by a blunt hook and the free edge cut to give adequate drainage. The infection not infrequently burrows under the mucosa, causing a submucosal abscess.

ANORECTAL ABSCESS

Infection may be localized under the mucosa of the rectum or of the anus, as described under cryptitis, or the infection may be outside the muscular layer, either above or below the levator ani. Cases in which the infection is above are usually due to diverticulitis or ulcerative colitis, while those below originate in the ischio-rectal fossa. These abscesses and their situation are extremely important in themselves, but they become more so when considered from the point of view of their complications, of which the main one is *fistula-in-ano*.

The commonest type of anorectal abscess is the *perianal abscess* which may arise from the crypts, hair follicles, or sweat glands, associated with pruritus or systemic infection. The patient complains of pain on defecation, which is persistent and steady, and gradually increases in intensity until he cannot sit, stand, walk, or lie down without pain. The examination is difficult because the sphincter is in spasm and the induration is frequently masked. Examination under an anesthetic is necessary to deter-

mine the exact site of the abscess, and incision and adequate drainage should be carried out in order to prevent tracking of the abscess and fistula formation.

SUBMUCOUS ABSCESS

The mildest form of anorectal abscess is a submucous collection of pus, usually on the lateral wall of the rectum. It is often caused by abrasions but may follow thrombosis, injection of hemorrhoids, or cryptitis.

Treatment.—These abscesses may rupture by themselves into the rectum or anal canal; otherwise, they should be incised and drained and the edges cut to saucerize the area.

ISCHIORECTAL ABSCESS

The loose cellular tissue of the ischiorectal fossa may become infected from the rectum or skin, or infection may be brought there by the blood stream. The abscess may communicate with the rectum between the superficial and deep sphincters. It is very slow in development, with gradually increasing discomfort, deep throbbing pain, malaise, and a temperature up to 103° F. There are fullness and tenderness on examination, both externally and by rectum.

Treatment.—Under anesthesia an antero-posterior incision is made if the abscess is near the surface. If deep, a T-shaped incision is required, the edges are removed to saucerize the wound, and the cavity is packed. Postoperative sitz baths are instituted twice daily, and the wound is allowed to granulate.

PELVIRECTAL ABSCESS

Pelvirectal abscess is the rarest variety and involves the space above the levator ani. Infection usually arises from inside the pelvis. It may rupture into the rectum or through the levator ani. These patients have pelvic pain or discomfort, malaise, fever, and a marked leukocytosis.

Treatment.—The abscess should be drained through the ischiorectal fossa. It should be treated early in order to prevent complications, particularly high fistula-in-ano.

FISTULA-IN-ANO

Fistula-in-ano invariably results from a rectal abscess which burrows its way to the surface, producing a chronic granulating track. The track passes between the anal canal and the skin. Fistula-in-ano may be complete or incomplete. The complete variety has a fistulous connection between the skin and rectal lumen; there may be intermittent leakage of pus or feces. The incomplete form discharges either into the lumen of the bowel or through the perianal skin.

Etiology.—It is a disease of adult life, commoner in males. The infection may begin as an abscess in the submucosa and burrow its way to the surface as an ischiorectal abscess or, more rarely, from diverticulitis or ulcerative colitis.

Classification of Fistula-in-Ano.—

1 *Submucosal and subcutaneous fistulas*. These occur in about 15% of cases.

2 *Low anal fistulas*. Here, the track may be demonstrated by means of a probe passed from the skin to the anal canal, deep to the subcutaneous external sphincter. These make up about 75% of fistulas.

3 *High anorectal fistulas*. These usually begin from an ischiorectal abscess which has drained into the rectum as well as externally. They comprise about 10% of cases.

Diagnosis.—

Inspection.—Near the anus there is a small pimple from which pus may be discharging. The anterior fistulas are usually single, while those posterior frequently have multiple openings and side tunnels.

Palpation.—On inserting the finger into the rectum and squeezing downward, pus may be extruded, and with the passage of a probe, the track may be outlined. Should one suspect a complicated fistula, the injection of radiopaque Lipiodol is of assistance in estimating the complexity in a given case.

The diagnosis of fistula-in-ano presents little difficulty. The important point is to establish the bacteriologic cause, whether the lesion is due to pyogenic organisms or to the tubercle bacillus or actinomycetes. In tuberculosis there is usually a history of pulmonary infection, and if this is suspected, the specimen must be examined bacteriologically and pathologically.

ing. Some patients may have a relaxed sphincter with a continual leak of mucus. Others have excoriation of the skin which is heaped up in folds, with small fissures or ulcers between the folds. This is a warm, moist area suitable for the growth of parasites and mycotic infection. Lastly, there are those of obscure origin due to allergy or even psychoneurosis.

The investigation of a case in which the patient complains of itchiness in the anal region demands a thorough history and general examination, as well as a careful local examination. It is advisable to look especially for crypts, excoriation of the skin, and any other local cause, as well as careful examination for fungi, etc. The treatment is directed toward the following:

1. Hygienic measures Sitz baths twice daily, dusting powder, clean, well-fitting underclothes and clean bed linen
2. Dietetic measures If diet is a suspected causative agent, various elimination procedures may be tried
- 3 Medical therapeutics Treatment of constipation by mild laxatives, such as liquid paraffin, and attention to nutrition
- 4 Surgical treatment is reserved for intractable cases and consists of careful preparation of the patient for 2-3 weeks before admission with local antiseptics such as methylene blue or potassium permanganate. The definitive operative therapy provides wide excision of the skin and all the redundant folds in either a butterfly shape or sunflower pattern. This cuts the cutaneous nerves and enables the denuded areas to heal by granulation tissue and become smoothly epithelized

PROCTITIS

Proctitis is a comprehensive term, including all the types of inflammation of the mucosa of the rectum. Simple, catarrhal, nonspecific proctitis is a localized form of colitis. It may be due to vitamin deficiencies, endocrine dysfunction, allergy, vasomotor disturbances, or even psychologic causes

Nonspecific

Acute proctitis is an acute hyperemia of the rectal mucosa with edema, vasodilatation,

and even hemorrhage. There is a granular appearance of the mucosa, with small, pinpoint ulcers. Digital examination of the rectum reveals that the wall is thickened and the lumen narrowed. The proctoscope shows a red, granular mucosa, which bleeds easily. Bacteriologic culture may reveal streptococci, staphylococci, and hemolytic streptococci, as well as *Bacillus coli*.

Chronic proctitis is the more chronic form of the disease and may be either hypertrophic or atrophic.

Irradiation proctitis occurs after deep x-ray therapy to the pelvis or radium treatment for carcinoma of the cervix. It consists of a hyperemic, swollen mucous membrane with excess of mucus, and it is usually accompanied by rather severe steady pain. General treatment consists of liquid paraffin for soft stool formation and cleansing enemas. In more severe cases, a colostomy may be necessary for the relief of the recurring hemorrhage or stenosis

Specific

Bacillary or amebic dysentery must be confirmed by the growth and recognition of the causative organism

Tuberculous proctitis is usually associated with an active pulmonary lesion and may be complicated by tuberculous anal fistulas

Gonococcal proctitis is an acute proctitis with hyperemia of the mucosa and thick creamy pus. It must be bacteriologically diagnosed

Syphilitic proctitis. A primary chancre in the rectum may occur and usually presents as a small ulcer

Venereal proctitis is a lymphogranuloma venereum which may appear as an abscess, a stricture, or a small ulcer.

Treatment.—The treatment has been greatly simplified since the introduction of antibiotics. General medical measures may be used, such as sitz baths, oil enemas, and soothing ointments. The nonabsorbable sulfonamides will usually control acute nonspecific proctitis, while penicillin is specific for gonorrheal and syphilitic proctitis. Tuberculous proctitis is much more difficult to treat, demanding both general and local care. Streptomycin or its kindred antibiotics are efficacious for lymphogranuloma venereum

INJURIES OF THE RECTUM

Injuries of the rectum are not very common in civilian practice but increase during war-time. They may be divided into two classes—operative and traumatic. Accidents to the rectum occur during operations on the urethra, prostate, vagina, or uterus. The sigmoidoscope or enema nozzles may perforate the rectum during examination. A surrounding cellulitis or peritonitis may result. Recognition of such injuries usually occurs at the time of the accident, and the opening into the rectum can then be repaired immediately.

Gunshot wounds of the pelvis may affect the intraperitoneal or extraperitoneal portion of the rectum.

In crush injuries of the pelvis, a spicule of bone may perforate the rectum.

Rectal perforation also occurs from swallowed foreign bodies or from those introduced from below. These range all the way from a swallowed bone that has lodged transversely in the anal canal to the infinite variety of objects inserted into the rectum by children and lunatics.

Treatment.—Any foreign body should be carefully removed, and the rectum should be examined to make certain that there has been no damage to the wall. An anesthetic is frequently necessary as otherwise the procedure is extremely painful. Should there be perforation of the wall, a defunctioning colostomy is necessary until healing has occurred, then the colostomy may be closed.

STRICTURE OF THE RECTUM

Congenital stricture is mentioned in the section on imperforate anus (see Chapter 30). Post-traumatic stricture may follow perforating wounds of the rectum, and postoperative stricture may occur after hemorrhoidectomy or low intestinal anastomosis. Irradiation proctitis may result in a stricture formation. Lymphogranuloma inguinale is another common cause of stricture formation. This is a venereal infection caused by a filtrable virus and can be diagnosed by the Frei antigen test. Local examination of the rectum reveals a definite fibrous stricture, which is usually long and tubular,

associated with perirectal inflammation. In all cases of stricture the patient complains of progressive difficulty of defecation, straining at stool, and ribbonlike stools.

Treatment.—

1. *Dilatation* is done repeatedly using rectal bougies of graduated sizes.

2. *Operation.* Internal proctotomy consists of cutting the fibrous stricture in 5 or 6 places.

3. The patient may require a *colostomy*, usually on the left side. Many cases of benign stricture of the rectum may be prevented by the early use of antibiotics in the treatment of the specific cause.

4. The extensive stricture of lymphogranuloma inguinale may require an abdominoperineal resection.

BENIGN TUMORS OF THE RECTUM

The commonest tumor of the rectum is a benign adenoma. The connective tissue tumors such as lipoma, fibroma, myoma, lymphoma, and angioma are rare. An *adenoma* is a small swelling, usually about the size of a pea which later enlarges up to about 3 cm. in diameter. It may be sessile or pedunculated and may be the site of malignant changes. The usual symptomatology is bleeding, discharge, and occasionally constipation or diarrhea. In children, the adenomas which usually cause bleeding are single and after removal cause no further trouble. In adults, the adenomas vary in size, number, and situation, as well as in symptomatology. Diagnosis is confirmed by sigmoidoscopic examination, when the polyp may be seen. It can then be grasped by forceps and removed by either a snare or cautery. The only point that should be stressed is that if there is a hereditary influence or the papillomas are multiple, then it is wise to have a radiographic examination in order to be sure that they do not extend along the bowel. *Multiple polyposis* of the colon, no matter what the age, is definitely a premalignant condition, and treatment is total colectomy. (See Familial Polyposis.) *Anal warts* or multiple papillomas of the skin around the anus occur. Treatment consists of removal by scissors under local anesthesia. The radiating wounds are then packed with petrolatum gauze.

CARCINOMA OF THE RECTUM

Incidence.—Cancer of the rectum and lower sigmoid comprises approximately 7% of all malignant tumors of the body. Men are affected more commonly than women in the ratio of 3 to 2. While no age is exempt, the most common decade for the occurrence of this form of cancer is 50-60, although many cases have been reported under the age of 30 years.

Etiology.—Though the true etiology of cancer is as yet unknown, certain lesions may be considered premalignant in the rectum. This is particularly true with multiple or single polyps.

Pathology.—A carcinoma of the rectum, excluding that of the anal canal, is an adenocarcinoma. It is in its early stages a local disease which begins as a local change in the epithelium or as a malignant change in a pre-existing adenoma. Varieties of adenocarcinoma which have been described include medullary, scirrhous, mucoid, and papillomatous.

Of more importance is the microscopic grading of the neoplasms, as has been described by Broder, who has divided them into four groups according to the degree of differentiation of the cells and the number of mitoses. The more undifferentiated the cells, the more malignant the tumor.

Malignant lesions of the rectum spread by direct extension and venous and lymphatic channels. It has been pointed out by Dukes that carcinoma of the rectum extends by direct spread through the rectal wall, and that lymphatic spread does not take place until the growth has reached the extrarectal tissues. His classification divides them into A, B, and C groups. A cases are those in which the growth is limited to the bowel wall (15%), B cases have involvement of the extrarectal tissues (30%); and C cases are those with metastases in the lymphatic nodes. This latter group may be further subdivided into near (C_1) and more distant (C_2) node involvement. The intramural lymphatics cause a spread within the wall of the bowel but for only a short distance above or below the lesion.

Of much more importance are the extramural lymphatic channels. Three methods of

extension occur: (1) downward, (2) laterally along the levator ani muscles, (3) upward to the retrorectal nodes, and then along the superior hemorrhoidal and inferior mesenteric vessels toward the periaortic nodes.

Venous spread, while much more serious than lymphatic involvement, occurs in 20% of cases.

Symptoms.—Carcinoma of the rectum in its early stages gives symptoms which may be slight or even absent, and the lesion may occasionally be discovered on routine physical examination. However, the most frequently encountered symptoms are bleeding, alteration in bowel habits, and pain.

Blood in small amounts, accompanied, as a rule, by the passage of excessive mucus in the stool, is the most common complaint. The blood is bright red and may either be mixed with the stool or may be streaked on the surface. The actual total blood loss is small, and anemia is not as marked as with carcinoma of other parts of the colon.

Alteration in bowel habit may be the earliest symptom. This may take the form of constipation or diarrhea. Tenesmus may be a prominent feature.

Pain is a late symptom and denotes a far advanced malignancy.

It is obvious that none of these symptoms is diagnostic of rectal cancer, but their presence should direct one's attention to a thorough examination of the rectum and colon.

Treatment.—The diagnosis having been established by biopsy, choice of treatment will depend on the site and size of the tumor. The results of radiation therapy have been disappointing, so that the only treatment is surgical removal.

For the noninvasive carcinoma confined to the mucosa and for carcinoma-in-situ, particularly in a polyp in the rectum which is within reach of the proctoscope, local excision with regular follow-up examinations afterward is a safe treatment. Invasive carcinoma, no matter how small, must be treated by radical excision. Carcinomas situated above the peritoneal reflection are usually classified clinically as situated in the rectosigmoid. In these cases, if the patient is not too fat or the pelvis too

small, it is possible to do a resection with end-to-end anastomosis. When the growth is at the level of the peritoneal reflection and below, then it is wiser to perform the abdominoperineal resection of Miles with terminal colostomy. This operation has been modified by the use of the two team synchronized combined abdominoperineal operation. The advantage of this method is wider

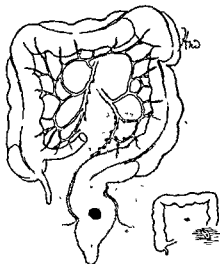


Fig 348—Classical abdominoperineal resection of Miles, for growths of Stages A, B, and C₁ of the rectum below the peritoneal reflection. The stippled areas represent the extent of the resection for primary lesions indicated by the black dots.

excision, and more extensive growths can be removed because direct vision from above and below at the same time is possible. Some authors advocate a proctosigmoidectomy with preservation of the anal sphincter. This method is suitable for selected cases, groups A and B only, with the growth at least 3" above the anal margin, and where the architecture of the sigmoidal arteries permits adequate mobilization of the sigmoid down to the anus. For advanced cases there is considerable debate whether an abdominoperineal operation should be performed when metastases are present in the liver. However, if the local growth can be removed, the last days of the patient will be more comfortable. In obstructed cases the obstruction must be relieved by means of a proximal colostomy, followed at a later date by abdominoperineal resection.

CARCINOMA OF THE ANUS

Carcinoma of the anus is comparatively comprising about 4% of rectal malignancy. It is squamous cell in type and produces stenosis of the canal. The diagnosis is easy because of pain, early bleeding, and the presence of an ulcer with hard everted edges, not accessible to the examining finger, and not visualized.

Adenocarcinoma may spread downward the rectum. Basal cell carcinoma is extremely rare in this region. Malignant melanoma can be distinguished from a thrombosed hemorrhoid, which can be done by biopsy.

Treatment.—The treatment of choice is surgical excision. Removal of the inguinal lymph nodes is sometimes debated but seems a wise insurance. Radiation therapy cannot be relied upon to control spread of disease into the hollow of the sacrum.

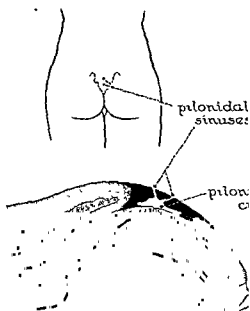


Fig 349—Pilonidal cyst and sinus

PILONIDAL SINUS (SACRO-COCYGEAL SINUS)

Pilonidal sinus is a sinus lined with modified squamous epithelium which is situated along the midline in the region of the sacrococcygeal junction. When the sinus track becomes blocked, it may go on to cyst formation. It may contain hair and retained secretions,

inflammatory changes frequently occur. The sinus opening may be single or multiple.

It is thought to arise from faulty development in the medullary canal or failure of union of the median raphe in this region.

Clinical Features.—In childhood, this condition is asymptomatic and consists of one or more small dimples in the midline. Symptoms do not, as a rule, develop until young adult life and are due to infection in the sinus which intermittently closes, fills up, and discharges again. Occasionally infection may spread and break out, causing new sinus openings. This may be precipitated by trauma, such as falls on the buttocks. It is more common in males.

Treatment.—In the acute stage abscesses should be drained. During the quiescent period all diseased tissue should be excised. If the resected area is not too large, the skin edges should be approximated, care being taken to obliterate all the dead space. So-called recurrences are due to inadequate primary removal of the sinus track.

An alternate treatment consists of marsupialization of the sinus track.

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Film Reference

Title	Running Time	Sound or Silent	Processible From
Abdomino-perineal Resection for Carcinoma of the Rectum (Illustrates technique of this operation as applied to treatment of carcinoma of the rectum) (1950) (By Richard B. Cattell MD Boston)	18 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn

Chapter 28

Female Genital Tract

George A. Simpson, M.D., and William R. Foote, M.D.

Gynecology is defined as the study of the diseases of women, and in practice the gynecologist's efforts are centered on the diagnosis and treatment of diseases of the female reproductive organs. This does not free him from considering the other systems of the patient which may create symptoms referable to the pelvis. Contrariwise, disease of the pelvic organs sometimes simulates very closely those of acute and chronic surgical conditions of the other systems.

The greater proportion of gynecologic practice is formed of patients for whom the treatment is medical. One must not lose perspective while reading this section which tends to place special emphasis on the disorders that do require surgical measures.

SURGICAL ANATOMY

The female reproductive system has been differentiated into the lower and the upper genital tracts, and for our purpose the *lower genital tract* includes the vulva, vestibule, vagina, and the vaginal portion of the cervix, the *upper genital tract* comprises the supravaginal portion of the cervix, the body of the uterus, the fallopian tubes, and the ovaries, including the supporting structures of each division.

The lower genital tract may be inspected and palpated in its entirety so that localized lesions are ordinarily diagnosed by these simple means, with bacterial studies, or tissue biopsy being used where indicated.

The upper genital tract is contained in the pelvic cavity, with the bladder anterior and the rectum posterior. While there are no superficial anatomic landmarks to aid one, these organs are usually readily palpated through the vaginal fornices or the rectum. With the other hand on the lower abdominal wall for counterpressure, it becomes only a matter of experience before the student becomes adept at diagnosis. There are pitfalls, of course, and these will be mentioned subsequently.

There are three anatomic features of significance: (1) As the *nerve supply* of the pelvic organs is incompletely understood, referred pain from pelvic disease is not definitive. There are apparently afferent pain tracts from the pelvic organs other than the presacral nerve, this fact, together with the multiplicity of the presacral nerve fibers sometimes present, suggests that presacral neurectomy will only give relief of pelvic pain in very carefully selected patients. (2) There is direct continuity between the vaginal orifice and the pelvic peritoneal cavity. (3) The pelvic peritoneum of the pouch of Douglas extends down to the upper fifth of the posterior wall of the vagina. In this area collections of pus, blood, endometriosis deposits, and metastases of malignant disease tend to accumulate. The pouch of Douglas can be drained by incising the posterior fornix (posterior colpotomy), and it is at this point that the culdoscope is inserted to obtain a direct view of the pelvic organs in selected cases.

GYNECOLOGIC HISTORY

When the patient visits the doctor, she has the history of her present complaint on the tip of her tongue, and it is usually wise to hear this in her own words before a more formal type of history is obtained.

The date of her last menstrual period is a useful starting point, and the information as to whether it came at the expected date and was normal in all respects is elicited. A more detailed menstrual history is then developed, and it is ascertained if there is irregularity, excessive loss, menstrual pain, intermenstrual spotting, painful intercourse, or abnormal vaginal discharge. Bladder and bowel habits and the presence or absence of abdominal pain are all ascertained as well as possible without too many leading questions. The history of all previous pregnancies, illnesses, and operations is important. These are often recalled vaguely, due in part to the paucity of detail given the patient by the doctor.

CLINICAL EXAMINATION

The abdominal examination is completely described in Chapter 32

The *pelvic examination* should be methodic and, with constant repetition which results from making it a routine part of the physical examination, the student is soon able to appreciate and detect the presence of abnormal findings

The patient, having emptied her bladder, is placed in the lithotomy position, with the hips well to the edge of the table. With good illumination, the external genitalia are inspected and then a speculum is introduced into the vagina and the cervix exposed. Smears for cytology are taken at this time with the Ayre spatula. The inspection of the cervix being completed, the vaginal walls are examined as the speculum is slowly withdrawn. A specimen of vaginal discharge is taken at this time for hanging-drop examination. With the gloved fingers lubricated, the urethra and the glands of Bartholin are gently palpated. Then, with the fingers depressing the perineum, the patient is asked to strain. This act usually exaggerates the relaxation and protrusion of the vaginal walls and causes descent

of the uterus if vault prolapse is present. Two fingers are then more deeply inserted into the vagina; the bimanual examination enables the cervix to be palpated and the position, size, and mobility of the uterus are ascertained. The internal fingers then deviate to the right and left fornices in an effort to outline the appendages. Normal appendages are not usually felt unless the abdominal wall is very thin and well relaxed. The posterior fornix is more easily examined by a rectovaginal technique which is ordinarily more disturbing to the patient than a vaginal examination. A simple rectal examination, however, will sometimes be as useful and less uncomfortable. In virgins, the latter is the only one that can be done unless the hymen is dilatable.

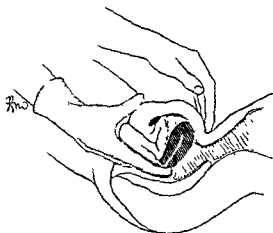


Fig. 350—Bimanual pelvic examination

Sometimes if the examination in the lithotomy position has been inconclusive, it is advisable to place the patient in the Sims' (left lateral) position and with a Sims' speculum repeat the procedure. If the patient is young or bashful this method may prove to be more acceptable, and it is the position of choice if the patient has to be examined in bed.

While the patient dresses, the examiner records the pelvic findings, correlates them with the history, and so arrives at a tentative diagnosis and the appropriate treatment.

On occasion a pelvic examination under an anesthetic is advisable, and this is usually combined with definitive treatment. Such examination should be routine before any laparotomy.



- A — Aorta
- B — Bladder
- BL — Broad Ligament
- Cx — Cervix
- CU — Corpus of Uterus
- E.F. — Epigastric Fold
- E.I.V. — External Iliac Vessels
- F.U. — Fundus of Uterus
- I.P.L. — Infundibulo-pelvic Ligament
- M.S.V. — Middle Sacral Vessels
- OL — Ovarian Ligament
- Ov — Ovary
- P.D. — Pouch of Douglas
- RL — Round Ligament
- S — Sigmoid
- S.U.L. — Sacro-uterine Ligament
- T. — Fallopian Tube
- U.F. — Utrachal Fold
- Ur. — Ureter

- CLV — Common Iliac Vessels
- ML — Mackenrodt's Ligament
- OA — Obturator Artery
- OC — Obturator Canal
- OF — Obturator Fascia
- RF — Rectal Fascia
- S.F.P.D. — Superior Fascia of Pelvic Diaphragm
- SP — Sacral Promontory
- UV — Uterine Vessels
- UVF — Uterovaginal Fascia
- VF — Vesical Fascia



Plate 34.—Pelvic Viscera and Support From Above.

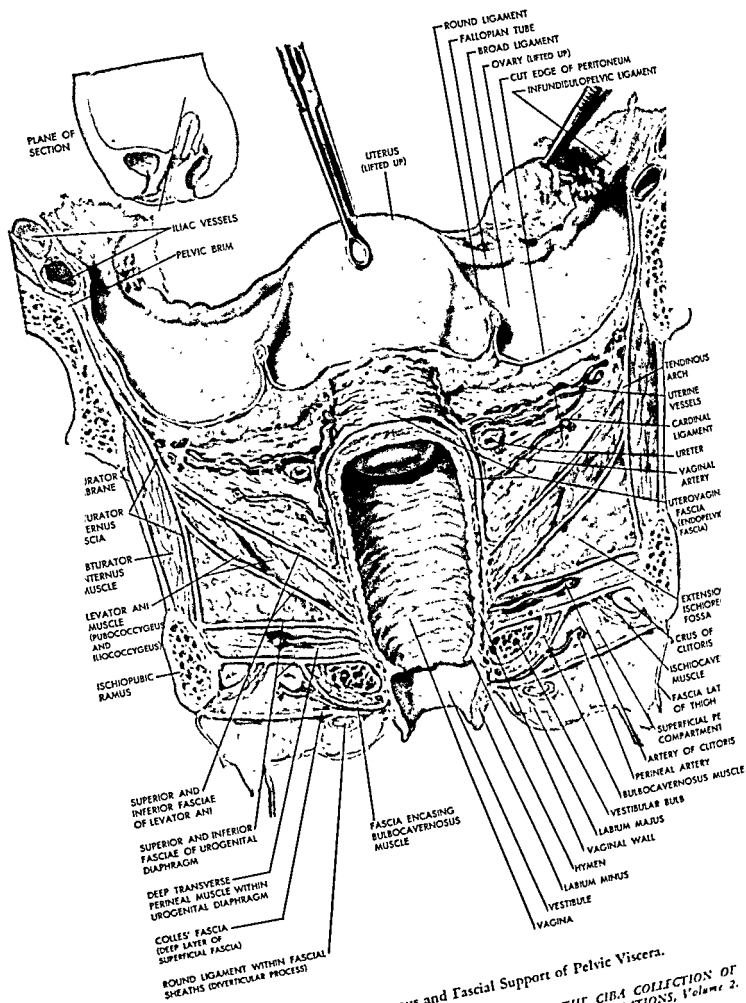


Plate 35.—Ligamentous and Fascial Support of Pelvic Viscera.

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Uterine Anomalies

When the 2 müllerian ducts remain separate in their entire course, *uterus didelphys* results; 2 uteri, 2 cervixes, and 2 vaginae with a single tube and ovary attached to each uterus are found. The *uterus bicornis* with one cervix and vagina, the *uterus with a rudimentary horn*, and the *uterus with a septum* restricted to the body of the uterus are other varieties.

Diagnosis.—The diagnosis of these anomalies is not usually made until they are disclosed by routine hysterosalpingography performed in the investigation of infertility, or a dilatation and curettage are performed for an abortion. When pregnancy occurs and progresses to the third trimester, obstetric complications are common.

Treatment.—This is only indicated and satisfactory for minor anomalies.

PELVIC INJURIES AND DISPLACEMENTS

Vulvar Injuries

The larger proportion of these are noted during delivery, but others are caused by direct force to the vulva as the result of a fall or kick. In the first instance a hematoma forms, and this, if it remains small, usually subsides spontaneously; if the bleeding continues into the soft tissues, the hematoma becomes very large and pain may be severe. Soon the thin mucous membrane of the lower vagina is eroded, and disturbing venous bleeding occurs. In such serious injuries, the patient must be treated for shock and, as soon as practical, anesthetized and the hematoma evacuated. The tissue distortion is usually so marked that no single bleeding point is apparent, but the bleeding can be controlled by packing both the cavity of the hematoma and vagina and then applying firm vulvar pressure. A retention catheter is usually inserted.

A puncture type injury may involve deeper pelvic structures with but little apparent external injury.

Vaginal Injuries

A laceration of the hymen at coitus causes slight bleeding which stops spontaneously.

Rarely, the hemorrhage continues and it is necessary to suture the bleeding point.

In recent years potassium permanganate crystals have been inserted high in the vault of the vagina in an effort to produce a criminal abortion. The copious bleeding, which was believed to be uterine in origin, resulted from a burn. On speculum examination the cervix is found closed and intact, and the ulcerated burn lesion is apparent. Remaining crystals must be washed out with saline solution and a firm vaginal pack inserted to control the bleeding. Sutures are often necessary.

Vaginal Fistula

Vesicovaginal fistulas are a result of operative or obstetric injury, pelvic neoplasm, or radium burn. The incidence which follows obstetric trauma is less than formerly; immediate injuries are usually successfully repaired at the time and those due to bruising and ischemia have largely been prevented by improved obstetric practice. *Fistulas due to radium therapy are not as common with modern techniques and are usually a sign of persistent neoplasm.*

Vesicovaginal fistulas cause urinary incontinence which is complete unless the fistula is small. The drainage is noted within 10 days of the pelvic surgery or the removal of a retention catheter if one had been used. The site of the fistula is found by injecting methylene blue into the bladder and observing the leak by direct inspection. The Sims' position and the use of a Sims' speculum give very adequate exposure for this test. Sometimes cystoscopic examination is needed to localize the fistula and is required before any repair.

A small fistula may heal spontaneously, but most will require surgical closure after local cellulitis and infection have been controlled. The fistulas due to malignant invasion or radiation burns are particularly difficult to repair.

Rectovaginal fistulas may develop after breakdown or infection in a third degree perineal tear, after trauma during a rectocele repair, as a result of pelvic abscess drainage either spontaneous or surgical, or as a result of malignant invasion or radiation of malignant pelvic growths. Passage of gas and liquid bowel content through the vagina is the usual

symptom. Direct observation usually discloses the level of the fistula, and this may be verified by probing and proctoscopic examination.

A small fistula may heal spontaneously, but most will require surgical repair after proper sterilization of the bowel. Sometimes in an older patient with pelvic malignancy or radiation burn, a colostomy will give adequate relief.

Displacements of the Uterus

When one does a bimanual pelvic examination the position of the uterus is usually described as being anteflexed and anteverted. *Anteflexion* refers to the manner in which the body of the uterus is flexed anteriorly on the cervix, *anteversion* means that the uterus as a whole has a tilt which carries the body of the uterus anterior to the axis of the pelvis. This position of the uterus means that when a woman stands erect the uterus lies almost in a horizontal plane and at an angle of almost 90 degrees to the axis of the vagina. In about 20% of women, however, the uterus is in a position of retroflexion and retroversion without any reason and with no apparent ill effect. *Retroflexion* means that the body of the uterus is flexed posteriorly on the cervix; *retroversion* means that the uterus as a whole is tilted so that the body of the uterus lies posterior to the axis of the pelvis to a greater or lesser degree. Sometimes one finds on examination that the uterus is anteflexed, but it seems to fit into the curve of the sacrum; in this position the uterus is said to be *retroposed*. A rectovaginal examination is useful if the position of the uterus is not readily apparent.

A uterus that is retroflexed and retroverted but freely mobile rarely causes symptoms. Most gynecologists have had the experience of replacing and supporting a retroflexed and retroverted uterus with a pessary in an infertile female and then conception has occurred. Infertility may be an indication for surgical replacement, but the patients for this operation must be very carefully selected. In others who suffer from backache and dyspareunia with retroversion, a trial period with a pessary which gives complete relief would warrant a surgical suspension.

A uterus that is fixed in the position of retroflexion and retroversion usually has a pathologic cause. There may be chronic pelvic inflammatory disease with adhesions, endometriosis of the pelvis, enlargement of the uterus due to subinvolution, or fibromyomas. The tubes and ovaries as well are often fixed in the pouch of Douglas. The symptoms of backache, lower abdominal discomfort, leukorrhea and menorrhagia with dyspareunia and infertility may be present. The treatment in these patients must be that of the primary disease.

Does retroversion of a pregnant uterus predispose to abortion? Not ordinarily, but it is wise to caution the patient to refrain from intercourse until spontaneous correction occurs, and the knee-chest exercises may be prescribed even though their value is questioned by some.

Prolapse of the Vagina and Uterus

Prolapse of the uterus and a relaxation of the vaginal walls refer to the descent of the uterus and the protrusion of the vaginal walls which result from relaxation or tearing of the structures that support them.

Supports.—The pelvic floor is formed largely by the 2 levatores ani muscles and their fascial coverings which form a sling supporting the pelvic organs. The urethra, vagina, and rectum pass through the segment that is formed by the fusion of the 2 pubococcygeus muscles. These 2 muscles are attached to the posterior surfaces of the pubic rami and pass downward and backward to be inserted in the raphe between the rectum and the coccyx as well as to its tip. The fibers in relation to the rectum serve to support it. More anteriorly, the most medial fibers of each muscle give support by fusing with the urethral and vaginal walls, and they decussate between the vagina and rectum. These decussating fibers are the ones subjected to the greatest stretching and tearing during childbirth or by any condition that greatly increases the intra-abdominal pressure.

The levatores ani fuse with the vagina at the level of its middle and lower thirds and with the rectum at the level where it angles sharply backward and becomes the anal canal.

The urethra and vagina receive supplementary support below the pelvic floor from the urogenital diaphragm with its fascia and muscles, the insertions of which fuse with fibers from the levatores ani and the sphincter ani to form the *perineal body*.

The vault of the vagina and the cervix are well above the pelvic floor but still receive considerable support from it. However, their main support is supplied by the strong ligamentous-like tissues which lie superior to the fascia covering the upper surface of the levator ani muscles. These ligaments are called the *pubovesical ligaments* which extend anteriorly, the *uterosacral ligaments* which extend posteriorly and outline the pouch of Douglas and the *cardinal ligaments* which are the broadest and strongest and extend fanwise on either side to the lateral walls of the pelvis. The real strength of the supporting structures described above probably lies in the manner in which the fascial planes are so closely attached and fused together.

The round and broad ligaments play but a small role as supporting structures of the uterus.

A cystocele is a relaxation of the anterior vaginal wall, so that the bladder prolapses and the anterior vaginal wall protrudes. If the urethral support is stretched as well, a urethrocele is present.

A rectocele is a relaxation of the posterior vaginal wall which allows the rectum and posterior vaginal wall to protrude. When an enterocele is present, the relaxation of the tissues of the posterior fornix of the vagina is so marked that the upper part of the posterior vaginal wall with the peritoneum of the pouch of Douglas prolapses to the vulva. Coils of small intestine are often present in this hernial sac.

Prolapse of the uterus is of three degrees: first degree, the cervix descends in the vagina; second degree, the cervix presents at the vulva with straining; third degree, the cervix protrudes outside the vagina. *Complete procidentia* signifies that the uterus and vaginal walls are protruding below the vulva.

Elongation of the cervix may occur with no relaxation of the vaginal walls or vault prolapse.

These various conditions usually are present in combinations, and an over-all title, *sacro-pubic hernia*, is sometimes applied.

It is apparent from the above definitions that the patient will most commonly complain of a bulge in the region of the vulva and that this is aggravated by long standing, heavy lifting, or coughing. Lower abdominal discomfort and backache are often present, but bladder symptoms such as frequency and stress incontinence may be the most troublesome. With complete procidentia the bulk of the structures and the bleeding caused by friction on the mucous membranes distress and alarm the patient. It must be remembered that considerable relaxation of the vaginal walls may be present yet the patient is quite free of symptoms. However, as years pass, protrusion becomes more marked and symptoms develop which require treatment. Thus prolapse of a degree which causes symptoms is seen more commonly in the menopausal age group. At that time the relaxation and descent due to lacerations occurring during childbirth are accentuated by the atrophy and weakening of the pelvic supports, and symptoms become more apparent. Other predisposing factors are chronic cough, constipation, or chronic debility. Spina bifida and large ovarian cysts are rare causes.

Diagnosis.—The diagnosis of relaxations and prolapse is ordinarily not difficult and becomes apparent when the patient is asked to strain with the examining fingers depressing the perineum. A complete pelvic examination and smears of the cervix for cytology should be made. Urinary infection is often present, and in complete prolapse, ureteric obstruction may have caused upper tract renal infection and nitrogen retention. Before and after meal blood sugar tests should be done as a routine, since senile diabetes is more common in the patient with sacropubic hernia than in the population at large.

Treatment.—Treatment of prolapse may be conservative or surgical. During the child-bearing age, the pelvic support is strengthened by improving the health of the patient and instructing her in abdominal and pelvic floor muscle exercises. Insertion of a pessary to improve the position and support of the sub

involved uterus may be advisable. If repair is necessary in this age group, some authorities advise tubal ligation as well. If this is not done, subsequent deliveries may either progress normally, using a wide episiotomy, or cesarean section may be indicated. In the older patient a pessary will give temporary relief, but surgical treatment is ordinarily preferable.

The type of operation depends upon the degree of relaxation and prolapse, the presence of chronic cervicitis and hypertrophy of the cervix, associated uterine enlargement, or abnormal bleeding. Repair of the cystocele and rectocele may be all that is required, but if there is any degree of vault prolapse and uterine descent, one should perform the Manchester type operation which consists of amputation of the cervix, shortening of the cardinal ligaments, and repair of the anterior and posterior walls. If abnormal bleeding is a complaint then a vaginal hysterectomy with anterior and posterior repair is necessary. The LeFort operation (partial obliteration of the vagina by apposing the vaginal walls) is being used more rarely as geriatric surgery and anesthetic procedures improve.

PAIN DUE TO PELVIC DISEASE

Acute pain of pelvic origin may at first be described by the patient as being worse in one or the other lower quadrant, but commonly it becomes diffuse over the whole lower abdomen. Chronic pain of pelvic origin, too, is usually diffuse, but the patient is often very indefinite about its severity and location, and she is disturbed mostly by the persistence of the discomfort.

The pain tolerance of the individual is sometimes difficult to assess, and this difficulty is increased because pelvic pain may be present to a disabling degree with very little evidence of pelvic disease. Conversely, a rather large pelvic mass may be present without causing symptoms. Interest has recently been revived in the clinical syndrome caused by chronic pelvic congestion. In this, lower abdominal pain, backache, and fatigue are the most characteristic symptoms, and the degree of tenderness is quite out of keeping with the pelvic findings. To assess the emotional factor in this type of pelvic pain is indeed difficult, but it

is very vital that this be considered if needless surgery is to be prevented.

The relation of the pain to the menses is important. If there is a history of delayed, abnormal, or missed periods, the pain is commonly caused by a complication of pregnancy, e.g., ectopic pregnancy. Midcycle discomfort may be due to ovulation, and pelvic inflammatory disease usually flares up in the post-menstrual phase.

Intense, acute, lower abdominal pain is associated with acute salpingo-oophoritis, ectopic pregnancy with rupture, torsion of the pedicle of an ovarian cyst, or degeneration of a fibromyoma. Except in the presence of continued internal hemorrhage, there is no marked immediate deterioration in the patient's condition and it may, in fact, gradually improve. This is unlike the course of an acute appendicitis, a ruptured viscus, or a spreading peritonitis.

Colicky pain suggests dysmenorrhea, a threatened or inevitable abortion, tubal abortion, or the presence of a large uterine polyp. Vaginal bleeding is almost always present with any of these conditions and serves to exclude lesions of the gastrointestinal or genitourinary tract, such as constipation, intestinal obstruction, or ureteric calculi.

Dull lower abdominal pain may be caused by the pressure of pelvic tumors, and in this instance the discomfort is eased by change in posture. If the tumor is malignant, metastases and local infiltration will cause persistent pain.

Pain in the back in women is commonly caused by conditions other than pelvic disease, e.g., pyelitis. Backache, if due to gynecologic conditions, is usually in the lumbar or sacral areas. Sacropubic hernia or prolapse of the uterus causes this type of backache, with an associated vague discomfort in both groins. With other pelvic disease, backache is a late symptom and the condition is obvious on bimanual examination. Chronic cervicitis with a minimal parametritis may cause this persistent low back pain. Uncomplicated retroversion of a normal, mobile uterus seldom produces symptoms.

Pelvic pain of an acute character requires immediate diagnosis, and the treatment must be instituted, be it medical or surgical. Chronic

pelvic pain without very apparent pelvic disease must be observed over a period of time until the correct diagnosis is made. Often there will be an emotional state present or a disease of another system (gastrointestinal, genitourinary, or skeletal), and the need for gynecologic surgery is thus contraindicated. The forceful opinion of a consultant is an invaluable aid in treating these problem patients.

ACUTE PELVIC INFLAMMATORY DISEASE

Acute Salpingo-oophoritis

Pelvic inflammatory disease is predominantly due to an ascending infection, and the cervical canal is the barrier serving to protect the upper genital tract. A menstrual period opens this pathway to upper tract infection, e.g., gonorrhea, but an abortion, delivery, and instrumentation such as a dilatation and curettage are important predisposing factors. Pelvic tuberculosis is not an ascending infection (the exception) since the pelvic organs are infected by a blood-borne organism from a primary focus elsewhere in the body. Once the uterine cavity is invaded, a transient endometritis results and rarely a pyometra develops. The spread from the uterine cavity may be by continuity along the fallopian tubes, e.g., gonorrhea, or the infection may be carried into the pelvic tissues via the lymphatics or blood vessels, e.g., a placental site infection after an abortion. The causative organisms are the aerobic and anaerobic streptococci, the staphylococci, and the *Escherichia coli* groups *Clostridium perfringens* (Welch's bacillus) and pneumococcal infections are very rare.

With acute pelvic inflammatory disease, general malaise, pain in the lower abdomen of fairly intense degree, a temperature of 101° F or more, and a rapid pulse develop. Nausea and vomiting are not ordinarily present. On abdominal palpation the peritoneal reaction is apparent over the lower abdomen and at first may be more marked on one or the other side before becoming bilateral. On vaginal examination there may be evidence of a lower tract infection, e.g., urethritis or cervicitis, with a copious discharge, and specimen material is taken of this for bacteriologic studies and sensitivity tests. If there has been a recent

abortion or delivery, the discharge may be foul-smelling and sanious in character. On bimanual examination, movement of the cervix causes pain, and there is marked tenderness in both fornices. The uterus is partially fixed, but due to the abdominal splinting it is not often possible to delineate the size of the uterus or accurately outline the appendages. There is almost always bilateral involvement though one side may be more enlarged than the other. A rectovaginal examination is useful to verify the pelvic tissue thickening. The leukocyte count and the sedimentation rate are much increased.

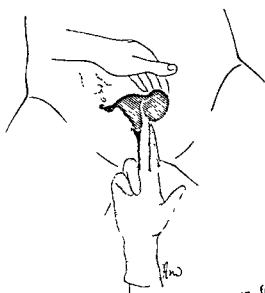


Fig. 351—Bimanual pelvic examination for pyosalpinx

An acute pelvic inflammatory disease may be present with uterine fibroids or be secondary to an infected ovarian cyst or pelvic appendicitis. In recent years the most fulminating cases have developed as a result of septic abortions.

It is most important to exclude an acute appendicitis. The onset of acute appendicitis is more typical and the symptoms of epigastric pain localizing in the right lower quadrant and an associated nausea and vomiting are characteristic. The temperature elevation and pulse rate increase are less marked. On abdominal palpation, the right lower quadrant splinting and a well-localized point of maximum tenderness about McBurney's point are present. On vaginal examination the lower tract is healthy.

and on bimanual examination the pelvic organs are normal and mobile. If there is tenderness present, it is in the right fornix and seems high up, out of the pelvis, unless the appendix is located in the pelvis or there is a marked peritoneal involvement. The leukocyte count and sedimentation rate are both increased and may not be of great help in the differential diagnosis.

An *ectopic pregnancy* with hemorrhage, complications arising from an *ovarian cyst* or a *fibromyoma*, *acute pyelitis*, or *renal calculus* must be carefully eliminated from the final diagnosis.

Prognosis.—The prognosis of uncomplicated pelvic inflammatory disease is good, and improvement is apparent within 4-5 days after treatment has begun. Chronic changes may persist even though the pelvic examination seems essentially normal, and these will be discussed later. If improvement is not noted, then a mixed bacterial infection is usually present and progression of the disease to pyosalpinx, pelvic abscess, generalized peritonitis, or septicemia must be suspected.

Treatment.—Treatment of acute pelvic inflammatory disease is nonsurgical and consists of bed rest, maintenance of fluid balance, with light diet if tolerated, and codeine for pain. Heat therapy may be applied to the lower abdomen, though some patients prefer an icebag. After the initial pelvic examination an enema may be given if required. Too frequent examinations, vaginal douches, or other pelvic manipulations are best avoided in the acute phase. Antibiotic therapy is started immediately and adequate doses of penicillin or the broad-spectrum antibiotics may be used. Laparotomy is indicated if the presence of an acute appendicitis or an infected ovarian cyst cannot be excluded.

Pelvic Peritonitis and Cellulitis

These cannot be differentiated since both are present in varying degrees. In the latter the inflammatory reaction is most evident in the broad ligaments and cellular tissues of the pelvis. The conditions develop more commonly after the evacuation of an infected abortion or after minor gynecologic operations performed in the presence of a lower tract infection which

seemed benign. Rarely, an exacerbation occurs as an isolated incident years after a previous attack.

Symptoms and Signs.—The early symptoms and signs are those described above, but improvement does not occur. The general malaise, a sustained fever, and a rapid pulse persist, and the abdominal pain becomes more marked. On examination one is able to palpate a diffuse tender mass arising asymmetrically from the pelvis, and this is formed by adhesions between the omentum, bowel, and pelvic organs. On bimanual examination there is marked tenderness, the cervix is fixed, and the pelvis is filled from side to side with a boardlike induration. This is the plaster-of-Paris pelvis of earlier writers. The induration may spread around the rectum in such a way as to suggest a malignant rectal lesion. The acute illness is prolonged, and abscess formation may result.

Pelvic Abscess

This is a late and uncommon complication of acute pelvic inflammatory disease, it may also arise as a complication of a ruptured appendix or an acute diverticulitis. There is by now a mixed infection present.

The patient appears more toxic and ill, the temperature remains high, and the leukocyte count and sedimentation rate increase. The pelvic pain is more marked and constant, and bowel pressure symptoms such as diarrhea may develop. On abdominal palpation there is usually some distention due to paralytic ileus, and the abdominal mass arising from the pelvis is more tender to palpation. Vaginal examination verifies the fact that the induration previously noted is more marked and the posterior fornix is bulging down below the cervix which is pushed well forward or to one side by the pelvic mass. Digital pressure on the mass causes marked pain, and sometimes fluctuation is noted. If the abscess is confined to a tubo-ovarian mass it will be less easily delineated because it will be higher in the pelvis and more to one side.

Treatment.—The treatment of these seriously ill patients is medical. The fluid and electrolyte balance must be carefully maintained, especially if gastric suction is necessary. Adequate doses of sedatives, blood trans-

fusions, and large doses of the suitable antibiotic drugs are given. A combination of sulfa drugs and antibiotics may be more efficacious. When abscess formation is apparent, a posterior colpotomy is performed, the sacculations are broken down gently with the examining finger, and a large drain is left in situ. If there is evidence that the abscess is pointing toward the peritoneal cavity, it must be drained through the abdominal wall. Sometimes an extraperitoneal approach is preferable, but in other cases the peritoneal cavity must be opened. The abscess is usually well walled off, and the pelvic structures are so disorganized that nothing more than drainage of the abscess is possible. *Paralytic ileus*, *generalized peritonitis*, and *fistula formation* may result if too much is attempted, while if drainage is inadequate the abscess will reform and the illness will be prolonged. Once the acute stage is past, further surgery to remove residual masses is better deferred for several months when the pelvic findings are reviewed.

Diverticulitis with rupture simulates pelvic inflammatory disease very closely. It occurs in an older age group, and the diagnosis may not be possible until investigations are completed after the subsidence of the acute symptoms.

CHRONIC PELVIC INFLAMMATORY DISEASE

This is the end result of the acute process and therefore the history is most important in making the diagnosis. It is said that gonorrheal salpingo-oophoritis is a self-limiting disease and leaves little residual damage, this may be true of a single episode of infection which is recognized and treated early. However, superimposed mixed infections or primary infection with other organisms is not uncommon and results in chronic tubo-ovarian disease. The fallopian tubes are chiefly involved. This may vary from an endosalpingitis with obliteration of tubal patency and a perisalpingitis with a few filmy peritoneal adhesions to the formation of a pyosalpinx or hydrosalpinx with marked peritoneal reaction and adhesions. These affect the tubes, ovaries, uterus, omentum, and the small and large bowel. The involvement is always bilateral, though one side may feel to be more grossly affected.

Chronic lower abdominal pain of a diffuse, aching character is the most common symptom, and this is especially apparent in relation to the menses, which may become more profuse and painful. There may be an associated lumbar backache. A vaginal discharge due to the chronic cervicitis is present, and intercourse is painful. Due to the tubal changes, infertility is a common complaint. An acute or subacute exacerbation may occur. There is little or no systemic reaction, though the patient is uncomfortable because of the chronic pain. On examination there is little but diffuse tenderness over the lower abdomen to be found. Pelvic examination shows evidence of a catarrhal cervicitis, and on bimanual examination the uterus is normal in size and partially fixed in retroversion and the body of the uterus is acutely tender to pressure. The appendages are palpably enlarged, tender, and bound down, their outlines often ill-defined, and, on recto-vaginal examination, they may be fixed in the pouch of Douglas. It is amazing how extensive the pelvic involvement is found to be at operation when bimanual examination only suggested moderate inflammatory changes.

Pelvic endometriosis, an organized pelvic hematocoele formed by a tubal abortion, or a diverticulitis of the bowel will simulate the pelvic findings of chronic pelvic inflammatory disease. Fibromyoma of the uterus or ovarian tumors may coexist and cause difficulty in diagnosis.

Treatment.—This is essentially conservative and should continue over a considerable period of time. The general health is improved by adequate diet and rest, and any anemia must be treated. Bowel habits must be regulated, especially around menstruation time. Daily copious warm douches may be comforting, but pelvic diathermy treatments are usually more efficacious. Mild analgesics are prescribed for the pain. Chemotherapy and antibiotic treatment have little effect on these lesions.

Ordinarily the patient can be reassured that the disease is not serious and will gradually improve with treatment. In a small percentage, the pain remains to an incapacitating degree, and the pelvic masses persist. Subacute or acute relapses further demoralize the patient. Surgical treatment is then advisable after consultation. The diseased organs, including the uterus,

should be removed, saving as much healthy ovarian tissue as possible, though this may not be practical. Pelvic peritonitis may develop in the immediate postoperative phase, and it is therefore wise to give adequate antibiotic therapy both before and after operation.

The possible extent of the operation must always be explained to the patient and to her husband, especially if she is in the below-40 age group.

Tuberculosis of the Pelvic Organs

This is usually a blood-borne infection from a primary site elsewhere in the body, and this focus of infection may have been quiescent for a number of years. In fact, it is unusual to have an active primary focus to aid in the diagnosis. Rarely, there may be a generalized tuberculous peritonitis with subsequent spread to the pelvic organs. In either instance the fallopian tubes are first affected, pelvic peritonitis develops, and the ovaries and body of the uterus are involved in the inflammatory reaction. The adhesions may be dense and extensive. The endometrium is almost always affected.

It is clear that a careful history must be taken to elicit the presence of tuberculosis in the immediate family or, more usual, of tuberculous disease in the patient herself.

Pelvic tuberculosis so closely simulates pelvic disease due to the gonococcus or the non-specific infection that follows abortion or delivery, that it is not commonly diagnosed as a separate entity. The patient is therefore treated conservatively with rest and penicillin or sulfonamides which characteristically do not improve the condition. It is only when the chronicity of the disease begins to undermine the general health and to incapacitate so that she is unable to earn her living that further investigation is deemed advisable. Endometrial biopsy or a curettage, if done in the late premenstrual phase, often enables the diagnosis of tuberculous endometritis on pathologic study of the tissue. Smears and culture of the endometrium are less reliable, but a portion of the endometrium should be used for guinea pig inoculation to make the diagnosis positive. If tuberculosis of the endometrium is present and there are also bilateral tubal masses, pelvic

tuberculosis may be diagnosed. If an acute exacerbation occurs it is usually precipitated by a mixed bacterial infection. In the older age group uterine fibroids may obscure the diagnosis.

Treatment.—The treatment in such a case should be conservative, with a sanitarium regime if possible. The use of para-aminosalicylic acid, isoniazid, and streptomycin is of great value, but masses already present do not often resolve completely. If permanent benefit is not obtained with the above regime, then laparotomy is advised.

On opening the peritoneal cavity the findings may be so like that of any pelvic inflammatory disease that the diagnosis is still not suspected. In another patient there are the typical retort-shaped tubal masses with tubercles studded over the pelvic tissues. In both of these situations bilateral salpingectomy and total hysterectomy should be done, leaving any ovarian tissue that is sufficiently healthy. With a more marked involvement, all structures of the pelvis, including small and large bowel, are densely adherent. In the rare case there is a widespread tuberculous peritonitis with ascites. It would be advisable in the latter two instances to close the abdomen after taking tissue for study, institute sanitarium and specific drug therapy, and then review the question of a second operation months later.

Prognosis.—This is good for most patients. A postoperative course of drug therapy should be given and convalescence in a sanitarium advised, since there is a remote chance of the primary focus becoming active again or a military type of tuberculosis developing.

BLEEDING

The average menarche begins between the ages 12-14 years and the outside limits of the normal are 10-16 years. Wider variations may be indicative of tumors of the ovary, adrenal, or pituitary, or of certain endocrinopathies. While the menstrual interval is usually 28-30 days, there are wide individual variations. The normal duration of flow is about 5 days, and the menstrual discharge is usually dark and free of clots. With more profuse flow the blood is brighter and may be clotted.

Associated symptoms are dysmenorrhea, heaviness in the pelvis, backache, headache, nausea and vomiting, change in the bowel habits, and emotional upset.

Changes in the patient's menstrual history are of utmost significance in diagnosing gynecologic disease. Excessive bleeding and prolongation of the period without alteration of the menstrual cycle is designated *menorrhagia*, too frequent cycles constitute *polymenorrhea*, and vaginal bleeding between periods is known as *metrorrhagia*.

Abnormal bleeding usually flows from the uterine cavity, but it also may be associated with lesions of other parts of the genital tract or even with general systemic disease and endocrine disturbances.

The local causes may be tabulated:

- 1 Vulvar bleeding
 - a Lacerations
 - b Rupture of varicose veins
 - c Vulvitis or ulceration
 - d Urethral caruncle or carcinoma
 - e Carcinoma
- 2 Vaginal bleeding
 - a Lacerations
 - b Ulceration
 - c Vaginitis
 - d Carcinoma
- 3 Cervix
 - a Erosion and ectropion
 - b Cervicitis
 - c Polyps
 - d Carcinoma and sarcoma
- 4 Uterine bleeding
 - a Trauma, perforation or rupture
 - b Infections
 - (1) Endometritis, acute or chronic
 - (2) Metritis and parametritis
 - (3) Chronic subinvolution
 - c Neoplasms
 - Benign
 - (1) Endometrial polyp
 - (2) Fibromyoma
 - (3) Adenomyoma
 - Malignant
 - (1) Carcinoma
 - (2) Sarcoma
 - d Conditions associated with pregnancy
 - (1) Abortion
 - (2) Extrauterine pregnancy
 - (3) Ante partum and post partum hemorrhage
 - (4) Hydatidiform mole
 - (5) Chorionepithelioma
 - e Functional uterine bleeding
- 5 Fallopian tubes
 - a Pregnancy
 - b Salpingo-oophoritis
 - c Carcinoma

Abnormal bleeding tends to have varied significance, depending upon the patient's age. In the first few days of life vaginal bleeding, often associated with engorged breasts, is due to excessive maternal estrogenic hormone and is without importance.

In childhood, bleeding may be caused by a precocious menarche, vaginal tumor, or foreign body, and rarely by a feminizing tumor of the ovary or malignant disease of the cervix.

In adolescence, menorrhagia can occur at the onset without organic lesion. This is functional in origin and is often associated with anovulatory cycles. It may alternate with periods of amenorrhea. Ovarian growths and malignancy of the uterus at this age are fortunately rare.

During the reproductive period (15th to the 45th year) abnormal bleeding is most commonly caused by disturbances of pregnancy, pelvic inflammatory disease, polyps, fibroids, and malignant growths.

At the menopause, changes in the cycle are common, but menometrorrhagia or postcoital spotting must be studied with the greatest care to rule out benign or malignant disease of the genital tract. Postmenopausal bleeding is most significant and is usually due to *uterine malignancy*, foreign bodies, senile vaginitis, or cervical polyps and rarely to a feminizing tumor of the ovary. It may follow estrogen therapy, and the patient must always be asked if such a hormone has been taken.

Abortions

Abortion is defined as the termination of pregnancy prior to fetal viability, which is the 28th week of pregnancy. The incidence of abortion is higher than is generally believed, perhaps 15-20% or more, and the greatest cause by far is the abnormal ovum itself, faultily developed, improperly fertilized, or abnormally implanted.

Abortions are classified as threatened or inevitable and, if the latter, as complete or incomplete, noninfected or infected.

Threatened Abortion.—Abortion is most often threatened in the first 3 months of pregnancy, before the placenta is fully formed. It is characterized by vaginal bleeding or spotting and lower abdominal pain similar to

menstrual cramps, although the cervix remains closed. Differential diagnosis includes ectopic gestation, cervical erosion, polyps, and cervical carcinoma. Treatment should be rest in bed, sedatives, and analgesics. Vitamin E, estrogens, and progesterone are popular medications but are of doubtful value. A urine pregnancy test may be useful, as pelvic examination should be avoided at the onset of symptoms for fear of further disturbing the pregnancy. If the symptoms persist or progress, this examination must be done.

Inevitable Abortion.—If the pain and bleeding increase and examination shows that the cervical canal is dilating, the abortion has become inevitable. The internal cervical os as a sphincter will never close until the contents of the uterine cavity have been expelled.

Complete Abortion.—If the products of conception are expelled, cessation of pain and subsidence of bleeding follow. If one is absolutely sure the abortion is complete, no operation is required. The patient is kept in bed a few days and given oxytocics to aid in uterine involution.

Incomplete Abortion, Noninfected.—In this case the products of conception have not been completely passed. Dilatation of the cervix, gentle curettage, and evacuation with ovum forceps are necessary to remove the remaining tissue. Excessive loss of blood must be replaced by transfusions at the time of curettage. It may be necessary to control any uterine hemorrhage after curettage by packing and oxytocics.

Incomplete Abortion, Infected.—Septic abortion is caused by the infection of retained products of conception. This is commonly introduced by foreign bodies or solutions used in an attempt to terminate pregnancy. Uterine and tubo-ovarian infection, pelvic peritonitis, thrombophlebitis, pyemia, or septicemia may result. It is best to treat first the infection with antibiotics, transfusions, and bed rest. When the temperature has been normal for 48-72 hours, the uterine contents are gently evacuated. The only exception is when the infected incomplete abortion is associated with uncontrollable hemorrhage; immediate evacuation of the uterus is then imperative.

Missed Abortion.—The uterus does not expel the perished ovum. First there may be

vague contractions and little bleeding, but these subside. The diagnosis is made by later noting involution of the breasts, failure of the uterus to enlarge, and the negative pregnancy tests. If, in time, the uterus does not expel its contents it may be stimulated to do so by large doses of estrogenic hormone, followed by Pitocin. If ineffective, dilatation of the cervix and evacuation of the uterine cavity are indicated.

Hydatidiform Mole.—This condition occurs about once in 2,500 pregnancies. Whether it is degenerative or neoplastic in origin is unknown. There is proliferation of the trophoblastic covering of the villi, edema of the stroma, scantiness of the villous blood vessels, and ordinarily no trace of the fetus. Because of the vesicular change of the villi, the mole has the appearance of a bunch of grapes.

The chief symptom is continued bleeding in the 3rd or 4th month of pregnancy, sometimes with passage of characteristic villous tissue. The uterus is often larger than normal for the period of amenorrhea and there is an absence of fetal movements, although the subjective signs of pregnancy are present and the urine pregnancy tests are strongly positive.

Hormonal studies are important. There is an overproduction of chorionic gonadotropic hormone which is found in great quantities in the urine, blood, and spinal fluid. If the urine shows more than 200,000 mouse units of the hormone, the diagnosis usually is justified. Treatment is evacuation of the uterus and blood transfusions as required. A careful follow-up is necessary because of the danger of subsequent chorionepithelioma. The incidence of malignant change is 1-2%. Urinary studies must be continued for 12-24 months, even if the tests become negative. Persistent high chorionic gonadotropic hormone indicates retention of actively functioning trophoblastic tissue, and a second curettage is indicated. If the test continues positive (and there is not a superimposed new pregnancy), especially with bleeding and subinvolution of the uterus, hysterectomy must be performed. An x-ray of the lungs should be taken to exclude metastatic chorionepithelioma.

Chorionepithelioma.—This highly malignant disease, although rare, may develop after

hydatidiform mole, full-term delivery, or abortion. Vaginal bleeding is the chief symptom, but the disease may first be detected by metastases in the lower genital tract or in a distant organ such as the lung. Cough and hemoptysis may develop, and x-ray of the chest will show the typical metastatic invasion of the lung. Hormonal studies are invaluable, but the final diagnosis must be made by study of the tissue secured by curettage. There is destruction of the myometrium by invading trophoblastic tissue, which is disorderly in pattern and malignant in cellular appearance. Treatment is total hysterectomy and bilateral salpingo-oophorectomy followed by irradiation therapy since chorionepithelioma is radiosensitive. Metastatic growths may disappear either because of the latter treatment or because the primary uterine tumor has been removed, but the prognosis is poor.

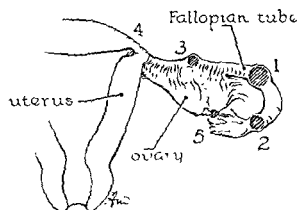


Fig 332—Sites and relative frequency of ectopic pregnancy

Ectopic Pregnancy

This occurs when the fertilized ovum implants itself outside the cavity of the uterus. The most common site for this to occur is in one of the fallopian tubes, a *tubal pregnancy*. Implantation and development of the fertilized ovum progress. The endometrium of the uterus responds to the hormonal stimulation of the tubal pregnancy and amenorrhea results. Other symptoms and signs of pregnancy may or may not be present. Before long the erosion and distention of the tubal wall result in rupture of the tube or the expulsion of the pregnancy through the fimbriated os of the tube. Either

of these events causes pain and intraperitoneal hemorrhage. When the hormonal balance is upset by interference with the implantation site, intrauterine bleeding occurs. This is apparent to the patient as vaginal spotting, which later may become moderate bleeding.

Tubal pregnancy with rupture and diffuse intraperitoneal bleeding creates the classic syndrome of amenorrhea of 5-6 weeks' duration, sudden, severe, stablike lower abdominal pain, and collapse, with all the signs of internal hemorrhage. On examination the patient's pallor is marked and she is in a state of collapse. The lower abdomen is tender, usually soft, perhaps distended; on pelvic examination there is almost always slight vaginal bleeding and exquisite pain on manipulation of the vault of the vagina or cervix. The cervix is soft and closed, and the uterus, if palpable, is enlarged slightly.

With tubal abortion and slower hemorrhage, the clinical picture is less spectacular. The diagnosis is not commonly made on the first examination. There is an indefinite history of a missed period, then crampy intermittent lower abdominal pain develops, and unilateral abdominal tenderness is present. Vaginal bleeding occurs and often persists, though the symptoms and signs become less marked. Often the patient is treated for pelvic inflammatory disease or an incomplete abortion until, later, further internal hemorrhage occurs despite bed rest. Then there is a recurrence of the colicky pain, an increasing anemia develops, and reflex shoulder pain may be present due to diaphragmatic irritation. On pelvic examination an indefinite tender mass, a *pelvic hematocoele*, is felt in the pouch of Douglas or in relation to the appendages.

Differential Diagnosis.—Aids to the differential diagnosis of ectopic pregnancy are a pregnancy test, an examination under an anesthetic, curettage, and posterior colpotomy. The pregnancy test, if negative, does not exclude an ectopic pregnancy.

This is differentiated from *acute appendicitis* by the sudden onset, the presence of uterine bleeding, and the evidence of internal hemorrhage with the associated vaginal findings.

Acute pelvic inflammatory disease usually produces quite a different course of events. However, chronic salpingo-oophoritis is fre-

quently the initial diagnosis when a tubal abortion is actually present and either may be superimposed on the other. The history of a missed period, the crampy pain, the vaginal bleeding, and a tender pelvic mass which increases in size while the patient is under observation are most characteristic of a tubal pregnancy. If a tubal pregnancy is suspected, the pelvic examination should be gently done, because brisk intraperitoneal hemorrhage may be started. This is particularly important if the patient is at home where shock treatment is not available.

An incomplete abortion creates more profuse bleeding and less pain. On examination the uterus is usually larger and there is more marked cervical dilatation. The products of conception are expelled, sooner or later, through the cervix.

An ovarian tumor with torsion of the pedicle causes sudden, severe abdominal pain and local peritonitis which may simulate an acute appendicitis or intraperitoneal hemorrhage. Ordinarily the cyst is of moderate size but enlarges rapidly after torsion and intracystic hemorrhage. The patient may be in a state of collapse with a subnormal temperature. If the tenderness of the abdomen excludes proper abdominal palpation, it is sometimes possible to feel the rounded, tender mass in the pelvis. Examination under anesthesia will verify the diagnosis. Rupture or infection of an ovarian cyst is less common and produces peritonitis. Diagnosis may be difficult. The treatment is prompt laparotomy and removal of the tumor.

Rupture of a graafian follicle at ovulation time at midcycle may be painful (mittelschmerz) if minor intraperitoneal hemorrhage occurs; this bleeding is usually minimal, the acute episode passes, and while regional tenderness may persist, the progressive signs which suggest an ectopic pregnancy with tubal rupture or acute appendicitis do not develop.

Treatment.—In any ectopic pregnancy the treatment is surgical after preparations are made for the treatment of the blood loss and shock. The affected tube is removed, leaving the ovary if this is practical.

Uterine Polyps

Polyps arise from the cervix or body of the uterus. *Cervical polyps* are mucous in type and

are small. They arise in the cervical canal. Symptoms are mucous discharge, irregular spotting, or postcoital bleeding. *Intrauterine polyps* arise from the endometrium or from a submucous fibroid; rarely are they placental in origin. They cause menorrhagia and intermenstrual spotting.

Speculum examination will show a cherry-like cervical polyp or a uterine polyp protruding through the cervical os.

Although small cervical polyps may be destroyed by the cautery, it is usually wiser to hospitalize the patient. Under anesthesia the cervix is dilated, the polyp removed by torsion from its origin, and the uterine cavity carefully curetted for associated disease or other polyps. The tissue can then be histologically examined for possible malignant changes.

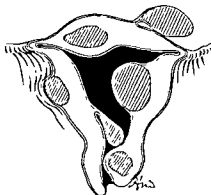


Fig. 353.—Positions of uterine myomas

Fibromyomas (Fibroids)

These are benign new growths of the uterus and are usually multiple. They may be of various sizes and may arise in varying parts of the uterus, thus causing asymmetric enlargement. The rate of growth in the absence of complications is slow, and a very large proportion are asymptomatic. The tumors are most commonly diagnosed in patients 35-45 years of age, during which period symptoms tend to develop. Fibroids, as they enlarge, are prone to secondary degeneration due to impaired blood supply. Hyaline, cystic, and fatty changes may develop. During pregnancy, red degeneration may occur and cause much pain. After the menopause, simple atrophy is usual, but sometimes a calcified

fibroid may be seen. Sarcomatous degeneration occurs rarely, 1% or less.

Symptoms.—These are profuse prolonged menstrual bleeding with secondary anemia, discomfort due to pressure, and a relative infertility. As the size increases, abdominal swelling may be noted, and a feeling of heaviness in the lower abdomen may be described. Marked epigastric discomfort is a rather unusual and misleading symptom. Acute pain arises if the fibroid undergoes degeneration or torsion. These complications cause a rapid increase in the size of the uterus, but an associated pregnancy must be excluded.

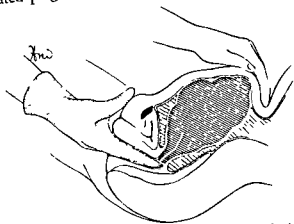


Fig. 354.—Bimanual examination for fibroid uterus

On lower abdominal palpation a firm asymmetric mass, which is usually not particularly mobile, may be demonstrable. On bimanual examination the hard mass is felt arising from the pelvis, it is continuous with the cervix and is defined as the asymmetrically enlarged body of the uterus. The mobility is more apparent on bimanual examination unless there is fixation due to adhesions from an associated pelvic inflammatory disease or endometriosis or there is impaction of the fibroid in the pelvis. The fornices are clear unless the fibroid extends into the broad ligament. No fetal parts or movements are apparent. The mass is dull to percussion, and on auscultation a soufflé may be present. The pregnancy test is negative, and the fibroids grow more slowly than a pregnant uterus.

In certain cases a uterogram to visualize the interior of the uterus may be an aid in diagnosis. The cavity is usually asymmetric if fibromyomas are present.

Treatment.—Completely asymptomatic fibroids may be left alone, but the patient should have a routine bimanual examination every 6 months. A small fibroid with irregular bleeding warrants a diagnostic dilatation and curettage. The treatment of fibroids, which are causing secondary anemia due to hemorrhage or discomfort due to the size and pressure, is surgical. Myomectomy may be successful in a limited number of younger patients who wish to have children. Hysterectomy is usually the operation of choice, and the ovaries should be conserved if there is no ovarian disease.

Carcinoma of the endometrium may be associated with fibroids. For this reason all uteri removed must be opened at once and carefully examined by an assistant. If gross inspection is suggestive of carcinoma of the endometrium or sarcoma of the fibroid, then the ovaries should be removed.

Functional Uterine Bleeding

In functional uterine bleeding, the normal cycle may be maintained with the bleeding prolonged or profuse, the cycle quickened or even doubled, and intermenstrual bleeding may occur. This may alternate with periods of amenorrhea. The mechanism is not clearly understood and is probably due to a variety of causes, such as imbalance between the two ovarian hormones, estrogen and progesterone, failure of the spiral arterioles to contract, or abnormal uterine muscle response. An important feature is that the abnormal bleeding must have persisted over several cycles.

In some cases there is failure of ovulation and persistence of unruptured graafian follicles with resultant hyperplasia of the endometrium (metropathia hemorrhagica). In other cases the endometrium is found to be atrophic, and in others progesterational, indicating ovulation. Behind this is an imperfect knowledge of the effect of the gonadotropic hormones of the pituitary gland upon the ovaries. There may be an emotional or stress factor in this type of bleeding.

Treatment.—This will vary according to the age of the patient, the severity of the bleeding and the need to preserve reproductive function. In the young girl ovulation may not occur and she may have irregular cycles with

increased bleeding. These will be spontaneously regulated in time. Rectal examination will most often reveal normally developing pelvic organs. Care is taken to control anemia. Hormone therapy, other than thyroid, is used in carefully selected cases. Examination under anesthetic and curettage are only occasionally required for persistent heavy bleeding.

During the active *childbearing period* functional bleeding may be a distressing problem. It is at this time that hormone therapy has its greatest value. It is important to emphasize that before it is used, organic lesions must be excluded by physical examination, diagnostic curettage, or endometrial biopsy.

The hormones used in treatment are as follows:

1. Estrogens, synthetic, e.g., diethylstilbestrol, and natural, e.g., Premarin or ethinyl estradiol. These may be given cyclically for several weeks and gradually withdrawn. The purpose is to raise the estrogen level in cases where bleeding is caused by such deficiency.

2. Progesterone, by subcutaneous, sublingual, or vaginal absorption. When bleeding is due to persistent proliferative endometrium, progesterone may bring about the secretory phase, with cessation of bleeding.

3. Combined estrogen and progesterone therapy. This is often effective, perhaps by re-establishing a normal balance between the pituitary gland, the ovary, and the endometrium.

4. Androgens, sublingually or intramuscularly. When administered in less than masculinizing doses they are helpful in controlling functional bleeding.

5. Thyroid extract. This is indicated where hypothyroidism exists.

6. Gonadotropic preparations.

If the bleeding cannot be controlled with hormones or by curettage, more radical treatment is necessary. With a patient in the late 30's hysterectomy may be advisable after a second opinion has been obtained.

Radiotherapy should be avoided if possible as its effect on the ovaries varies.

At the menopause the treatment is more exact. Again organic disease such as carcinoma or polyps must be excluded, and this diagnostic curettage may control the abnormal bleeding. Since a curettage is not always permanently

effective, total hysterectomy rather than repeated curettage is the treatment of choice. In surgically poor-risk patients, intrauterine radium or x-ray irradiation must be considered.

CARCINOMA OF THE UTERUS

Cervix

With the exception of the breast, the cervix is the most common site of cancer in women. It is at least 3 times as frequent as cancer of the uterine body, but together they make up 30% of all cases. Because the cervix is accessible for study and diagnosis, and because the cancer is frequent and grave, the examining doctor is offered an opportunity for early detection and a correspondingly high percentage of cures.

Predisposing factors are trauma and chronic irritation. Much has been written of the higher incidence of cervical cancer in females who have married early and borne many babies, as opposed to the much lower rate in virgins and nulliparae. However, the physician should concentrate on elimination of cervical irritation, infection, erosion, and on the detection of pre-invasive cancer when and if it occurs.

There are two types of cervical cancer; 95% arise from the squamous epithelium and 5% develop from the columnar epithelium of the endocervical glands, i.e., adenocarcinoma. In the early stages the cervix may look normal, especially if the cancer is confined to the cervical canal; there may be a vascular erosion that bleeds on touch or a granular area at the squamocolumnar junction. In the more advanced stages the cancer may form a crater-like ulcer or a cauliflower-like growth.

The microscopic appearance is that of cancer cells showing disparity in size and nuclei, excess mitosis, hyperchromatosis, and karyorrhexis, although in some the departure from normal differentiation is relatively slight. This must be coupled with an abnormal pattern or architecture so that the epithelial cells push through the basement membrane and invade the stroma.

Spread may be (1) direct to the vagina and uterine body, outward to the parametrium where it may involve the ureter, forward to the bladder, and backward along the uterosacral ligaments to the rectum; (2) by lym-



Fig 355—Technique of cervix ring biopsy

A, Cervix with positive cytology adequately exposed

B, Ring biopsy begun with sharp knife with narrow angulated blade.

C, Ring biopsy excised, minimal bleeding

D, Raw surface cauterized with hot wire for hemostasis

(From Latour, J P A : Surg Gynec & Obst 94 : 270-272, 1952)

phatics, primarily to the paracervical node, obturator, hypogastric and external iliac nodes, and secondarily to the sacral, common iliac, inguinal, and aortic nodes, (3) by the blood stream to the liver, kidneys, lung, and bones, a late and rare occurrence.

Classification follows that of the League of Nations Health Organization.

Stage 0—Carcinoma-in-situ, also known as preinvasive carcinoma, or intraepithelial cancer

Stage I—Carcinoma confined to the cervix.

Stage II—Carcinoma with infiltration of the parametrium on one or both sides without involvement of the pelvic wall. Stage II vagina: The vagina is infiltrated in the upper two thirds only. Stage II corpus: The corpus uteri is infiltrated

Stage III—The cancerous infiltration of the parametrium has invaded the pelvic wall on one or both sides, or the lower third of the vagina is invaded, or isolated malignant nodules are felt on the pelvic wall.

Stage IV—The cancer involves the bladder as determined by cystoscopic examination or by the presence of vesicovaginal fistula; or the cancer involves the rectum, or has spread outside the true pelvis.

Carcinoma-in-situ has aroused much discussion. It may be defined as a complete replacement of the normal squamous epithelium by undifferentiated cells morphologically indistinguishable from cancer. However, there is no break in the basement membrane, although the cells may creep into the lumen of the glands. This condition, without recognizable signs or symptoms, may precede, or exist at the periphery of, invasive cancer of the cervix. Positive cervical smears occasionally will not be supported by the subsequent ring biopsy. Cases of carcinoma-in-situ under observation may become negative on cytologic study after biopsy.

Symptoms.—The first symptom is abnormal vaginal bleeding, usually intermenstrual spotting which occurs after intercourse, douching, or straining with defecation. This may be preceded by a yellow or brown watery discharge which becomes blood-stained and foul-smelling. Pain is a late symptom and suggests invasion of the tissues outside the cervix.

Pain may be only a pelvic discomfort or there may be low backache with radiation into the thighs. Bladder irritability and rectal discomfort indicate extension to those organs.

Diagnosis.—The onset of cervical cancer is insidious and the initial symptoms are trivial in character and comparatively late in appearance. Cervical cancer may be diagnosed and treated before any symptoms appear. This can only be achieved by thorough and periodic examination of the cervix and willingness of the patient to cooperate and to report the slightest symptoms. Vaginal spotting, discharge, and pain are not pathognomonic, but they do suggest a lesion, either cancer, erosion, inflammation, or polyp.

Pelvic examination and visualization of the cervix are necessary. If the clinical diagnosis is not apparent, a cervical smear is obtained with a spatula, and an aspiration smear of the uterine cavity is recommended. The smears are examined by a trained cytologist. When anaplastic cells are found, a ring biopsy at the squamocolumnar junction is taken for serial section examination. At the same time the cervix is dilated and fractional curettage of the endometrium and endocervix is performed.

The Schiller test, based on the fact that iodine will stain normal cervical cells a deep mahogany brown while cancer epithelium remains unstained, may be used to distinguish areas for biopsy. Unfortunately leukoplakia, traumatized mucosa, eversion, and erosions take the stain poorly and make the test less useful.

Biopsy with microscopic examination is the important diagnostic method.

Treatment.—

Carcinoma-In-Situ.—Preinvasive carcinoma occurs in younger women in whom sexual function and childbearing must be considered, and it may be permissible to use surgical conization of the cervix rather than hysterectomy, with prolonged postoperative follow-up. Where further reproduction is not desired a total hysterectomy is advisable, removing the cervical fascia and a wide cuff of the vagina. The ovaries may be conserved.

Carcinoma of the Cervix.—Treatment may be irradiation, surgery, or a combination of both. Controversy still exists. Irradiation ther-

apy can be used in any stage of cervical cancer and is particularly applicable to the poor-risk patient. The gamma rays of radium and deep x-rays are applied in a dosage to destroy the tumor cells in the cervix, parametrium, and pelvic lymph nodes with minimal damage to normal tissues. The technique varies, but in this center a modified Stockholm technique, a combination of radium followed by deep x-ray, is used. The radium is applied in the uterine cavity, around the cervix, and in the vagina in such a way as to radiate both locally and to the paracervical tissue and nodes. It is given in two doses of 3,000 mg./hours each, 10 days apart. This is followed by deep x-ray therapy through two anterior and two posterior ports to reach the lateral tissues of the pelvis. The total skin dose varies from 3,000-4,000 r to each field. Thus the total dose of x-ray in the average case is 3,500 r to the tumor area, given over a 7-week period.

Irradiation may cause nausea and vomiting, rectal and bladder irritation, or the flaring up of an old pelvic infection. Later reactions are fibrosis, vaginal atresia, proctitis, bladder ulceration, and vesicovaginal or rectovaginal fistulas.

Surgical treatment has been successfully performed for Stage I and early Stage II cancer in surgically fit women by use of the radical Wertheim hysterectomy and pelvic lymphadenectomy.

Two further indications for surgery are the detection of radioresistant cancer or recurrence after adequate irradiation. The surgery depends on the extent of the lesion and may be a radical hysterectomy with pelvic lymphadenectomy or pelvic exenteration, anterior, posterior, or complete. These have a high mortality, with extensive suffering and prolonged hospitalization and convalescence. When curative treatment has failed, the physician will have much to offer for the relief of pain with drugs or with resection of the spinothalamic tracts.

Corpus Uteri

Carcinoma of the body of the uterus is recognized more frequently today and the ratio to cervical carcinoma is about 1:3. It occurs later in life than cervical carcinoma, as a rule, and its

association with obesity, diabetes, relative infertility, abnormal menstrual cycles, and menopausal hyperplasia has been noted.

Carcinoma arises in the endometrium, from the epithelium, or from the glands. The adenomatous type is the most common and may be localized to a single area, or polypoid processes may project from an extensive area and fill the uterine cavity. The microscopic appearance is almost always glandular. There is a marked increase and disorderly arrangement of the acini, and the individual cells show varying degrees of immaturity and anaplasia.

Adeno-acanthoma is an interesting variant in which patches of squamous epithelium are found intermingled with adenocarcinomatous tissue.

Spread.—The carcinoma remains limited to the uterus much longer when occurring in the body than when arising in the cervix. It is restrained by the uterine musculature and proceeds only in the advanced stage by the lymphatics to the broad ligament, tubes, ovaries, and the lumbar chain of lymph nodes. Secondary growths can occur in the vagina by direct extension. Pyometra is a common complication of the advanced stage.

Symptoms.—Abnormal vaginal bleeding is the most frequent and important symptom. In the menstruating age, menorrhagia and metrorrhagia are common, and in the postmenopausal woman, spotting is suggestive. Abnormal discharge, at first watery, then brownish, bloody, or foul-smelling is significant. Pain is a late symptom.

Diagnosis.—Negative cervical and endometrial aspiration smears do not exclude endometrial cancer, and a diagnostic curettage is mandatory. In more than half the cases the bleeding will be due to benign hyperplasia, polyps, submucous myoma, unsuspected incomplete abortion, or senile endometritis.

The microscopic study of the curetted tissue will give the diagnosis in most cases. In some it is difficult, and pathologists may differ in their interpretation of hyperplasia and endometrial polyps.

Treatment.—Total hysterectomy, including removal of the cervical fascia and a wide cuff of the vagina, together with bilateral salpingo-oophorectomy is the most satisfactory treat-

erated blood (*chocolate cysts*). Endometriosis of the pelvic organs is the most common form; however, the small bowel, large bowel, and bladder may be involved, usually secondarily to the pelvic condition. More rarely, extra-pelvic tissues are involved.

Pelvic endometriosis may occur at any time during menstrual life. Diagnosis is difficult since the symptoms and signs are diverse and develop slowly. With *adenomyosis uteri*, the clinical diagnosis is rarely made. Menstrual pain and menorrhagia gradually develop in multiparae over 35 years. On bimanual examination the uterus is smooth and symmetrical but may be bulky and tender. The appendages seem normal. These findings do not warrant surgery at first, but continued ill health after curettage on one or more occasions indicates more radical treatment and a total hysterectomy is necessary.

With *external endometriosis*, chronic symptoms develop which are aggravated with the onset of each menstruation, and these increase in severity as the invasion spreads. There is present the symptom-complex of acquired and increasing menstrual pain, dyspareunia, and pelvic discomfort due to pressure. Menstrual irregularities may be present, and a suggestive feature is a prolonged period of infertility.

In the early stage, pelvic examination discloses nothing palpably abnormal, though thickening of the uterosacral ligaments may be noted. Later, the findings are almost indistinguishable from those of chronic pelvic inflammatory disease. There is, however, no history of fever or previous infection. As the involvement extends, the uterus becomes fixed in retroversion, and bilateral, tender, cystic ovarian masses are felt. The definite hard, irregular nodules in the rectovaginal septum are usually diagnostic. Rectal symptoms may be produced by this septal involvement.

The wall of the large bowel may be invaded by endometrial implants which cause dimpling due to fibrosis and later, stricture of the bowel lumen. Abdominal pain and constipation recur with the menses, and eventually partial obstruction may result. Neoplasm of the colon may be suspected, but the rectal mucosa is usually intact, and this serves as a diagnostic aid in excluding carcinoma of the

bowel. The bladder wall is not often sufficiently involved to cause characteristic cyclic symptoms and signs.

At operation minute purplish spots distributed over the pelvic peritoneum suggest the diagnosis, and there are marked cystlike formations (*chocolate cysts*) with gross dense adhesions involving the pelvic structures. The lines of cleavage of the adhesions are found with difficulty, and usually the endometrial cysts are ruptured, allowing varying amounts of chocolate-like material to soil the pelvic region. It is apparently innocuous but peritoneal cleansing should be as adequate as possible.

Treatment.—Treatment with hormones and continued observation may be effectual in young adults with menstrual pain and minimal pelvic findings. With greater involvement, surgery is advised, and the present trend in surgical treatment of pelvic endometriosis is to be as conservative as possible. The condition will regress if pregnancy occurs or if the ovaries are removed. In the younger age group (25-35 years), therefore, one removes only grossly disorganized structures, and, if practical, one leaves some ovarian tissue. The patient usually accepts the conservative operation in preference to spaying, even though a second operation or deep x-ray therapy may be necessary later. Suspension of the uterus and presacral neurectomy may be combined with conservative surgery, but each case must be carefully assessed. In the older group (35-45 years) the tissues are more likely to be extensively involved, and total hysterectomy and bilateral salpingo-oophorectomy may be necessary. If scarring of the rectovaginal septum is marked, this may be technically difficult, but as much of the cervix as possible is removed.

PELVIC TUMORS

Excluding the pregnant uterus at or near term, neglected fibromyomas, very large ovarian cysts, tuberculosis of the peritoneum, or malignant ovarian cysts with metastases and ascites, pelvic tumors do not cause generalized enlargement of the abdomen. Obesity, ascites, and phantom tumors should also be remembered. A full bladder or a loaded bowel is

easily excluded by catheterization and emptying the lower bowel with an enema.

At puberty, if a persistent vaginal septum be present, a lower abdominal tumor may be formed, and the menstrual fluid accumulates in the vagina (a hematocolpos) and the uterus (a hematometra).

Abdominal distention of a greater or lesser degree is a common complaint at the menopause. The distention comes and goes, and its very indefiniteness ordinarily indicates its minor importance.

A tumor localized to the lower abdomen may be uterine or ovarian in origin. Pregnancy, fibromyoma of the uterus and ovarian cysts are the most common conditions which may produce an abdominopelvic mass in the absence of marked pelvic infection. It must be remembered that pregnancy and a fibromyoma or pregnancy and an ovarian cyst may coexist.

The Pregnant Uterus

This is the most common pelvic tumor. The history of amenorrhea, associated with breast changes and the other symptoms and signs of pregnancy, should prevent errors of diagnosis. The pregnant uterus is ordinarily uniformly enlarged and is soft on bimanual palpation. A pregnancy test may be necessary. Otherwise one can postpone the diagnosis until another month has elapsed, and then the rapid, symmetric globoid enlargement due to the pregnancy will be apparent. Later, of course, fetal movements, fetal heart sounds, or x-ray demonstration of fetal bones gives the diagnosis absolutely. The physician must remember the value of auscultation of the tumor, an x-ray of the abdomen, and a pregnancy test.

Fibromyoma

(See previous section.)

Ovarian Tumors

These are predominantly cystic in character, but some are solid. They vary tremendously in size and contour, but the size does not always account for the symptoms nor is it a measure of the potential danger to the patient. Certain types tend to involve one ovary only, and if bilateral tumors are present, the possi-

bility of malignant changes are more likely. Some are remarkably mobile because of the long pedicle, while others are impacted and fixed in the pelvis. The former usually rise up into the general cavity, while the latter remain in the pelvis and distort the normal relations of the pelvic structures. The rate of growth is faster than that of a fibroid and is especially rapid if intracystic hemorrhage occurs.

TABLE 24
CLASSIFICATION OF OVARIAN TUMORS

1 Retention cysts of the ovary	
a Follicular cysts	
b Corpus luteum cysts	
2 Cystic and solid tumors	
Benign	Malignant
A Cystic	
Serous papilliferous cystadenoma	Papilliferous adenocarcinoma
Pseudomucinous cystadenoma	Pseudomucinous adenocarcinoma
Endometrioma	
Cystic teratoma (Dermoid cyst)	Epithelial element carcinoma
B Solid	
Fibroma	Teratoma
Granulosa cell tumor*	Sarcoma
	Granulosa cell tumor*
Thecoma*	Thecoma*
Arrhenoblastoma*	Arrhenoblastoma*
	Dysgerminoma
Brenner tumor	Brenner tumor
	Primary carcinoma
	Secondary carcinoma
	Bowel
	Stomach
	Uterus
	Breast

*Tumors that are functioning or that secrete sex hormones

Follicular cysts may be created by hypersecretion rather than by pure retention, but the end result is the same. The ovary involved is not greatly enlarged and usually contains more than one enlarged follicle. The other ovary may be involved as well. The ovaries are usually quite discrete, though there is often an associated chronic pelvic inflammatory disease, retroverted uterus, or uterine fibromyoma—all believed to be causative factors. There are no specific symptoms, though it is feasible that anovulatory cycles may result or a metrorrhagia develop. Simple puncture of the largest follicles is usually sufficient, but the presence of many small cysts may necessitate a wedge resection.

A corpus luteum cyst is a small cystic area in the ovary which may look like a hematoma. The whole ovary is seldom larger than a hen's egg, the surface over the cyst may be cratered but the ovary itself is smooth and discrete. Because of the hormonal imbalance caused by the persistent corpus luteum, a menstrual upset may be present. If a hydatidiform mole is present, both ovaries may be enlarged to a greater extent due to the existence of multilocular theca lutein cysts. *In any event, an ovary which contains only a follicular or corpus luteum cyst should not be sacrificed.*

Serous cystadenoma may be quite a minute cyst but usually is the size of a large grapefruit when the diagnosis is made. The surface is smooth, shiny, and whitish, and the remaining ovarian tissue is thinned out so as to be indistinguishable. A single ovary is generally affected, and the tumor is discrete and freely mobile. It is usually on a pedicle which renders it subject to torsion with resultant intracystic hemorrhage. The cyst, in the absence of hemorrhage, contains clear fluid and its lining may be quite smooth, *the simple type*, or it may be lined with many papillary growths, *the papilliferous type*. The tumor is usually asymptomatic until symptoms from pressure or torsion of the pedicle occur. The papilliferous cysts have a tendency to become malignant, and this is suggested by the presence of bilateral cysts with external papilliferous projections or the presence of ascites with papilliferous metastases throughout the peritoneal cavity. In any event the specimen must be opened and examined carefully in the operating room and the decision regarding malignancy made on the gross appearances.

Pseudomucinous cystadenoma is the most common ovarian tumor. The cyst is multilocular and varies in size from a small lesion to one that fills the entire peritoneal cavity. The locules give the cyst an irregular form and the contained material is sticky or mucinous, with a characteristic yellowish tinge. The other ovary is also involved in a considerable proportion of cases, but the tumor masses remain discrete and the tough outer wall usually persists intact. If rupture should occur, the mucinlike material may fill the entire pelvis or peritoneal cavity, *pseudomyxoma*.

peritonei, but this is rare. There is a high incidence of malignancy which, however, may be restricted to one small loculated area. The borderline between the benign and malignant tumor is very difficult to distinguish even for the pathologist, and at operation the characteristic signs listed above are not often apparent. If adenocarcinoma is diagnosed on pathologic study, the remaining pelvic organs must be removed as soon as practical. Deep x-ray is an alternative therapy in selected cases.

Endometrioma (see endometriosis).

Cystic teratomas (dermoid cysts) are among the commoner ovarian tumors and are generally present in a younger age group than other cysts. The weight and size cause discomfort, the tumor usually being about the size of a large grapefruit. There is a long pedicle and one ovary is involved, though the other may be the site of a much smaller dermoid cyst. On inspection the mass is discrete with a smooth, dense, bluish-white surface free of any lobulation. On opening the cyst the presence of sebaceous material and hair confirms the diagnosis, and there is an ectodermal plate which includes skin, teeth, and cartilage. There is little danger of malignancy. The dermoid cyst can be dissected from the ovarian tissue if the latter is not too attenuated, but if the other ovary appears quite normal, the affected ovary is removed. Because of the young age of the patient the treatment is as conservative as possible.

Solid teratomas originate from tissues derived from all three primitive germ layers and occur in the younger age group. The tumor is unilateral, occurs rarely, and is highly malignant, so that the prognosis is poor.

Fibroma is a solid tumor which may involve only a small part of the ovary, or it may diffusely replace the ovarian tissue. The one ovary is involved, the tumor is smooth and solid, but, as in a fibroid, cystic changes may be present. The size is seldom greater than a large orange. An important clinical feature is that ascites and hydrothorax may be present with this benign tumor.

Sarcoma of the ovary is rare. Both ovaries may be affected, and again the tumor is solid, though cystic changes may be present.

Granulosa cell tumors and thecomas are solid ovarian tumors known as functioning

or feminizing tumors because they secrete estrogen and thus upset the normal hormonal balance. One ovary is usually affected; the smooth tumor remains small and its size rarely equals that of a grapefruit. When sectioned, small cystic areas may be present, and the granulosa tumors may show a yellowish tinge. The tumor can occur at any age and produces precocious puberty in the young child, irregular bleeding during the reproductive years, and uterine bleeding in the postmenopausal period. These symptoms, plus those due to pressure which may develop, bring the patient for advice. The tumors are potentially malignant. In the younger age group, only the affected ovary is removed, but in anyone over 40 years bilateral salpingo-oophorectomy and total hysterectomy should be performed in the presence of such a solid tumor.

Arrhenoblastoma is a rare ovarian tumor which develops during young adult life, and due to its secretion of androgen it is known as a functioning or masculinizing tumor. Secondary amenorrhea and breast atrophy occur, hirsutism develops, and the clitoris becomes hypertrophied. The ovarian tumor is usually unilateral, solid, and smooth. The symptoms and signs of masculinity regress after the tumor is removed. Again the tumor or tumors may be malignant, and the other ovary must be carefully inspected and even incised at the time of operation.

Brenner tumors are pathologic curiosities. The tumor is solid, remains small, and has no special clinical character.

Dysgerminoma is another pathologic curiosity. The tumor is solid and causes pelvic symptoms through pressure due to its size and weight. The clinical feature to be remembered is that it occurs in young adults and in children. It must be considered malignant.

Carcinoma of the Ovary.—From what has been said it is obvious that carcinoma of the ovary may develop in an apparently benign tumor, while in other cases the initial growth is malignant. The latter are particularly lethal since the neoplasms grow silently until metastases occur and give rise to symptoms of pressure, with vague gastrointestinal or abdominal distress. By the time ascites is present, both ovaries are commonly affected, though to a variable extent.

Secondary carcinoma of the ovary may be a metastatic spread from other pelvic organs such as the uterus or rectum, or it may be secondary to a distant focus such as the colon, stomach, or breast. It has become apparent in recent years that two primary lesions may occur in the same patient. The secondary involvement of the ovaries is almost always bilateral, and although the typical *Krukenburg tumor* is a solid type, other metastatic tumors may be cystic and show few malignant characteristics until pathologic study is made. If, on the other hand, there is a generalized carcinomatosis of the peritoneal cavity, it may be difficult to establish the primary source even at autopsy.

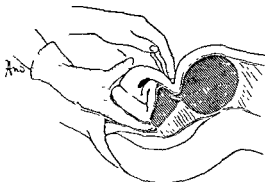


Fig 357.—Bimanual examination for ovarian cyst.

Symptoms and Signs Common to Ovarian Tumors.—The smaller tumors tend to be asymptomatic, but as the size increases, the patient complains of a feeling of heaviness or dull ache in the pelvis, with possibly bladder pressure symptoms. If the tumor arises out of the pelvis it usually is a midline tumor, and the swelling is sooner or later noted by the patient. Menstrual irregularities are not common (unless the tumor secretes hormones), but as pelvic congestion and disorganization of the ovary proceed, the menstrual loss may vary in quantity and interval.

On abdominal examination the swelling may be apparent. On palpation it is usually a smooth cystic midline tumor arising from the pelvis to a point at or below the umbilicus; it is dull to percussion and silent on auscultation. Ascites is rarely demonstrable with benign neoplasms but is common with malignant changes. On bimanual examination the cervix

and body of the uterus are felt to be quite separate from the mass arising out of the pelvis, but the bulk of the latter may displace the uterus back or to one side. With bilateral tumors the differentiation of the uterus is more complex. The mass almost always feels cystic, and this point plus the fact it is separate from the uterus differentiates it from a fibroid in most cases. Rectovaginal examination is important since in endometriosis or malignant metastases the pouch of Douglas is involved. An x-ray of the abdomen outlines the contour of the tumor, and the presence of teeth indicates a dermoid cyst.

Acute pain usually is caused by a *complication* such as torsion of the pedicle, intracystic hemorrhage, and rarely rupture or infection. These create all the symptoms and signs of an acute abdominal emergency.

The greater proportion of ovarian tumors is innocent in type, but malignant changes either primary or secondary are present in approximately 25% of such cases. Malignant tumors are more common in the older age groups. The symptoms are exaggerated, the pain is more marked, and on examination the debility of the patient, the ascites, the irregular abdominal masses, the fixation of the pelvic masses, and the secondary deposits in the pouch of Douglas are all suggestive.

Treatment.—The treatment of a well-defined ovarian tumor is surgical removal. Some of the smaller cysts may be observed for 2-3 months, and a small proportion will disappear only to recur later. In the menopausal age group, any ovarian tumor should be removed.

If the tumors are bilateral and apparently innocent, the larger cyst is removed while the smaller may be shelled out, leaving some ovarian tissue. It is difficult to define the presence of carcinomatous changes, and therefore all cysts should be opened and carefully examined in the theater after removal. If copious ascites is present and if the tumors are bilateral and papilliform projections are present on the exterior of the cyst, the pathologic report will bear out the clinical impression of malignancy. The uterus should be removed in these cases. In patients with cancer of the ovary with metastases, little can be accomplished, but

biopsy of the tumor should be done to aid in the diagnosis. If practical, the other abdominal organs should be examined for a primary source. The use of radioactive colloidal gold in the peritoneal cavity appears to control ascites and small metastatic seedlings in some cases. Deep x-ray therapy gives variable results with carcinoma of the ovary. The use of chemical derivative therapy appears to give promising palliative results.

VAGINAL DISCHARGE (LEUKORRHEA)

Since the vaginal mucosa lacks intrinsic lubricating glands, the lining of the vestibule and vagina is moistened by a vaginal transudate and also by secretions from Bartholin's glands and from glands of the cervix. The vagina is practically always nonsterile from a bacteriologic point of view, but the organism are nonpathogenic. The mucosa is thick and the cells rich in glycogen. The hydrogen ion concentration is regulated at a pH of approximately 3.5-4.5. Should this state of acidity be disturbed, the bacterial balance is altered and vaginitis results from the multiplication of pathogenic organisms. There is a normal variation of the vaginal pH in the child and again after the menopause. In both, the reaction is alkaline, pH 7-8, the mucosa is thin, and the cells have scanty glycogen. The resistance of the vagina to infection is lowered at these ages. General debility, diabetes, and pregnancy are other predisposing causes of vaginitis.

Leukorrhea is a symptom and denotes an abnormal vaginal discharge which is not blood stained. The exaggerated discharge may be mucoid, purulent, offensive, curdlike, or watery in character and, in the absence of a foul odor or vulvar irritation, may be tolerated by the individual. Modern cancer propaganda has served to make women conscious of such discharges. Leukorrhea is usually present with precancerous lesions of the cervix, and if routine smears for cytology are taken, unsuspected cancer is sometimes disclosed.

Mucoid Discharge

Mucoid discharge may be clear or milky and is ordinarily benign in origin. It is pro-

duced by stimulation of the cervical glands and is increased just before and after menstruation. Unless very profuse, no specific treatment is required, although any simple cervical erosion or laceration should receive treatment.

Purulent Discharge

This denotes the presence of pathogenic organisms, and while gonorrheal infections must always be kept in mind, the more common infections encountered in practice are due to *Trichomonas vaginalis*, *Monilia (Candida) albicans*, *Escherichia coli*, streptococci, or staphylococci, or any combination of these. The infections are most common through the childbearing period, and there is usually a cervicitis present with varying degrees of erosion and laceration. Other symptoms are foul odor, pruritus, and chafing of the vulvar area, with painful intercourse.

Trichomonas vaginitis is common during the reproductive period, and the causative organism is the *T. vaginalis*, a flagellated protozoa. The *T. vaginalis* is often found in the vagina without symptoms and thrives in a medium, pH 5.5-6. With overwhelming infection the patient is in continuous distress, and there is a sensation of burning on micturition. Examination of the worst cases reveals an excoriated vulva with the redness extending to the thighs. On gently inserting the speculum, a copious, frothy, greenish, foul-smelling discharge is observed. The inflamed vagina and cervix, when swabbed clear of discharge, have a peculiar strawberry appearance. The presence of motile *T. vaginalis* in great numbers on microscopic study of a smear diluted with normal saline confirms the diagnosis.

The treatment is restoration and maintenance of a high vaginal acidity to destroy the infecting organisms. The vagina is cleansed and one of the many proprietary medications insufflated or inserted. The patient continues the insertion of the suppositories night and morning as directed. This treatment is maintained throughout the whole cycle, and an occasional cleansing douche of vinegar solution may be used. Intercourse is temporarily prohibited. A cure is not to be assumed until several consecutive periods have passed with-

out relapse, which is most likely to occur in the postmenstrual phase. When the vaginitis is persistently recurrent, the husband's prostatic secretions should be examined as a possible source of infection.

Monilia (Candida) albicans vaginitis creates less discharge, although the pruritus is severe. The external irritation is more marked if the patient is pregnant or has diabetes mellitus. *Moniliasis* may occur at any age. On examination the typical vaginal findings are the whitish, curdlike plaques which are adherent to the vaginal wall and cervix. The inflammatory reaction is moderate, though the introitus is often reddened. The diagnosis is apparent on inspection but may be substantiated by demonstrating the mycelia and spores of the fungus in a smear diluted with normal saline. In an atypical vaginitis both *T. vaginalis* and *Monilia* may be present.

The treatment is careful vaginal toilet with a cleansing solution and then the application of a preparation such as gentian violet either in fresh 1% aqueous solution or as a gel. This may be repeated once or twice as required. Glycosuria should always be excluded, and if the condition persists, blood sugars should be estimated.

Mixed bacterial vaginitis results from staphylococcal, *E. coli*, or streptococcal infections, and its only characteristics are foul odor and persistence of the discharge. This type of infection seems more prone to develop in the young girl, nullipara, or postmenopausal woman. Smears and cultures will demonstrate the predominant bacteria, and if sensitivity tests are performed, the applicable antibiotic may be given. In other instances sulfonamide cream or antibiotic suppositories may be inserted. Recurrence often occurs after the antibiotics are discontinued; but if they are continued too long a troublesome monilia may be superimposed. If instillations are used, local allergic reactions may be very distressing. Normal saline or vinegar solutions may be used as cleansing douches.

If the discharge is very foul and profuse, the presence of advanced carcinoma or foreign body in the vagina may be suspected. In the postmenopausal patient, intermittent escape of foul-smelling purulent discharge may be caused by a pyometra.

Watery Discharge

Watery discharge, if continuous, may be due to a vesicovaginal fistula. If intermittent, it is possible that it arises from the cavity of the uterus or the fallopian tubes. In pregnancy, it may indicate escape of amniotic fluid in the last trimester.

Senile Vaginitis

This results from the marked atrophic changes due to deficiency of estrogen after the menopause. A mixed infection is present and is usually mild in character. The chief complaint is a sensation of dryness with dyspareunia. The discharge may be watery, though, on rare occasions, bleeding may occur from the atrophic mucous surfaces.

The only treatment required is reassurance associated with the use of a simple lubricating jelly. If this does not improve the condition, estrogen may be given either as a local instillation or by oral administration.

Acute Gonorrhea

Acute gonorrheal infection usually develops within a few days following intercourse. The acute inflammation, which involves the urethra, Skene's and Bartholin's glands, and the cervix, causes profuse discharge. This creates soreness and irritation of the vulva which is aggravated on voiding. The history and physical findings suggest the diagnosis. Smears of pus obtained from the urethra or cervix may reveal the gonococcus, an intracellular gram-negative diplococcus, but cultures should be taken for positive diagnosis. Blood specimens should be sent for serologic study. However, therapy should be immediately instituted and penicillin is specific. Proof of cure includes subsidence of the inflammation and repeatedly negative smears and cultures taken at weekly intervals and after menstruation.

It is advisable to have the patient in the hospital for a few days for bed rest and for antibiotic treatment which can then be given under careful supervision. The aim is to control the infection before it invades the upper genital tract. All contacts if possible should be identified and treated.

Chronic Gonorrhea

Chronic gonorrhea of the lower genital tract is difficult to diagnose. The history of acute illness, infection of the male partner by intercourse, and the presence of residual inflammatory changes in the lower or upper genital tracts are suggestive. Positive smears and cultures from the cervical discharge are difficult to obtain unless there is an acute exacerbation appearing after a menstrual period or in association with pregnancy. A new infection must be excluded in both these instances.

ABDOMINAL PAIN IN PREGNANCY

Abdominal discomfort or pain is a very common complaint during pregnancy. In most instances this is fleeting in character and not of serious consequence. There are other pains, however, which cause more distress. While most of these pains are peculiar to pregnancy and involve the uterus, the appendages, and supporting ligaments, it must be emphasized that a pregnant woman may develop any acute surgical condition. Furthermore, because of the stretching of the abdominal wall and displacement of abdominal organs due to the enlarged uterus, the classic symptoms and signs may be obscured and the true diagnosis delayed.

An important point has been stressed about pyrexia when one is dealing with acute abdominal pain in pregnancy. It has been observed that the onset of abdominal pain associated with pyrexia is due to some conditions outside the pregnant uterus; but the absence of early pyrexia does not, of course, exclude extrauterine lesions, either obstetric or surgical. An infected abortion and a degenerating fibroid are exceptions to this rule.

Nausea and vomiting of early pregnancy, when associated with vague abdominal pains or intestinal colic, may suggest the diagnosis of acute appendicitis, unless the menstrual history is elicited. The consequences are more serious, however, if the nausea, vomiting, and pain caused by an acute appendicitis are attributed to an associated early pregnancy.

As a matter of convenience the pregnant period is divided into trimesters, and various

conditions which are encountered in each trimester follow.

First Trimester.—A frequent complaint of early pregnancy is that of recurring sharp pains in both lower quadrants. They usually occur when the patient moves suddenly and are thought to be due to *tension on the round ligaments*. These pains tend to disappear when the enlarging uterus becomes more fixed.

Constipation is very troublesome during this time and may alternate with attacks of intestinal colic and diarrhea due to dietary indiscretions or purgation.

Abortion and *ectopic pregnancy* are most common in the first 3 months. Abortion of an intrauterine pregnancy begins with lower abdominal discomfort, regular uterine contractions develop, vaginal bleeding increases, and as the cervix dilates the products of conception are expelled, either completely or incompletely. When intrauterine or pelvic infection follows, the clinical picture is that of a pelvic peritonitis. Attempts to induce criminal abortion cause symptoms and signs which are most atypical. *Hemorrhage and pelvic peritonitis* are perhaps the most common complications. *Ectopic pregnancy* is the most frequently misdiagnosed gynecologic disease.¹ Bleeding from a corpus luteum of pregnancy simulates an ectopic pregnancy, but the acute signs and symptoms rapidly subside.

The pregnant uterus is often found to be retroflexed and retroverted in the first 8 weeks, but usually between the 8th and 12th weeks spontaneous correction occurs. If the retroversion persists, impaction in the pelvis may result, and this may create very acute symptoms. Acute lower abdominal pain and retention of urine develop in a woman who is known to be pregnant. Overflow incontinence occurs. The large tender bladder is palpable and must not be mistaken for the enlarged uterus. On pelvic examination, the cervix is drawn up under the symphysis, and the impacted, retroverted uterus fills the pelvic cavity. After the bladder is drained, it will be apparent on bimanual examination that the pelvic mass is the uterus. Its position, as a rule, becomes corrected spontaneously with sedation and posture if the bladder is kept empty.

A *pelvic hematocoele* or an *ovarian cyst* always displaces the uterus, and therefore the

latter is felt separate from the mass. A cervical fibroid produces pain, retention of urine, and displacement of the cervix, but the symptoms and signs of pregnancy are not present.

Second Trimester.—In pregnancy, *physiologic distention and dilatation of the ureters* cause stasis of urine, and acute pyelitis is a frequent complication. Right-sided pyelitis is more common. Ordinarily the symptoms, signs, and urine analysis are diagnostic. Subacute renal infection may give rise to less typical symptoms and signs which are referable to the back or upper abdomen. This type of pyelitis may remain undiagnosed unless an acute exacerbation develops and a more complete renal tract investigation is made.

Renal calculi may cause attacks of colic for the first time during pregnancy. The symptoms and signs suggest the diagnosis, and this is verified by routine investigation.

Acute appendicitis in pregnancy is a serious complication. An attack, very rarely the first, is most likely to develop in the first or second trimester but may occur later. Nausea, vomiting, and abdominal discomfort, associated with the increased white cell count and rapid sedimentation rate, all normally present in pregnancy, tend to obscure the diagnosis. After the 5th month, the anatomic displacement of the cecum and appendix gives rise to atypical physical signs. An acute pyelitis of the right kidney must be excluded.

Once an acute appendicitis is diagnosed, operation is imperative. If there is peritonitis present, the chance of abortion or premature labor ensuing after operation is increased, and the maternal prognosis is more grave. It is important to note that peritonitis may cause uterine spasm which simulates the clinical signs of concealed retroplacental hemorrhage.

Perforation of a peptic ulcer during pregnancy has been reported. A diagnostic aid is the history of prior investigation or treatment for a suspected ulcer.

Intestinal obstruction due to adhesions from previous operations may occur as the adherent intestinal coils are displaced and kinked by the enlarging uterus. Paralytic ileus is a rare complication of acute pyelitis with pregnancy.

More commonly, acute abdominal pain in pregnancy is caused by *degeneration of a*

fibroid or by an *ovarian cyst* which has undergone torsion, intracystic hemorrhage, or rupture. Such pelvic tumors are usually identified at antenatal examination, and the fact that their presence is already known aids in the diagnosis and treatment when complications develop.

A fibroid complicating pregnancy is ordinarily treated conservatively until the child is close to term. The treatment then is an obstetric problem. An ovarian cyst associated with pregnancy should be removed by operation at a time when abortion is least likely to be precipitated but before the bulk of the pregnant uterus makes the operation difficult.

A *hematoma in the rectus sheath* is a rare accident which occurs in the second trimester of pregnancy or later. The rupture of the muscle or blood vessel is usually caused by unusual muscular stress. The sharp pain and the tense abdominal wall with very acute local tenderness create a surgical emergency. The findings on abdominal palpation and pelvic examination while under the anesthetic clarify the diagnosis, which is fully established when the hematoma is incised on opening the abdominal wall.

Third Trimester.—Again, in the third trimester, the patient may complain bitterly of discomfort due to the *pressure of the enlarged uterus*. This, of course, is aggravated if an acute hydramnios or a multiple pregnancy is present. Fetal movements may make certain points on the uterus very tender. Pain in the liver region in a patient with toxemia of pregnancy is a warning symptom of an impending eclampsia.

Retroplacental hemorrhage, if small, causes considerable distress which may subside in time. If the concealed hemorrhage is large, the distention of the uterus becomes marked, acute severe pain is present, and a state of shock ensues. There is usually an associated toxemia of pregnancy or a history of local trauma. On palpation the uterus is distended, hard, and tender. The fetal heart sounds are absent.

Rupture of the pregnant uterus is a serious occurrence which usually happens near term or during labor. *Spontaneous rupture* is more common, and it is so frequent in cesarean

section scars, whether in the upper or lower segment, that the dictum, "once a section always a section," is almost a rule. An obstructed labor will end in rupture of the overdistended lower uterine segment if it is not recognized and treated. The unwise use of Pitocin in labor before the cervix is dilated may be the cause. *Traumatic rupture* may result from external trauma such as blow on the abdominal wall or a crushing injury. More frequently it is a direct result of forceful obstetric procedures such as internal version late in labor.

If the patient is in labor, the contraction cease and abdominal pain and tenderness develop. Shock with evidence of internal hemorrhage ensues, the fetal parts will be easily palpable, and the fetus usually dies. If the rupture occurs during delivery, the third stage is abnormal; there is delay in expression of the placenta, postpartum hemorrhage, and progressive shock. The diagnosis will not be made until one explores the uterine walls by a bimanual examination.

Rarely an *extruterine abdominal pregnancy* survives to the last trimester, and abdominal pain and discomfort are distressing. The diagnosis is rarely made before the abdomen is opened because of the abdominal symptoms or for a proposed cesarean section.

The pain of abortion or the pain due to the onset of premature labor or labor at term are usually obvious and remain the most common causes of abdominal pain in women of the childbearing age, despite the misleading history sometimes given by the patient.

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Endometriosis (Demonstrates gross and microscopic distribution, and characteristics of commonly encountered endometrial lesions, conservative treatment of ovarian pathology is particularly stressed) (1953) (By Edward D. Allen, MD, Chicago)	31 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn
Ovarian Tumors (Demonstrates the more common benign and malignant ovarian tumors in situ and after removal) (1953) (By Herbert E. Schmutz, MD, Chicago)	26 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn
Uterine Cancer. The Problem of Early Diagnosis (Illustrates the complete practicability of drastically reducing deaths from cancer of the uterus by adherence in general office practice to the routine pelvic examination of all adult women) (1951) (By National Cancer Institute, U.S. Public Health Service, Bethesda, and American Cancer Society, New York)	22 min	Sound Color	National Cancer Institute Bethesda, Md
Manchester Operation (Donald Fothergill Operation) for Uterine Prolapse (1941) (By Louis E. Phaneuf, MD, Boston)	27 min	Silent Color	American Cyanamid Co. Surgical Products Division Danbury, Conn
Vaginal Hysterectomy and the Repair of Prolapse Demonstrating Heaney Technique With Modifications (1956) (By Fred J. Hofmeister, MD, Milwaukee)	17 min	Sound Color	Ohio Chemical & Surgical Equipment Co. 1400 E. Washington Ave. Madison 10, Wis

Chapter 29

Hernia

W. Mason Couper, MD

Hernia may be defined as a protrusion of a viscus or a part of a viscus from the cavity in which it is normally contained through a congenital or acquired aperture or weakness. Subsequent descriptions will be restricted to types of abdominal hernias, e.g., inguinal, femoral, umbilical, and postoperative.

CONSIDERATIONS OF TERMS COMMON TO ALL HERNIAS

A *reducible hernia* refers to one in which the contents of the hernial sac can be replaced into the cavity from which they protruded, while an *irreducible hernia* is one in which the contents of the hernial sac cannot be so replaced.

An *incarcerated hernia* or obstructed hernia is one in which the hernial mass is irreducible. There is obstruction to the flow of intestinal contents in the occluded loop, but the blood supply and lymph drainage remain intact.

A *strangulated hernia* is one in which the blood supply and lymph drainage of the hernial contents are partially or completely occluded. This condition will result in gangrene unless relieved within a few hours.

INGUINAL HERNIA

Embryologic Features.—In the male, the close association of inguinal hernia with the descent of the testis necessitates a review of the embryologic features and the path traveled by the testis in its descent. The testis develops

as a retroperitoneal structure in the region of the kidney. During its descent it carries with it a tube of peritoneum through the inguinal canal and into the scrotum. This protrusion of peritoneum is known as the *processus vaginalis*. Before birth, this processus becomes obliterated throughout its length, except for that portion which surrounds the testicle. This portion is known as the *tunica vaginalis*. When the processus vaginalis remains patent, there is a direct communication between the peritoneal cavity and the scrotum. The patent processus constitutes the potential sac for a future hernia.

Acquired and Congenital Factors Governing Inguinal Hernia.—In the male the majority of indirect inguinal hernias are considered to be congenital in origin. The peritoneum of a patent processus vaginalis has been found in 35% of infants up to the 4th month, and many adults carry with them throughout life, a patent funicular process. Factors such as coughing, straining, lifting in the adult and crying in the child all tend to raise the intra-abdominal pressure and so put a strain on a partially closed or a poorly obliterated processus vaginalis, with the development of a hernia. Unilateral or bilateral hernias are common in infants with phimosis or pin-point meatus.

Classification of Inguinal Hernia.—There are two main types.

- 1 Oblique or indirect inguinal hernia
- 2 Direct inguinal hernia

There is a combination of the above types which may fall into a third group and is known as direct-indirect type of inguinal hernia. The protrusion is to both sides of the inferior epigastric artery.

Oblique or Indirect Inguinal Hernia

Oblique hernia is so designated because of the oblique course the hernia takes in passing through the inguinal canal. The indirect hernia has been described according to the degree of patency of the funicular process. Where the

process is open down to the testicle, the condition is described as the *congenital* or *testicular type*. When the distal portion of the processus has been obliterated with patency of the proximal portion, it is described as the *funicular type*.

Again, there are cases in which the proximal and distal portions of the processus are obliterated, and a small intervening portion remains patent. This gives rise to a collection of fluid along the cord, cystic in nature, which is described as an *encysted hydrocele of the*

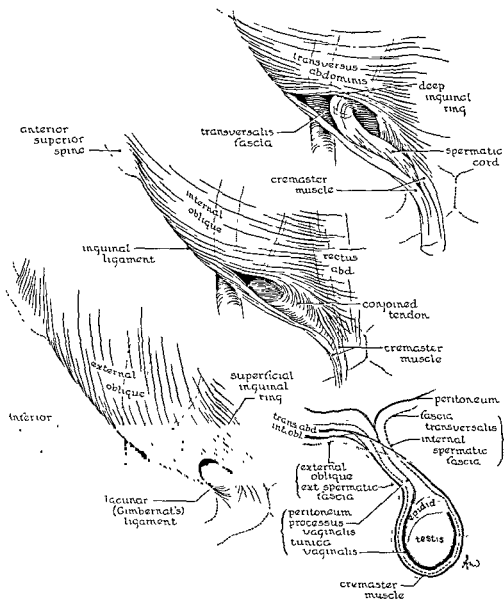


Fig 358—Anatomy of the inguinal canal.

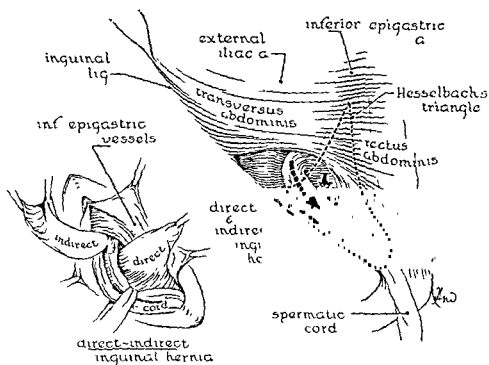


Fig. 359 —Types of inguinal hernia and anatomy of the posterior wall of the inguinal canal

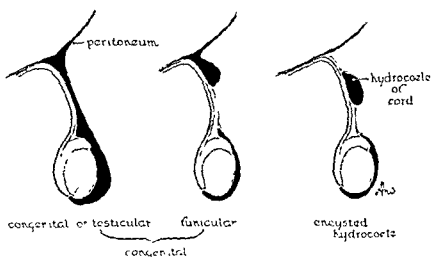


Fig. 360 —Congenital anomalies of processus vaginalis.

cord. The cystic area may be unilocular or multilocular.

Oblique inguinal hernia occurs much less commonly in females than in males. The patent peritoneal diverticulum in the female corresponding to the processus vaginalis is called the canal of Nuck.

Coverings of Indirect Inguinal Hernia.—The covering layers are as follows:

- 1 The skin
- 2 Superficial fasciae
3. Intercolumnar fascia from external oblique aponeurosis
4. Cremasteric fascia
5. The transversalis or infundibuliform fascia
- 6 The extraperitoneal connective tissue

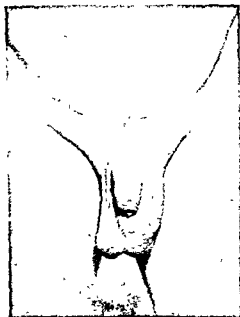


Fig 361—Left complete (scrotal) inguinal hernia

The separation of the coverings into layers will not be possible in the old, long-standing hernia, as the layers become fused together.

Contents of Sac.—Practically every abdominal organ, with the exception of the liver and pancreas, may, at one time or another, descend into the hernial sac. When the sac in the oblique type contains loops of small bowel, the hernia is known as an *enterocele*. At times the omentum forms the only structure within the sac, and this condition is described

as an *omentocele*. Occasionally a part of the wall of the small bowel is caught in such a way that only a portion of its lumen is occluded; this is known as *Richter's hernia*. When a Meckel's diverticulum is found in the hernial sac, it is known as *Littre's hernia*.

Clinical Features and Diagnosis.—At the onset, the characteristic feature is a dragging sensation in one or the other groin. Following this pulling and dragging sensation a lump appears as a rounded swelling just above Poupart's ligament and lateral to the spine of the pubis. Straining, coughing, and running make the lump more obvious. On lying down, the swelling usually disappears. When the swelling emerges at the external ring, the

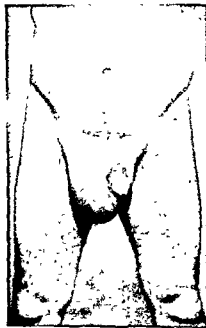


Fig 362—Right complete (scrotal) inguinal hernia, operation demonstrated a hernia-en-glissade (sliding hernia).

condition is named an *incomplete inguinal hernia*, and when it descends beyond the external ring to gain the scrotum, it is designated a *complete or scrotal hernia*.

Examination of the patient is usually carried out with the examiner seated directly in front of the subject. The clothing is removed from the umbilicus to the knees. When the patient is asked to cough, a swelling may be

seen in one or the other inguinal region. With the examining finger introduced into the inguinal canal (by invaginating the neck of the scrotum through the external ring), a definite impulse may be palpated after coughing, or at times the examining finger is displaced beyond the external ring by the hernial protrusion. The course and obliquity of the hernia allow one to appreciate the indirect type. Symptoms of pain referred to the testicle of that side or gastrointestinal disturbances with occasional nausea are generally due to traction by a large hernia on the mesentery. Percussion of the hernia may enable the examiner to distinguish between intestine or fluid in the sac. A resonant note, on percussion, suggests gas in the intestines. The clinical differentiation between the oblique inguinal hernia and the direct type, by the palpation of the inferior epigastric artery, is extremely difficult and therefore cannot be relied upon as an important point in making the differentiation between these two types of hernia. More significant is the diffuse bulge passing directly forward from the peritoneal cavity characteristic of the direct type.

Differential Diagnosis of Inguinal Hernia.—

Inguinal adenitis A mass somewhat oval in shape presents in the groin. It is tender and painful and usually moves freely under the skin. Occasionally other lymphatic nodes in the neighborhood, showing some enlargement and tenderness, are noted. The inguinal canal is found empty on examination, and the enlarged node or nodes are palpated to one or the other side of the canal.

Ectopic testis or a partially descended testis may appear as an irreducible swelling in the inguinal region. This condition is readily recognized by the shape and consistency of the tumor, by the testicular sensation experienced by the patient when the swelling is compressed, and by the fact that no testis is present in the corresponding side of the scrotum.

Lipoma of cord consists of a soft lobulated fatty mass, not tender, not increasing greatly in size, and usually present for a considerable period of time.

Encysted hydrocele of cord appears as a tense oval tumor, attached to the cord mass when the cord is made taut by traction on

the testicle. It may be readily palpated in the external inguinal ring. The cyst can be transilluminated, which fact makes the diagnosis an easy one.

Psoas abscess may present just above Poupart's ligament or extend beyond it. The swelling is soft and fluctuant and may be diminished in size by compression. It is dull to percussion and gives a slight impulse on coughing. Clinical and radiologic examination of the spine will disclose the source from which the abscess originated.



Fig. 363.—Congenital indirect inguinal hernia in a female patient. (Note descent of hernial mass into labium majus.)

Hematoma of the cord. A history of injury, with ecchymosis of the skin above Poupart's ligament and with swelling along the cord, usually enables one to make the diagnosis of a hematoma due to trauma.

Treatment of Indirect Inguinal Hernia.—

Three methods have been used:

1. Mechanical, by use of truss
2. Surgical repair
3. Fibrosis of the sac by injection of solutions

Use of Truss—This method has been used since ancient times, and the use of a type of bandage or belt has always been associated with the early references to hernia treatment. Not all inguinal hernias are suitable for operation, and although there are many difficulties attending the use of trusses, there are, nevertheless, indications.

For the use of a truss for the infant, see Chapter 30.

Adults are less likely to obtain a cure from the wearing of a truss but may derive considerable benefit by having well-fitted support. The adult should remove the truss after retiring and put it on before getting out of bed. It is preferable to wear the truss next to the body. Any underclothing beneath the pad does not facilitate proper fitting and encourages the hernia to slide down beneath or to one side of the truss. Should the hernia become irreducible, the truss cannot be worn. Trusses are contraindicated in cases of obesity, hydrocele, varicocele, and incomplete descent of the testicle. For those patients with a chronic cough or in the presence of a large hernia, it may be advisable to wear the truss at night. Irritation of the skin caused by the pad of the truss can be overcome by careful bathing and dusting the area liberally with talcum powder. Attention should be paid to the cleansing of the truss itself. When a truss is properly fitted, it should be worn without discomfort, and the patient should be able to stand up, bend down, cough, and do his work without protrusion of the hernia around the pad.

Surgical Treatment of Inguinal Hernia.—The fundamental principles which underlie the surgical repair of inguinal hernia are thorough exploration of the canal, identification and excision of the peritoneal sac, and repair of the canal floor, or reconstruction of the defect in the abdominal wall.

The methods of carrying out the surgical repair are many and varied. Indeed, practically every known procedure has been either rediscovered, modified, or rewritten in an effort to seek the perfect repair. No attempt will be made to review or describe the many modifications and methods by which a repair of the inguinal canal can be accomplished. Several well-recognized standard procedures will be used to illustrate the underlying principles.

The *Bassini operation* embodies two main principles. removal of the hernial sac and repair of the defect in the posterior wall of the canal.

The operation is carried out by making an incision, parallel with Poupart's ligament and one-half inch above it, extending from the spine of the pubis to beyond the internal ring (one-half inch above the midpoint of Poupart's ligament). Camper's fascia, represented by the thick subcutaneous yellow fat, as well as the deeper Scarpa's fascia, is divided to expose the shiny aponeurosis of the external oblique muscle. The external ring should be isolated. At times the clear outline of the margins of the external ring cannot be seen, but the edges can be readily palpated by introducing the finger along the course of the cord where it emerges at the pubic spine; by following the cord upward, the prominent edges of the ring can be palpated. Now the aponeurosis is divided in the direction of its fibers downward through the external ring and upward to just beyond the internal ring. The ilio-inguinal nerve will then be seen running along the cord lying on the cremasteric fibers. The cremasteric fibers are separated to expose the cord with its accompanying vessels. The sac in an indirect oblique hernia is found lying superficial to the vas. It is picked up and dissected from the surrounding structures. The neck is cleaned. The sac is opened and inspected for contents. The contents if viable are reduced. When strangulated nonviable bowel is found, it is resected. Adherent omentum is dissected free and returned to the abdominal cavity. To close the neck, a transfixion suture is used, or, if the neck be wider than two fingerbreadths, a purse-string suture may be used. The redundant part of the transfixed sac is excised. The second phase in the operation consists of a repair of the defect in the posterior wall of the canal. This is carried out by suturing the conjoined tendon down to the reflected edge of Poupart's ligament posterior to the cord. The external oblique is then closed over the cord and the superficial fasciae and skin sutured to complete the procedure.

Halstead added a modification to the above method by transposing the cord superficial

to the sutured external oblique aponeurosis. In this way the posterior wall is given added strength by the use of the external oblique aponeurosis. In his earlier work, like Ferguson, he did not transpose the cord but sutured the conjoint tendon to Poupart's ligament, anterior to the cord except at the level of the external ring where an opening was left for the passage of this structure.

ligature of the sac and its excision close to the internal ring. Closure of Scarpa's fascia and the skin is all that is necessary.

Where a very large inguinal hernia exists the external and internal rings may come to lie across one another, thus leaving a large defect in the posterior canal wall to be repaired. It is in this type that modifications of the above methods are used. The use of

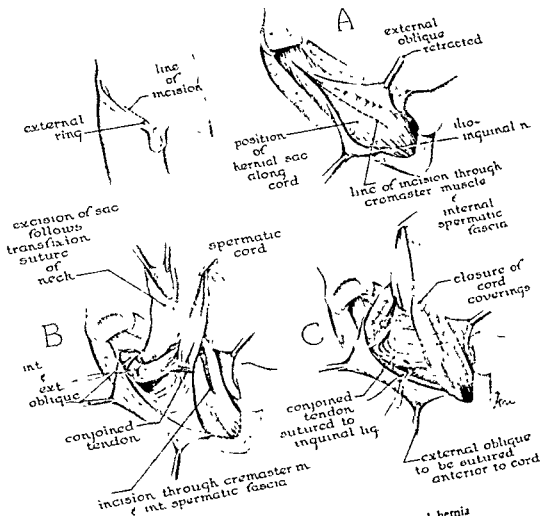


Fig 361—Bassini repair of indirect inguinal hernia

In the treatment of an oblique hernia in a very young infant or child, the procedure of high ligation of the sac is widely practiced. No attempt is made to disturb the contents of the inguinal canal. The sac is picked up at the external ring and freed at this point from the cord and vessels. Sufficient cleansing can be carried out at this level to permit the

living fascia as strands to interlace the defect has been described by McArthur and Gallie. A large variety of materials has been used to overcome the defect—floss silk has been woven through the defect, also filagree wire, and lately tantalum or stainless steel mesh or skin have all been used in selected cases.

Sliding Hernia or Hernia-en-Glissade

This type can be defined as a hernia in which the posterior wall of the sac consists of the cecum on the right side or the pelvic colon on the left side. It is found in the inguinal region and is brought about by the slipping, in a downward direction, of the viscus and the posterior parietal layer of peritoneum.

Symptoms and Diagnosis.—*Sliding hernia* of the large intestine is generally of the indirect inguinal type. It occurs in males over the age of 45 years and is more common on the right side. The condition, however, does occur quite frequently on the left side. Typical examples are shown in Figs 364 and 366. If the hernia is reducible, the symptoms are very similar to those found in the ordinary inguinal hernia, with the exception that the sac is somewhat larger. The internal ring becomes stretched and lies over the external ring so that reduction of the hernia is usually easy, and strangulation seldom occurs. As the signs are not well defined in this type of hernia, the diagnosis is not made until operation. On the left side, the presence of the colon in the sac may give rise to symptoms of constipation. The diagnosis may, at times, be established by radiologic examination of the colon, the barium being seen in the hernial sac. Intestinal obstruction, following such a procedure, can occur with the barium becoming inspissated in the herniated colon. This is more likely to occur when a free loop of sigmoid has descended into a left inguinal hernial sac. The condition is not a true sliding hernia which usually involves the lower part of the descending colon.

Treatment of Sliding Hernia.—The use of a truss is not satisfactory in this type of hernia because of the large size of the hernial opening. Radical operation is the treatment of choice. The exposure of the sac is the same as in the ordinary type of inguinal hernia. In this type, the bowel forms the major portion of the posterior wall of the sac. Accidental opening of the colon can easily occur while exposing the sac. The redundant portion of peritoneum is cut away and the colon mobilized to allow it to be returned to the abdomen. The repair of the defect is difficult. Many

writers suggest opening the abdominal cavity and fixing the colon to the posterior abdominal wall. Others complete the repair by bringing the conjoined tendon down to the reflected edge of Poupart's ligament, as in the ordinary oblique inguinal hernia.

Recurrence is very common following the repair of this type of hernia because of the extensive defect and the tendency of the viscus to slip down again.



Fig. 365.—Demonstration of sliding hernia by use of barium enema (Note descent of descending colon into scrotum.)

Direct Inguinal Hernia

A direct inguinal hernia is one in which the protrusion takes place through Hesselbach's triangle. This triangle is bounded medially by the lateral border of the rectus abdominis, below by Poupart's ligament, and laterally by the inferior or deep epigastric artery. The hernia is rarely if ever on a congenital basis. It may be unilateral or bilateral. It is almost entirely confined to males after the fourth decade. It is thought to be due to weakness in the transversalis fascia with atrophy of the conjoined tendon.

Clinical Features and Diagnosis.—There is an oval swelling in the groin, which presents above Poupart's ligament. The swelling increases in size on coughing and if reducible, disappears on lying down. On examination, the finger, when inserted into the external

inguinal ring, gives the examiner the sensation of going directly back into the abdomen, whereas in the indirect type, the finger takes an upward course along the *inguinal canal*. The swelling rarely descends into the scrotum. There is a dragging sensation but less pain than in the indirect type. At times it is impossible to differentiate between the two types; the final diagnosis is made at operation. Palpation of the deep epigastric artery to distinguish between the indirect and direct types is seldom possible and rarely can be relied upon in establishing the diagnosis. The differential features are those described in the oblique type.

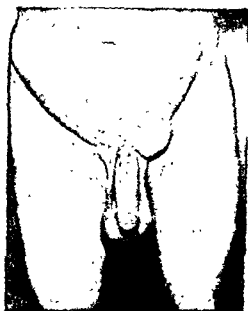


Fig. 366.—Direct inguinal hernia.

Treatment of Direct Inguinal Hernia.—In selected cases, conservative methods, such as the wearing of a truss, may suffice, but the majority of these cases come to operation. The operative features follow the pattern described under indirect hernia, except for the handling of the sac. When small, it need not be excised but may be pushed back through the defect in the transversalis fascia by invagination, with repair of the fascial defect. The conjoined tendon is sutured to Poupart's ligament over the invaginated sac beneath the cord. When a larger sac is present, it should be opened and the redundant tissue excised. The peritoneal defect is then closed. The re-

maining features in the repair are as described previously.

The combined direct-indirect inguinal hernia is converted into a true oblique type and dealt with as such.

Complications Following Operative Repair of Hernia.—Careful preoperative care and thorough cleansing of the area before operation do a great deal to lessen the incidence of *wound infection*. The introduction of antibiotics has further helped to reduce this problem, and the practice of giving an antibiotic routinely is used by many surgeons. The use of nonabsorbable suture material (silk, cotton, or wire) results in less post-operative tissue reaction and a lower recurrence rate. Occasionally sinuses may supervene if infection occurs from the presence of this foreign material.

Hematoma is one of the more frequent complications of *herniotomy*. It is most common after the removal of the large scrotal sac. The hematoma may be confined to the cord or to the inguinal canal, or subcutaneous tissues, or it may be large and fill the scrotum. If it is extensive, the wound should be reopened at the earliest moment and the blood evacuated. Chemotherapy should be routine used to prevent secondary infection.

Lung complications, mostly due to atelectasis, may develop. It has been found that these complications occur least with general anesthesia and somewhat more frequently with spinal and local infiltration. Pulmonary embolism has occurred.

The later complication of *recurrence of the hernia* varies a great deal from clinic to clinic, the percentage of recurrences ranging from 5-20%. The mortality rate is low in this operation, being under 1%. This, of course, does not include the cases of inguinal hernia complicated by bowel obstruction or strangulation.

FEMORAL HERNIA

A femoral hernia is one in which the hernial protrusion takes place through the femoral ring into the femoral canal.

The boundaries of the femoral ring are medially the lacunar (Gimbernat's) ligament, laterally the femoral vein, posteriorly the

ramus of the pubis, covered by the pectineus fascia and ligament, and anteriorly the inguinal ligament.

Etiology, Sex, and Incidence.—Femoral hernia is found much more frequently in females than in males, in the ratio of 3:1. The condition is rarely found in children. When present in adults it may be unilateral or bilateral.

The etiology of the development of femoral hernia, on a congenital basis, is not so universally accepted as in the case of indirect inguinal hernia. There are many who feel there is, in this type of hernia, a preformed pouch or peritoneal diverticulum which has

the sac, it may be overlooked altogether in obese patients. The hernia appears as a small globular swelling just lateral to the spine of the pubis and below Poupart's ligament. Unless the sac contains intestine, there may be very little increase in size on coughing, and when the patient lies down, the contents of the sac may reduce slightly but never completely disappear. There remains a small fatty nodule after the main contents of the sac have been reduced. This remaining nodule may represent the fatty plug filling in the femoral ring along with the large lymph node at the apex of the femoral canal (gland of Cloquet). Pain is not a prominent feature, but if omentum or intestine is contained in the sac, the patient may complain of a dragging sensation or colicky pain. The pain usually radiates down the anteromedial aspect of the thigh. Gurgling may be heard and felt if intestine is present in the sac. Strangulation in this type of hernia is more common than in the inguinal variety because of the small size of the ring and the rigidity of the surrounding structures. Richter's hernia, in which only a part of the lumen is caught by the femoral ring, is more likely to occur in femoral than in any other variety of hernia.

Differential Diagnosis.—Those conditions most commonly mistaken for femoral hernia are saphenous varix, incomplete inguinal hernia, psoas abscess, and regional lymphadenopathy.

The *saphenous varix* disappears when the patient lies down and can be readily compressed by gentle pressure over that site. There is no gurgling, but occasionally a venous hum is heard on auscultation over the area. A venous thrill may be felt on coughing. The skin may show a bluish discoloration over the saphenous varix, and there may be varicose veins on that extremity.

Progressive enlargement of a femoral hernia occurs by protrusion through the fossa ovalis, the sac being turned upward by the fascial arrangements, and the differentiation between it and an *incomplete reducible inguinal hernia* is at times difficult. Here, the inguinal hernia can be reduced by pressure exerted in an upward, outward, and backward direction, and continued pressure over the inguinal canal will prevent its descent

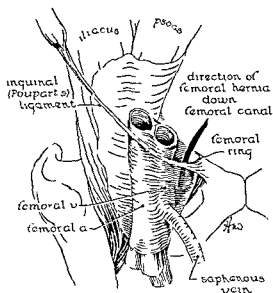


Fig 367—Anatomy of femoral canal

failed to become obliterated. This fact is borne out by the finding on many post-mortem examinations of a preformed peritoneal sac in the femoral canal. A great many femoral hernias, especially in the older age group, are thought to be acquired. In the older age group, the existence of lax muscles, attended by loss of weight and poor tissue tone, seems to predispose to the development of this type of hernia.

Clinical Features and Diagnosis.—The symptoms are much less marked in femoral than in inguinal hernia because the hernia is small, and if no intestine is contained in

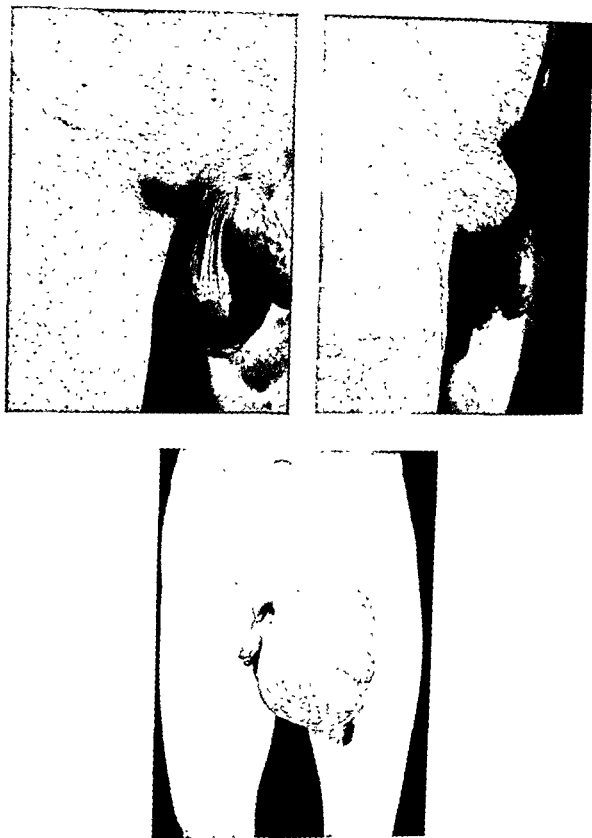


Fig. 368—Femoral hernias of various sizes (Courtesy Queen Mary Veterans' Hospital)

The reduction is much quicker than in femoral hernia, where the necessary pressure is usually downward and medially. The inguinal hernia will be seen to lie above a line running from the spine of the pubis to the antero-superior spine, whereas the femoral hernia is found below such a line.

When a *psoas abscess* presents below Poupart's ligament, it is a soft fluctuant mass transmitting an impulse on coughing. It is reducible in some cases without any gurgling

tain cases, the diagnosis may be established only at operation.

Treatment.—The cure of femoral hernia is by operation. The tendency for this type of hernia to strangulate indicates early surgical intervention. Mechanical treatment is unsatisfactory and offers little chance of cure; however, there are cases in which, for some medical reason, operation is inadvisable and a truss may be worn. The site of this hernia makes the wearing of the truss difficult, as the pres-



Fig. 369.—Lipoma simulating femoral hernia on left side, small direct hernia in right groin (Courtesy Queen Mary Veterans' Hospital.)

sound or sensation. Fluctuation may be present in a dumbbell-shaped abscess above and below Poupart's ligament. Examination of the spine usually reveals other evidence of tuberculosis.

Lymphadenopathy in the femoral region may give rise to difficulty in diagnosis. Here the swelling is firmer, with tenderness on pressure; usually there are other enlarged nodes in the neighborhood and a search may reveal some focus of infection about the leg or foot to give rise to the adenopathy. In cer-

sure pad can be readily displaced by the movements of the body and thigh. For these reasons the operative treatment is recommended. The operation is directed toward removal of the sac and closure of the femoral ring.

Operations for Femoral Hernia.—There are two recognized approaches for the surgical repair:

1. Inguinal route
2. Femoral route

Each method has its advocates and a brief description of each will be given.

Repair by the Inguinal Route.—This procedure was first described by Annandale and continues to retain that name. The incision is made 1 cm above Poupart's ligament and parallel with it for a distance of 3-4". The aponeurosis of the external oblique muscle is divided in the direction of its fibers over the inguinal canal. By retracting the edges of the divided aponeurosis of the external oblique muscle, the conjoint tendon, spermatic cord, and transversalis are brought into view. Gentle retraction of the cord upward exposes the floor of the inguinal canal, which consists of the transversalis fascia. An incision is then made in the transversalis fascia along the line of the inguinal canal and over the neck of the sac. This exposes the peritoneum, which is traced to the neck of the sac, and the sac, if small, can be freed and pulled back, or reduced. The fundus of the sac is picked up in forceps and opened, and tags of adherent omentum are freed. The neck is transfixed as in the ordinary inguinal hernia operation or closed with a purse-string suture. The redundant portion of the sac is removed, and the transfixed portion is allowed to drop back into the abdomen. The opening in the femoral ring is now closed with interrupted sutures. These run from the lower margin of Poupart's ligament to the ligamentous reflection along the ramus of the pubis (Cooper's ligament), or occasionally to the pectineal fascia. Three or four sutures usually suffice to close the opening. The conjoint tendon is then brought down to Poupart's ligament, beneath the cord. The external oblique aponeurosis and skin are closed in the usual manner.

In the Lotheisen repair, the conjoint tendon is sutured behind the cord to Cooper's ligament.

Repair by the Femoral Route.—The incision is made below and parallel with Poupart's ligament over the prominent part of the swelling. The soft tissues and fat are separated to expose the sac lying embedded in the fat of the thigh. The sac is dissected free to expose the femoral ring from below, care being exercised not to damage the femoral vein lying lateral to it. The constriction about the

neck is stretched to enlarge the opening. The sac is opened with care to avoid the possibility of injuring the contents. If the sac contains omentum or intestine, this is reduced and the neck of the sac is pulled down and closed in the method described above, and the redundant portion is removed, allowing the transfixed neck to slide back through the femoral ring behind Poupart's ligament. The femoral ring is then closed by bringing the lower margin of Poupart's ligament down to the pectineal fascia.

Strangulated Femoral Hernia

Here, the inguinal approach is preferable. The peritoneum, when exposed, can be opened to identify the ring and the obstructed intestine. If the intestine is not viable, a resection is much easier to carry out than by the femoral approach. There are cases where the constricting ring cannot be overcome by this approach, and a combined inguinal and femoral exposure may be necessary in order to free the adherent mass in the femoral region. At times the constricting ring may not yield, and it may be necessary to incise Poupart's ligament to free the constriction and expose the area.

UMBILICAL HERNIA

An umbilical hernia is one in which the protrusion takes place through the umbilical ring. The majority of such hernias are congenital in origin and appear in children. The condition is caused by failure of the umbilical ring to close following the section of the umbilical cord. This type of hernia may also be acquired and appears later in life in adults.

The discussion of umbilical hernia will include what is often described as paraumbilical hernia. This type is characterized by protrusions through small fascial defects adjacent to the umbilical ring and comprises a large group of umbilical protrusions occurring in the aged or obese female.

Umbilical Hernia in the Infant
(See Chapter 30.)

Umbilical Hernia in the Adult

This term is a rather loose one, and the group includes the paraumbilical protrusions occurring through fascial defects in and adjacent to the true umbilical opening.

In the older patient, this type of hernia is usually acquired. It appears between the ages 30-55 years. The etiologic factors are increased intra-abdominal pressure, relaxation of the abdominal muscles, obesity, ascites, multiple pregnancies, and diastasis recti. The umbilical opening, being a weak point in the abdominal wall, gives way and the protrusion develops. Women are more prone than men to develop this type of hernia. The size of the hernia varies and may reach considerable proportions.

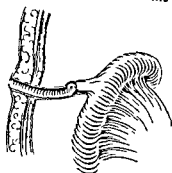
Symptomatology.—In the early case, in the obese patient, a small hernia may not be visible and is found on palpation as a rounded, firm nodule in the region of the umbilicus.

In the early stages an expansile impulse is seen when the patient coughs or strains; the tumor if reducible disappears when the patient lies down. At times the hernia appears to emerge at one or the other side of the ring and for this reason the term paraumbilical hernia has been introduced. When the hernia increases in size, it presents as an oval or rounded mass, which, as it enlarges, becomes pendulous and may reach considerable proportions. The sac becomes lobulated and irregular in outline. The contents are usually the omentum, along with the transverse colon and occasionally the small intestine. Because of the tendency of the omentum to become adherent to the walls of the sac, this type of hernia rapidly becomes irreducible. When bowel is included, peristalsis may be observed.

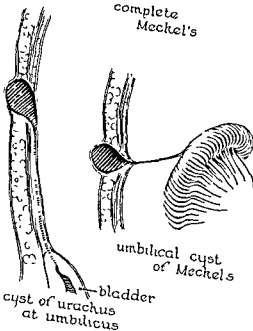
The patient complains of pain, usually dragging in character. Traction on the colon gives rise to attacks of colicky pain with constipation and at times nausea and vomiting, especially when strangulation develops.

Treatment.—The treatment is difficult as the majority of these patients are in the older age group, usually obese, and with poor musculature. The small hernia, if reducible, may be controlled by a truss. In the obese subject, an elastic belt may be worn. For the large hernia, operative repair is necessary except in those cases in which there are contraindications, such as cardiac or pulmonary disease. In some patients with very large protrusions, repair is inadvisable because of the danger of

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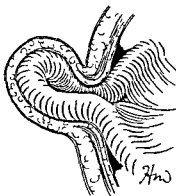


complete Meckel's



umbilical cyst of Meckel's

bladder cyst of urachus at umbilicus



umbilical hernia

Fig 370.—Pathologic conditions of the umbilicus (Complete Meckel's anomaly is often referred to as persistent omphalomesenteric duct.)

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The discussion of umbilical hernia will include what is often described as paraumbilical hernia. This type is characterized by protrusions through small fascial defects adjacent to the umbilical ring and comprises a large group of umbilical protrusions occurring in the aged or obese female.

Umbilical Hernia in the Infant

(See Chapter 30.)

Epigastric Hernia, or Hernia in the Linea Alba

Epigastric hernia is a protrusion of the abdominal viscera through an opening in the linea alba. It occurs above the umbilicus and is found rarely in children. The actual protrusion is properitoneal fat which forces its way through the fascial interstices of the linea alba, at times following the perforating arteries. Once the fatty protrusion has started, intra-abdominal pressure augments its size until a real sac of peritoneum is finally pushed out from the abdominal cavity.

Symptoms and Diagnosis.—The condition may be present and give rise to no symptoms whatsoever, being found accidentally on examination. A small, soft tumor, at times no larger than the tip of one's finger, may be palpated in the midline between the xiphoid and the umbilicus. The tumor may be present just to the right or left of the midline. There may be localized tenderness. When major symptoms are attributable to this hernia, a true peritoneal sac is usually present which may contain local viscera. In lesser degrees of such herniation where symptoms are present, a careful investigation of the gastrointestinal tract should be carried out before correction of the hernia. At times, symptoms of gastric distress, nausea, belching, vomiting, and a dragging sensation in the pit of the stomach may be present. The condition may be aggravated by bending over or sneezing. When the patient lies down, the small tumor mass may be reduced, with relief of the symptoms. A small fibrous ring is palpable at times with the tip of the finger. In a large number of cases reduction cannot be carried out, and pressure on the protrusion simply mushrooms it over the fibrous opening.

Treatment.—There is little place for mechanical treatment in this type of hernia. Operative correction is carried out by making a longitudinal or transverse incision over the hernia, exposing the fatty tumor of properitoneal fat and, if small, removing it after ligating the small artery running alongside. The defect in the fascia is closed with one or two interrupted sutures. In the larger protrusions, when a tent of peritoneum is present in the ring, the neck of the sac is ligated as described for umbilical hernia, and the fascia is closed.

Hernia in the Linea Semilunaris

The semilunar line runs from the cartilage of the 9th rib to the pubic spine and corresponds roughly to the lateral border of the rectus abdominis muscle on each side. A protrusion through this line has been called *Spiegel's hernia*. This hernia generally occurs in middle life; the sex incidence is about equal. The condition is generally acquired, though some writers refer to a traumatic variety. The vast majority develop slowly and spontaneously.

The hernia is commonly small and seldom reaches in diameter more than 1". The symptomatology is not characteristic. The presence of a small lump below the umbilicus at the lateral border of the rectus muscle, which in most instances disappears when the patient lies down, is diagnostic. The treatment is surgical following the method outlined for the epigastric hernia.

Incisional or Postoperative Hernia

It is in this group that the largest number of ventral hernias occur. These hernias occur through previous operative sites on the anterior abdominal wall.

Certain etiologic factors play a part in the development of this type of hernia:

1. Postoperative infection in a wound, especially when drainage was required.
2. Improper closure of the original wound, allowing the omentum to escape between the sutures.
3. Improper suture material.

Prophylactically, the incision should be placed to avoid unnecessary damage to motor nerves, muscles, fasciae, and blood vessels.

Symptoms and Diagnosis.—The patient complains of a weakness and a bulge in the region of a previous operative scar. The symptoms are similar to those of umbilical hernia and are aggravated by exercise and coughing. The protruded mass may not reduce on lying down, and complete reduction in the larger hernia is usually impossible, since the omentum becomes adherent to the walls of the sac as in the large umbilical hernia. In the incisional hernia, the protruded mass spreads out under the subcutaneous fat tissues in such a

increasing intra-abdominal pressure which may produce severe pulmonary embarrassment. The patient should be prepared for operation by suitable dietary regime and re-education of the abdominal musculature.

The method of choice is that described by Mayo. This consists of making a large elliptical incision in a transverse direction about the umbilical mass, usually including a large area of the adjacent fat. The incision is deepened to expose the aponeurosis on both sides of the umbilicus. The hernial mass and

The fascial edges with the peritoneum are picked up and overlapped, chromic catgut or heavy silk sutures being used. The free margin of the overlapped edges is sewn down with interrupted or continuous sutures. The thick layer of skin and subcutaneous tissue is then closed, care being taken to avoid any dead space. A dressing is applied and held in place by adhesive straps or a scultetus bandage.

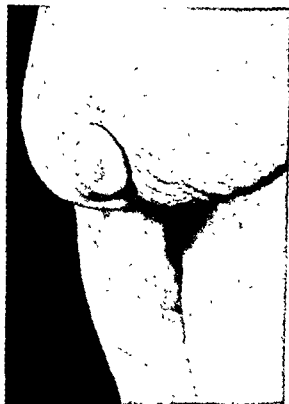


Fig. 371—Umbilical hernia in obese patient with relaxed abdominal musculature

fat are dissected toward the umbilical ring until the neck of the sac is exposed throughout its circumference. The sac is opened near the neck, the adhesions are separated, and the hernial contents are reduced into the abdominal cavity. To facilitate reduction, the hernial ring is enlarged and a portion of adherent omentum resected. The excess margin of the peritoneal sac is cut away and the edges picked up in forceps. To close the umbilical ring, the aponeurosis is incised in a transverse direction. It is usually impossible in the large hernia to close the peritoneum as a separate layer

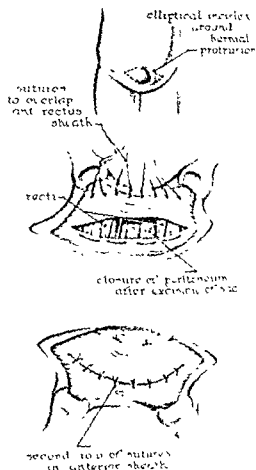


Fig. 372—Mayo operation for repair of umbilical hernia

VENTRAL HERNIA

Any protrusion through the anterior abdominal wall, other than at the inguinal, umbilical, or femoral region, is defined as a ventral hernia.

Ventral hernias may be classified into three groups.

- 1 Epigastric hernia, or hernia in the *linea alba*
- 2 Hernia in the *linea semilunaris*
- 3 Incisional or postoperative

Epigastric Hernia, or Hernia in the Linea Alba

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Treatment.—There is little place for mechanical treatment in this type of hernia. Operative correction is carried out by making a longitudinal or transverse incision over the hernia, exposing the fatty tumor of peritoneal fat and, if small, removing it after ligating the small artery running alongside. The defect in the fascia is closed with one or two interrupted sutures. In the larger protrusions, when a tent of peritoneum is present in the ring, the neck of the sac is ligated as described for umbilical hernia, and the fascia is closed.

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1. Postoperative infection in a wound, especially when drainage was required.
2. Improper closure of the original wound, allowing the omentum to escape between the sutures.
3. Improper suture material.

Prophylactically, the incision should be placed to avoid unnecessary damage to motor nerves, muscles, fasciae, and blood vessels.

Symptoms and Diagnosis.—The patient complains of a weakness and a bulge in the region of a previous operative scar. The symptoms are similar to those of umbilical hernia and are aggravated by exercise and coughing. The protruded mass may not reduce on lying down, and complete reduction in the larger hernia is usually impossible, since the omentum becomes adherent to the walls of the sac as in the large umbilical hernia. In the incisional hernia, the protruded mass spreads out under the subcutaneous fat tissues in such a

way that strangulation is not uncommon and, when present, manifests the symptoms of an acute intestinal obstruction with crampy pain, vomiting, and distention. The hernia may attain a considerable size and tend to hang down over the abdomen as in the large umbilical variety. The diagnosis is not a difficult one, with the history of an operation and the development of a slowly enlarging protrusion through that incision.

tantalum, and stainless steel mesh have been recommended.

DIASTASIS OF THE RECTUS MUSCLES

Diastasis of the rectus muscles is not a true hernia; it is brought about by stretching or widening of the linea alba in such a way as to separate the recti muscles. Two distinct types are found: infantile and adult.

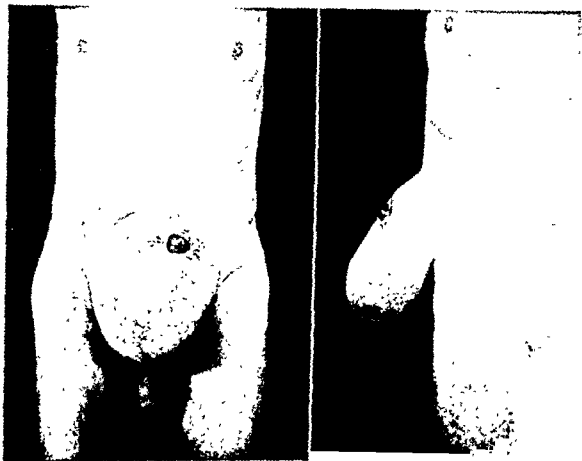


Fig. 373.—Incisional or postoperative hernia following abdominoperineal resection. Note colostomy opening at upper end of incision. (Courtesy Queen Mary Veterans' Hospital.)

Treatment.—In certain cases considerable relief may be obtained by a properly fitted abdominal support. Curative treatment is by operation if the general condition of the patient permits. The operative procedure follows that described for umbilical hernia. The old scar is excised, the contents of the sac are reduced, and the repair of the defect is carried out by overlapping the edges. When this is not feasible, some form of patch to fill in the defect may be used. Skin, fascia,

Diastasis in the Infant

The condition is noted shortly after birth when a large oblong bulge is seen. It runs from the xiphoid to the umbilicus and is made more prominent when the child cries or strains. The child is usually undernourished or premature. The gap can be readily felt by palpation along the linea alba. No symptoms are present and no active treatment is necessary. The condition disappears as the child develops.

Diastasis in the Adult

Separation of the recti is found most frequently in the middle-aged female, who has had numerous pregnancies. The diastasis is made more prominent by raising the head and shoulders from the bed. The large gap can be easily felt with the hand placed along the midline, as the hand sinks readily into the abdominal cavity. A large number of these cases are asymptomatic, but there are some patients who complain of a weak feeling and dragging sensation in the lower abdomen.

The treatment is usually supportive or mechanical, with the aid of a properly fitting abdominal belt, but when this does not relieve the condition, operation may be necessary. It is carried out along the lines described above under ventral hernia.

RARER TYPES OF HERNIA

This group includes a number of unusual hernias which are designated by the names of the anatomic sites from which the protrusions occur, e.g., obturator, sciatic, lumbar, or perineal hernia.

Obturator Hernia

An obturator hernia is one in which the protrusion passes through the obturator foramen. The hernia leaves the pelvis, following the course of the obturator vessels and nerve, to enter the thigh at the upper and inner aspect. The relationship of the obturator vessels and nerve to the sac varies. At times the sac may be to one or the other side of the vessels and occasionally it divides and is found on both sides of the vessels. This hernia is found mostly in older women and in emaciated patients.

Symptomatology and Diagnosis.—The symptoms may be intermittent with pain along the inner side of the thigh. There is a characteristic distribution of the pain which follows the course of the obturator nerve. The pain, because of its distribution and character, has been described as the Howship-Romberg sign. When present, it is pathognomonic of the condition. The neck of the sac can sometimes be felt on vaginal or rectal examination. How-

ever, a tumor can rarely be palpated on external examination except in the case of a very large hernia. Gastrointestinal symptoms, when present, are usually nausea, vomiting, colicky pain, and constipation. This hernia is rarely diagnosed before complications arise, that is, before intestinal strangulation occurs. The leg on the side of the hernia may assume an attitude of flexion, internal rotation, and adduction to relax the associated muscles. Coughing sometimes accentuates the pain in the thigh.

Treatment.—The treatment is operative, and the best approach is by the abdominal route. This has the advantage over the obturator approach of not requiring another incision when the diagnosis has been made and carries less risk of damage to the obturator artery and nerve. By opening the abdomen with a lower abdominal incision, the obstructed intestine is easily found and examined. The portion of obstructed bowel may be replaced by gentle traction from within, aided by pressure exerted from without over the thigh. If resection is necessary, it can be carried out readily. The sac is inverted and the base transfixed. The redundant portion is cut away and the defect closed by bringing together the adjacent tissues.

Sciatic Hernia

This very rare hernia is also referred to as a gluteal or ischiatic hernia.

The sciatic hernia is one in which the protrusion occurs through the greater or lesser sciatic notch. The protrusion emerges from the pelvis and takes a downward course in the buttock, following the line of the sciatic nerve. It is covered by the gluteus maximus muscle which prevents any upward extension of the hernia. The hernia may be recognized as a hard tumor mass under the gluteus muscle. This type must be differentiated from lipoma, gluteal aneurysm, or abscess.

Symptomatology.—The symptoms are those of small bowel obstruction, and the exact diagnosis of sciatic hernia is established at operation. Very rarely a soft tumor is palpated at the lower border of the gluteus maximus muscle which exhibits an expansile impulse on coughing and gurgles on reduction. Occa-

sionally there is pain referred along the course of the sciatic nerve.

Treatment.—The treatment is operative by the abdominal route. The Trendelenburg position allows easier exploration of the pelvis in following the loop of distended bowel to the site of obstruction. Reduction by gentle traction on the bowel and at times an even pressure over the buttock by the assistant brings about a satisfactory reduction. The sac may be grasped, pulled up into the pelvis, and removed. At times an approach by the sciatic route is used.

Lumbar Hernia or Petit's Hernia

In this hernia the protrusion takes place in the lumbar region, in the space between the 12th rib and the crest of the ilium, and through Petit's triangle. There are two types:

- 1 The congenital, which develops spontaneously as a diffuse bulge in this area.
- 2 The acquired, which occurs in adults and usually follows trauma or operations in this area.

The soft mass is easily reducible, and strangulation seldom occurs. Diagnosis is easy when a bulge develops in this location which presents an impulse on coughing and a resonant note on percussion.

Treatment.—This is usually conservative with the aid of a supporting abdominal belt. There are, however, certain cases in which the abdominal symptoms of dragging pain, nausea, and vomiting necessitate an operative repair.

Perineal Hernia

Perineal hernia is one in which the protrusion takes place through the muscles and fasciae of the pelvic outlet. It is most common in women 40-60 years of age. It can occur in males where the protrusion is into the ischio-rectal space. There is no definite symptomatology; the occurrence of a soft swelling in the region of the labia and ischio-rectal fossae should include this condition in the differential diagnosis.

Treatment.—The defect may be repaired by an abdominal route or at times by a combined perineal and abdominal approach.

Maydl's Strangulated Hernia

Maydl's strangulated hernia is not really a condition to be described as a rare hernia but represents a retrograde strangulation of intestine in an inguinal hernia. The condition is brought about by a loop of small intestine passing into a sac and then back again into the abdomen, in such a manner as to resemble the letter "W." All the loops may be strangulated, or only the loop returning into the abdomen may be obstructed. The signs and symptoms are those of a strangulated irreducible inguinal hernia, and the treatment follows that for the relief of strangulation.

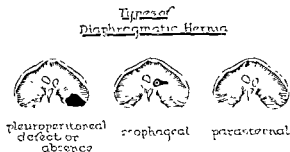
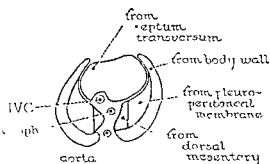
DIAPHRAGMATIC HERNIA

Definition.—A diaphragmatic hernia is one in which there is a protrusion of abdominal viscera into the chest through a normal or abnormal opening in the diaphragm.

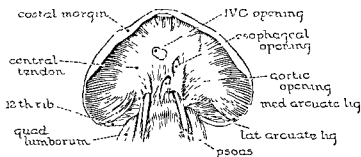
Classification.—The simplest classification is as follows.

- 1 Congenital (see Chapter 30)
- 2 Acquired
- 3 Traumatic

Embryologic Considerations.—A brief description of the embryologic features in the development of the diaphragm is necessary for a complete understanding of diaphragmatic hernia. The embryo in its early stages contains one large body cavity, the celomic cavity, which in time is separated into the two larger cavities, the pleural and abdominal, by the development of the diaphragm brought about by a fusion of the anterior and posterior portions derived from the ventral and dorsal muscle masses with the central tendon. If such a union fails to take place, an opening will remain on one or the other side, representing the pleuroperitoneal canal. This closure usually occurs in the 3rd month of intrauterine life. The size of these defects varies from small to very extensive openings. Indeed these openings may have a weak curtain of tissue separating the two body cavities, which in every respect represents a congenital weakness and allows one to explain why some hernias have a peritoneal sac while others have none. In other words the old saying that "all congenital diaphragmatic hernias have no sac" is not



Development of Diaphragm
(after Broman)



Anatomic Relations

Fig 374—Sites of diaphragmatic hernia

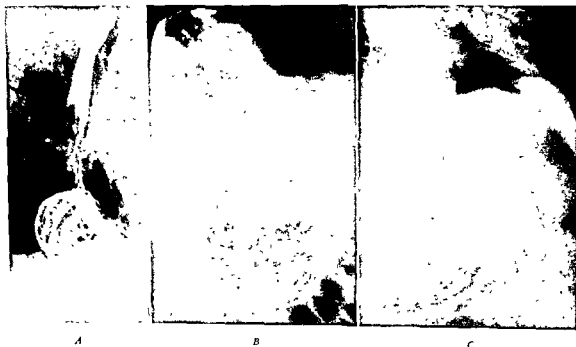


Fig 375—Hiatal hernia with nonfixation of the esophagus
A and B, Preoperatively. C, Postoperatively

entirely true. Occasionally congenital protrusions may occur through the normal openings in the diaphragm for the esophagus, aorta, or vena cava. See also Chapter 30

Acquired Types

The commonest type is the protrusion through the esophageal hiatus. Various authors have endeavored to classify this type into three groups. The classification depends upon the relationship of the esophagus and the posi-

the stomach remain in a fixed or normal position.

2. Hiatal hernia with nonfixation of the esophagus. The esophagus is tortuous and twisted by the upward displacement of the protruding cardiac portion of the stomach. This is the common type found especially in middle-aged women and gives rise to vague types of dyspepsia, especially after heavy meals or upon lifting or bending. Occasionally symptoms of dysphagia may develop later in the course of this condition

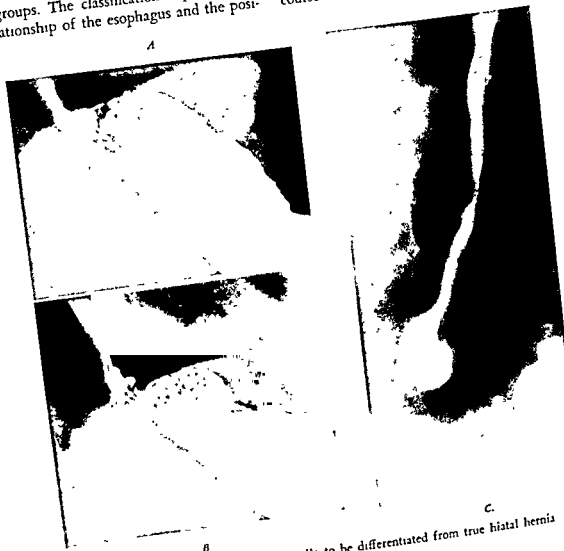


Fig. 376—A and B, Esophageal ampulla to be differentiated from true hiatal hernia
C, Hiatal hernia. Note protrusion shown above diaphragm

tion of its entrance into the cardia of the stomach

Three main groups have been described

1 Para-esophageal hiatal hernia, in which the esophagus is of normal length and its position and that of the cardiac portion of

3 The third type refers to that condition in which the esophagus appears shorter than usual and the stomach protrudes through the hiatal deficiency as a wedge, giving the appearance of having been sucked up into the chest

Symptomatology.—Small hiatal hernias are usually symptomless. The typical history of a moderately large hernia follows this pattern. A middle-aged, rather obese individual suffers from pain high up in the epigastrium especially after a heavy meal, with the pain referred to the back or across the precordium. There is regurgitation of sour or bitter-tasting material, especially upon bending to pick something up or to tie the shoes. Should this regurgitation persist, ulceration or esophagitis may develop with the persistence of the pain at the lower end of the sternum; indeed, if the ulceration is extensive, hematemesis may occur

establishes the diagnosis but emphasizes the frequency with which this condition in all its stages is found. Very occasionally the diagnosis may be suspected on auscultation of the chest when gurgling is heard.

Treatment.—Unless the condition is giving rise to symptoms no actual treatment is necessary. The vast majority of hernias found accidentally need not be touched surgically. Patients with mild symptoms are treated by diet, antispasmodics, and alkali to relieve gastric acidity, and propping up in bed. The avoidance of bending or lifting may bring about symptomatic relief



Fig 377—Traumatic diaphragmatic hernia

A, Simple posteroanterior x-ray to show bowel in left chest

B, Following barium enema Splenic flexure shown in left chest

(Courtesy Queen Mary Veterans' Hospital)

The differential diagnosis should exclude heart disease, cholecystitis, and peptic ulceration

Diagnosis.—The history may give the clinician the lead in establishing the diagnosis. X-ray examination with the screening of the patient in the recumbent position not only

When the hernia is large and respiratory distress or dysphagia is present, or ulceration has occurred with or without an attack of frank bleeding, or the persistence of retrosternal pain exists, surgical interference may be necessary. This procedure is usually carried out by the thoracic route, following a method described by Allison.

Traumatic Hernia

This type of diaphragmatic hernia is caused by a compression due to crushing injuries of the chest, by missiles such as bullets, or by knife wounds. Most traumatic hernias due to compression injuries occur in the dome of the left diaphragm. The right half of the diaphragm is rarely the site of a hernia because of the protection afforded it by the right lobe of the liver. The traumatic hernia has no sac. The viscera which protrude into the chest consist of the neighboring organs, usually the stomach, colon, spleen, and small bowel.

hernia apply. Symptomless hernia may not need treatment. On the other hand, large protrusions with gastrointestinal or respiratory symptoms require surgical intervention. This may be carried out by a thoracic or abdominal approach, or in some cases a combined approach may be necessary to bring about reduction and repair.

INTERNAL HERNIA

Definition.—An internal hernia is one in which there is protrusion into one of the peri-

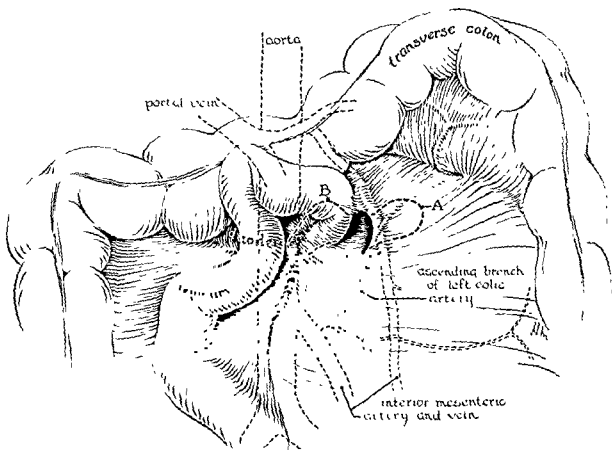


Fig. 378.—Paraduodenal fossae. A, Superior. B, Inferior.

Symptoms.—These are varied. Many such hernias are discovered accidentally by routine chest x-rays. At times symptoms of intestinal obstruction affecting one of the herniated loops in the chest may be the first indication of such a condition. At times severe dyspnea may follow a recent rupture of the diaphragm.

Treatment.—Here the rules governing the handling of other types of diaphragmatic

toneal fossae. This type of hernia is extremely rare and its presence is seldom diagnosed preoperatively. The condition is either found at autopsy or following laparotomy for intestinal obstruction.

There are several varieties of internal hernia. The classification follows the anatomic distribution in the abdominal cavity of various peritoneal fossae or pouches.

Classification.—

1. Paraduodenal
2. Pericecal
3. Intersigmoid

Internal hernias are most common in the neighborhood of the duodenojejunal flexure. In such cases the small bowel is invaginated into one of the inconstant peritoneal fossae found due to abnormal development in relation to the ligament of Treitz

Symptomatology.—A large number of cases do not give rise to any symptoms and are found accidentally on post-mortem examination. Usually the condition is found when laparotomy has been performed because of signs of an acute abdomen or the onset of intestinal obstruction.

Diagnosis.—The diagnosis may be suspected but is rarely made before operation. A large mass of distended loops of small intestine may present as a cystic swelling in the upper abdomen and percussion will give a tympanic note in such cases

Treatment.—Here the management depends on what the surgeon encounters at operation. Usually on opening the abdomen the absence of small intestine in the neighborhood leads one to suspect an internal hernia. By gentle traction the small bowel may be reduced and the opening in the mesentery closed with several interrupted sutures. Failing an easy reduction by traction, the neck of the sac may require gentle stretching to widen it. One must be extremely cautious and appreciate the dangers of incising the rim of the opening in the region of the duodenum because of the important vascular structures in the vicinity, i.e., inferior mesenteric artery and vein and the left colic artery. The viability of the bowel will determine what further procedures are necessary

guinal type. The complications, however, which may follow the injection of sclerosing fluids in the vicinity of delicate structures, such as the spermatic cord, have diminished the popularity of this method of treatment.

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INJECTION TREATMENT OF HERNIA

It was thought possible to cure hernia by the injection of a sclerosing solution into and around the neck of the sac, and successful obliteration of the sac has been reported in selected cases, particularly of the indirect in-

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Hernioplasty (An analysis of the basic pathology of direct and indirect inguinal hernia and appropriate surgical repair) (1953) (By Leo M Zimmerman, MD, Chicago)	33 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn
Repair of Inguinal Hernia The Surgical Residency Series (A residency teaching film illustrating repair of four different hernias) (1953) (By Francis D Moore, MD, and Staff, Boston)	42 min	Sound Color	Sturgis Grant Productions, Inc 322 E. 48th St New York 17, N. Y.
The Repair of Ventral Hernia (1956) (By Walter C MacKenzie, MD, Edmonton)	26 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn

Pediatric Surgery

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INTRODUCTION

The purpose of this section is to present the special considerations that must be given to the infant and child as a surgical patient. Pediatric surgery as an entity is relatively recent and indicates that the child does present a different surgical picture from the adult. The principles of general surgery apply to the treatment of the young patient. However, just as pediatrics, itself, has become a division of general medical care, it has been recognized that the child demonstrates surgical conditions and reactions to surgery which differ from the adult. With the increased interest in the surgery of childhood and the selected population of a children's hospital, more and more of the congenital anomalies previously considered rare are being recognized and treated successfully. Several of the surgical conditions of importance to the child have already been included in other sections of this text. Those that have been treated fully are omitted here.

Congenital anomalies constitute a portion of the interesting conditions that are peculiar to pediatric surgery. Certain of the anomalies still are gross aberrations of nature and cannot be salvaged to give a normal individual. These must be accepted as such. Some anomalies, such as atresia of the bowel, diaphragmatic hernia, or omphalocele, present as newborn surgical emergencies. If the condition is recognized and treated promptly, the child can survive to lead a normal, healthy

life. Other anomalies are compatible with life but represent a constitutional defect of varying degree. These are usually recognized early in infancy or may not become apparent until the child is older. When the diagnosis of a congenital anomaly is made, it is important to look for other coexistent anomalies.

The child also shows a number of acquired conditions which are predominantly found in the younger ages. These may be traumatic, infectious, or neoplastic.

THE CHILD AS A SURGICAL PATIENT

The general approach to the child as a patient presents a different problem from the adult. Patience and gentleness are particular essentials in the treatment of the child. Every effort must be made to obtain and preserve the confidence of the child. The parents can be of great help in this regard if they are aware of the treatments that the child requires; then if the child can comprehend, he should be told in simple terms that a procedure may be painful or that he is going to be put to sleep, but that he will be better when it is over. There can be no compromise in the execution of essential procedures. The history in most cases depends on the observations of the parents or nursing staff and must be carefully evaluated. Physical examination and treatment require the cooperation and coordination of the patient, parents, nursing staff, and surgeon.

Age

The age of the child per se is no contra-indication to operation. It is now common experience that major surgical procedures can be well tolerated in infancy if the child is adequately prepared, the surgery is done with care, and the postoperative care gives meticulous attention to the patient's requirements. In general, once surgery is indicated, specific treatment should be anticipated. Certain conditions are emergencies and must be treated as such. Other conditions may require delay until adequate growth has taken place. In some, a proper balance must be reached between the effect of the condition or defect on the child and an adequate growth of the part. This is seen in the treatment of cleft palate and cleft lip. It is also a factor in some of the skeletal deformities where osteotomy may be the considered form of treatment. Age may also affect the reaction of the child to the presence of an abnormality which sets him aside from other children. Psychic as well as physical reaction of the patient must be considered.

Prematurity may be a complicating factor in the surgical treatment of an infant, but again if the problem is understood it is no contra-indication to necessary surgery. These children are very labile in their reactions to changes in temperature, fluid, electrolyte balance, susceptibility to infection, and nutrition. Premature infants also pose a problem in the technical treatment. Surgery must be careful and gentle. Efforts at postoperative maintenance may be laborious and tedious.

Preoperative Preparations

The child must be prepared mentally and physically for operation. His natural fears and anxieties must be allayed. Parents and hospital staff can help considerably by presenting an unflurried and confident front. The child will have apprehensive questions. These can be answered simply and truthfully. Careful step-by-step explanations in terms that he can understand will be accepted. A child will cheerfully blow up a balloon although he may not otherwise tolerate an anesthetic mask suddenly thrust over his face.

Physically the child should be in optimum condition for surgery. For elective procedures, nutritional deficiencies should be checked and, if present, corrected. The hemoglobin should be 10.0 Gm. or more. Newborn infants should be given a prophylactic dose of vitamin K to counteract their bleeding tendency. Fluid and electrolyte balance should be normal. This is particularly important in infants as they can develop imbalance very rapidly. A history of diarrhea or vomiting is important in this regard. Physical signs of laxity of the skin with dryness and loss of turgor, sunken eyes and fontanelles, dry tongue, and the presence of acetone in the urine all point to dehydration. Persistent vomiting and abnormal respirations may indicate electrolyte imbalance. These must be corrected before surgery is performed. The child should have no feeding immediately prior to surgery. The last feeding should be given no less than 8 hours before operation. Clear fluids, such as 10% glucose and water, may be given up to 4 hours before operation. This is particularly important in infants to prevent depletion of their liver glycogen stores. In accident cases where the child has eaten just before the injury, it is best to delay anesthesia if possible 4-5 hours to decrease the danger of aspiration of stomach contents. If immediate surgery is necessary, gastric lavage and endotracheal intubation with inhalation anesthesia are indicated.

Premedication

Sedation is a valuable asset in handling the pediatric patient. Nembutal is a satisfactory barbiturate. Morphine and scopolamine are used in combination to produce a good degree of mental sedation, with reduction of mucous secretions, and a high level of analgesia. Demerol may be used in those cases where there is a known history of morphine sensitivity. Nembutal is administered orally or, if more convenient, the dose may be doubled and given rectally. For optimum effect the morphine and scopolamine are given subcutaneously 1 hour before operation. If necessary they may be given intramuscularly half an hour before operation or intravenously for an immediate effect.

A scale of recommended dosages for children, based on the average requirements for weight and age, is given in Table 25.

TABLE 25

PREMEDICATION

(Schedule Used at The Montreal Children's Hospital)

AGE	WEIGHT (KG)	NEM-BUTAL (MG) ORAL 1½ HR PREOP	MOR-PHINE (MG) 1 HR PREOP	SCO-POL-AMINE (MG) 1 HR PREOP
0-6 mo	3-7	~	~	0.10
6-12 mo	7-9	7.5	0.50	0.10
11-18 mo	9-11	7.5	0.55	0.10
18 mo - 2 yr	11-12	15	0.70	0.14
2-3 yr	12-14	15	1.00	0.14
3-5 yr	14-18	30	1.25	0.14
5-8 yr	18-25	30	1.65	0.21
8-10 yr	25-29	50	2.50	0.21
10-12 yr	29-36	65	5.00	0.32
12-14 yr	36-41	80	8.00	0.43

If it is wished to administer the Nembutal rectally the dosage may be doubled

Anesthesia

Some form of general anesthesia is most satisfactory in pediatric surgery, but the risks involved must be thoroughly understood. The small size of the patient is a technical disadvantage. The respiratory apparatus is not as well developed, and respiratory ventilation is not as efficient as in the adult. The small child does not tolerate as severe a degree of anoxia. Local anesthesia is a definite hazard in any major surgery as there is no control of the child's reactions and the child must be restrained forcibly. Spinal anesthesia, like local anesthesia, is unsatisfactory and requires very heavy sedation to control the child's apprehension.

Rectal administration of Avertin or Pentothal may be used to produce basal anesthesia. In general, however, this route does not give as certain control of the agent as does the inhalation method. It can be used to very good effect in the child who is excessively apprehensive or has been upset by a previous anesthetic experience. In children, inhalation agents are more easily controlled than intravenous agents and present less trouble in their administration. Open drop ether is the safest method and is considered advisable for those

who have only occasional experience in pediatric anesthesia. Endotracheal intubation has proved a very valuable adjunct for intrathoracic and intracranial procedures when administered by those with experience. However, the risks of endotracheal intubation must be kept in mind, i.e., hoarseness and laryngeal edema, laryngospasm, obstruction of the tube by kinking or secretions during operation, and unilateral intubation of a major bronchus. Temperature should be controlled during operation. Normal or just slightly below normal temperatures should be maintained for routine procedures. The young infant and premature child tolerate a slight cooling very well. More profound hypothermia has a use in some of the complicated cardiac and neurologic surgical procedures. During operation body temperature is best observed with a thermocouple placed in the rectum.

Fluid Replacement

Children, in particular infants, require close supervision of their normal fluid balance and the replacement of any deficits that occur. The margin of safety is much smaller in the child than in the adult for several reasons. Febrile reaction to comparatively minor insults is much more marked. The loss of fluids and electrolytes in diarrhea and vomiting can be fatal. Technically, the child presents a more difficult problem in the replacement and control of fluid balance. Normal requirements and deficits must be calculated and the deficits carefully replaced as overloading is as dangerous as inadequate hydration.

The normal daily requirements are calculated on the basis of age and weight and do not follow a straight line relationship. There is a gradual decrease in fluid requirements with age, and for children weighing over 45 kg, adult calculations are used.

TABLE 26
GENERAL REQUIREMENTS OF FLUID THERAPY
IN 24 HOURS

Fluid	100-300 ml/kg 100 ml/kg (newborn infant) 70 ml/kg (premature infant) 70-100 ml/kg (over 20 kg weight)
Saline	20 ml/kg (under 20 kg) 10 ml/kg (over 20 kg)
Blood	20 ml/kg

Milliliters per 24 hrs

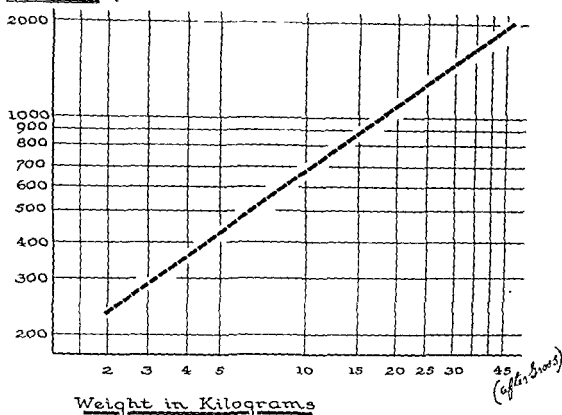


Fig 379—Basic daily requirements of fluid replacement in children (After Gross)

TABLE 27
COMPOSITION OF PARENTERAL FLUIDS FOR THERAPY

SOLUTION	CONCENTRATION IN mEq/L				LACTATE
	Na	K	Ca	Cl	
Plasma	137-146	13-50		95-108	
Normal saline (0.9%)	155			155	
1/2 M Na lactate	167				167
Darrow's solution	122	45		105	52
Ringer's solution	155	3	3	161	
Ringer's lactate	140	4	4	111	27
1 M KCl		1,000		1,000	
5% NaCl	510			510	
1/2 Solution	159			104	56
1/2 1/2 Solution	80			52	28
Solution for alkalosis	125	30		155	(160 ml. 5% glucose in water, 810 ml. 0.9% NaCl, 30 ml. 1 M KCl to make 1 L.)
Oral Fluids					
Orange juice	4	48	6	2	
Milk	27	41	59	28	
mEq Values					
1 Gm KCl—13.1 mEq of K or Cl	$\text{mEq/L} = \frac{10 \times \text{mg}/100 \text{ ml} \times \text{valence}}{\text{Atomic wt (or molecular wt)}}$				
1 Gm NaCl—17.1 mEq of Na or Cl					
1 Gm NaHCO ₃ —11.9 mEq of Na or HCO ₃					
1 Gm Na lactate—8.9 mEq of Na or lactate					
1 Gm CaCl ₂ —18 mEq of Ca (361 mg. of Ca)					
1 Gm Ca gluconate—4.4 mEq of Ca (89 mg. of Ca)					
1 Gm Ca lactate—6.1 mEq of Ca (130 mg. of Ca)					
Transfusion requirements					
Whole blood					10-20 ml/kg
Packed cells					10-20 ml/kg
Plasma					10-20 ml/kg
Exchange transfusion					65 ml/kg

The figures in Table 26 are maintenance requirements. Additional fluids and electrolytes must be given to replace losses due to intestinal drainage or suction, excessive perspiration, and increased loss due to fever. To arrive at a satisfactory assessment of fluid balance, a careful record must be kept of the normal fluid loss and of abnormal losses such as result from increased drainage. The child must be assessed clinically at frequent intervals to determine the state of hydration as evidenced by skin turgor, urinary excretion, peripheral edema, fullness of the fontanels, and evidence of cerebral and pulmonary edema.

TABLE 28
PARENTERAL FLUID THERAPY

Daily Maintenance Requirements (ml/Kg)				
Under 1 mo	1-6 mo	6 mo-2 yr	2-4 yr	10 yr
110	110	100	90	70
Composition of Body Fluids				
Total body water	70% of body wt			
Extracellular fluid				
Infants	25-30% of body wt			
Adults	20% of body wt			
Plasma volume	5% of body wt			
Intracellular fluid	45% of body wt			
Calculation of Fluid Losses				
Average Figures Only				
Insensible water loss	40 ml/kg/24 hr			
Urine loss	40 ml/kg/24 hr			
Gastrointestinal loss	30 ml/kg/24 hr			
Infants				
Mild dehydration	5% loss body wt.			
Moderate dehydration	10% loss body wt			
Severe dehydration	15-20% loss body wt			

Calculation of Rate of IV Flow

Drops/minute $\times 4 =$ ml/hour (approx)

(If shock is present, two-thirds of total 24 hour requirement may be given in the first 12 hours initially at 30-40 drops/minute)

Guide to IV. Potassium Therapy

Average Requirements (/Kg)

Mild depletion. 1 mEq/24 hr
Moderate depletion 2 mEq/24 hr
Severe depletion 3 mEq/24 hr.
(Do not use solutions with concentration of K exceeding 3.5 mEq/100 ml)

Guide to 1/6 M Na Lactate Requirements in Severe Acidosis

(e.g., CO_2 less than 10 mEq)

42 ml of 1/6 M Na lactate/kg body wt will raise the CO_2 combining power approximately 1 mEq/L.

(This is a crude calculation, included here as a safety measure. Usually no more than two-thirds of the calculated requirement should be given in a 24-hour period.)

Severe problems of electrolyte imbalance must be followed by chemical determinations which will guide the course of replacement.

Children in a normal state of balance will tolerate many procedures without added fluid therapy. The newborn infant has adequate fluid reserves for the first 48 hours of life. The premature infant does not require as much fluid therapy as a normal infant. In fact, after surgery it is safer to keep the premature and young infant in a slight degree of dehydration than incur the risks of overhydration. Long-term intravenous therapy poses the problem of caloric and protein balance. Blood, plasma or human albumin, and protein hydrolysates must then be considered as a source to help maintain homeostasis.

Techniques of Fluid Replacement

Hypodermoclysis.—This route is used to give small amounts of 5% glucose in normal saline. Up to 30 ml/kg may be given. Hyaluronidase (1 ml.) given with the infusion will increase the rate of absorption. The infusion is given by placing a needle in the subcutaneous tissues over the scapulae, the pectoral muscles, or the thighs. The disadvantages of this method are that the volume and type of fluid is definitely limited, and there is the possibility of local overhydration and ischemia.

Venoclysis.—This is the most satisfactory route of fluid replacement. Scalp veins can be used as a site of injection. With careful management these veins will serve satisfactorily for short-term therapy. In situations where therapy is to be prolonged, or must be rapid, or certain as in major surgical procedures, fluid is given through a polyethylene catheter tied into the long saphenous vein at the ankle or a vein in the antecubital fossa. In older children superficial veins in the legs or arms are of sufficient size to insert a needle directly.

Postoperative Care

Aspiration of secretions or vomitus is a very real hazard in the postoperative child. Careful attention must be paid to the posturing of the patient. Continuous or intermittent gastric suc-

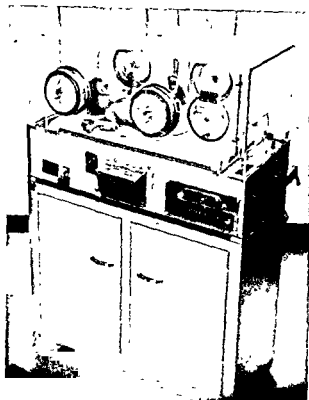
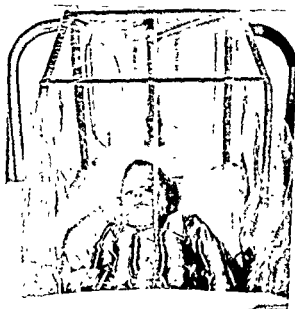
*A.**B.*

Fig 380—*A*, Isolette This apparatus allows easy observation and access to an infant and maintains a desired constant oxygen content, temperature and humidity. The infant is also protected by the easy maintenance of an isolation technique.

B, Oxyhumidity hood This is used for older children and maintains a desired atmosphere of oxygen and humidity.

*A.**B.*

Fig 381—*A*, Type of restraint jacket which is tied to the bed. The arms are splinted at the elbows, but there is considerable freedom allowed. The leg is immobilized with a padded splint.

B, Glove hitch with a single tie over the padded extremity as a form of restraint for children.

tion may be necessary. A Levin tube may be introduced into the stomach or a catheter introduced at frequent intervals. Endotracheal suction is sometimes required. *Constant nursing supervision is a necessity*

The infant also requires an atmosphere controlled for temperature, humidity, and oxygen. This is best facilitated by the use of the Isolette. It has the added advantages of an isolation technique, and the child may be exposed for constant observation and daily weight estimation. Nasal oxygen should be avoided in small children because of the danger of gastric distention. Older children requiring oxygen or humidity may be placed in oxyhumidity hoods

When the limbs must be tied down, a non-constricting, padded clove-hitch knot with an added single tie should be used. *Padded splints are used to restrain the arm or leg for intravenous therapy.*

Clear fluids should always be offered as the first postoperative feeding, and in nonintestinal cases these are started within 4-8 hours. In infants, when such fluids are tolerated, they are alternated with skim milk, and feedings progress to formula and diet for age. Older children graduate to a normal diet as tolerated. Gavage feeding is used in infants that are too weak to suck but will tolerate alimentary feedings. Older children may require tube feeding

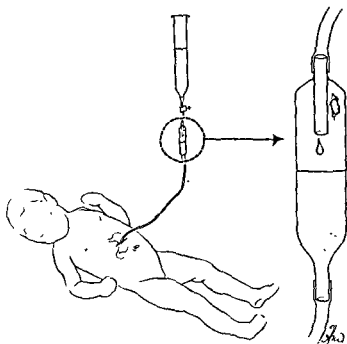


Fig. 382—Modified Murphy drip apparatus for gastrostomy or jejunostomy feedings.

Dressings should not be cumbersome or constricting. In most instances they can be dispensed with shortly after operation. The problem of soiling of wounds can be dealt with more efficiently if the wound is exposed, cleansed, and dried as necessary. Restraints may be required to prevent the child from damaging a surgical repair or disrupting intravenous therapy. Mild restraints, such as a jacket which can be tied to the bed or one in which the sleeves are splinted, are sufficient in some cases

as a means of ensuring an adequate intake. Gastrostomy or jejunostomy is indicated in the postoperative care of certain surgical conditions of the esophagus, stomach, and duodenum. These are administered slowly by means of a continuous drip. If the gastrointestinal tract is overloaded, the apparatus allows regurgitation to occur into an open Murphy drip chamber rather than up the proximal esophagus. Daily vitamin requirements are given by oral or intravenous route.

HEAD AND NECK

The majority of the conditions in the head and neck have been discussed in Chapters 10 and 12

CONDITIONS FOUND IN CHILDHOOD

Congenital anomalies

- 1 Choanal atresia
- 2 Cleft lip and palate
- 3 Micrognathus
- 4 Branchial cleft cyst and sinus
- 5 Midline cervical cleft
- 6 Fistula auris congenita
- 7 Dermoid
- 8 Thyroglossal duct cyst
- 9 Cystic hygroma

Infections

- 1 Stomatitis
- 2 Parotitis
- 3 Cervical adenitis
- 4 Cellulitis of the neck

Thyroid gland See Chapter 13

Choanal Atresia.—In this congenital anomaly the posterior nares remain closed. The infant is unable to breathe through the nose, and because of this respiratory obstruction, bottle or breast feeding is impossible. If the posterior nares are only narrowed, a small catheter is passed through the nose into the posterior nasopharynx to ensure a clear airway. In complete obstruction a tracheostomy is required. The child can then be maintained to an older age, when a definitive plastic repair of the posterior nares can be performed.

Micrognathus.—Micrognathus or retrusion of the mandible can be a hazard of the newborn infant. The mandible is underdeveloped, and the chin is markedly recessed. As a result, the pharynx is reduced in size and the tongue causes obstruction of the airway. Immediate treatment consists of pulling the tongue forward and posturing the child with the head in the hyperextended position. The tongue can then be fixed in a forward position by sutures through the chin. Aggravated cases require a tracheostomy. Once a clear airway has been ensured, plastic procedures can be considered at a later date to correct the deformity of the mandible.

Midline Cervical Sinus.—This represents an abnormality in the junction of the branchial arches in the midline anteriorly. It presents as



Fig. 383.—Midline cervical sinus showing the vertical fusion defect

a weeping, vertical cleft and may extend from the suprasternal notch to the level of the hyoid bone. It must be differentiated from sinuses of the branchial clefts which are situated laterally and from midline sinuses communicating with thyroglossal duct cysts. Treatment is excision, and the best cosmetic result is obtained with a Z-plasty incision.

THORAX

The Breast

Congenital anomalies are rare and have been discussed elsewhere (see Chapter 14). The common condition in infancy is that of *breast abscess formation in the neonatal period*. The infant breast responds to the hormonal stimulation of the mother, hypertrophies, and becomes engorged during the first 2 weeks of life. This state normally subsides, but a staphylococcal infection with abscess formation may occur during this period and may be localized to the breast or spread to the pectoral fascial planes and the anterior pectoral nodes. Treatment consists of warm saline compresses, antibiotics, and drainage when the abscess localizes. A radial incision is made over the localization adjacent to the nipple. This leaves a minimal scar and does not interfere with later breast development and continuity of the ducts.

Anomalies of the Thoracic Cage

These are encountered in infancy and childhood. Isolated anomalies of the ribs are usu-



Fig 384—Breast abscess in the newborn period

ally insignificant, although the presence of a cervical rib may become a problem in the adult. Pseudarthrosis of the first rib may be found on radiologic examination and must not be confused with a possible fracture.

Complete or partial defects of the sternum allow herniation of the heart and abdominal contents, a condition known as *ectopia cordis*. Major defects of this nature are usually associated with marked cardiac anomalies. Plastic procedures have been devised for the correction of the anomaly.

The thoracic cage may develop abnormally to produce a deformity in which the sternum is displaced either dorsally or ventrally. The etiology is obscure, but the condition is congenital, familial, and progressive. Dorsal displacement of the sternum, most marked in the lower portion, associated with prominence of the costochondral junction and indrawing of the costal cartilages is known as *pectus excavatum* or *funnel chest*. The reverse deformity, with ventral displacement of the lower portion of the sternum and a deep bilateral sulcus of the costal cartilages, is known as *pectus carinatum* or *pigeon breast*. These deformities can be corrected by reconstruction of the anterior thoracic cage. Treatment in infancy is a minor procedure. If delayed, treatment should wait until the thoracic cage is stabilized. This is done by several methods

Pectus Excavatum.—

Infants Under One Year.—

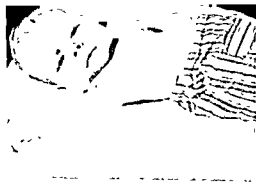
1. Retrosternal division of the attachment of the diaphragm
2. Wire traction of the sternum with or without osteotomy

Children Over 5 Years.—

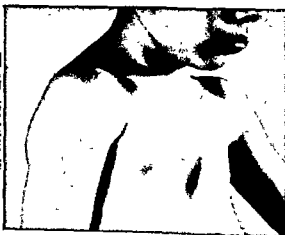
1. Wedge resection of the costal cartilages with osteotomy and elevation of the sternum
2. Subperichondral resection of the deformed costal cartilages and elevation of the sternum

Indications for Surgery.—

1. Cosmetic
2. Psychic



A.



B.

Fig 385—Pectus excavatum showing depression of the lower end of the sternum
A, In an infant B, In an older child

3. Rapid progression of the deformity
4. Cardiac compression, paroxysmal auricular tachycardia, and recurrent pulmonary infection

Satisfactory results are more consistently found with the more radical types of procedure and after the ribs have become fixed

Pectus Carinatum.—This is not as common a lesion but will respond to a similar type of surgical approach if indicated



Fig 386—X-ray of pectus excavatum showing the posterior deformity and angulation of the lower sternum. Barium outlines the deformity at the skin

Larynx, Trachea, and Bronchi

The branches of the pulmonary tree are involved in a variety of conditions, the importance of which is due to possible obstruction to the free passage of air or the accumulation of secretions. Obstruction may be congenital, infectious, or due to tumor growth and may occur within the airway proper or by compression from without. The most frequently encountered anomaly is *tracheo-esophageal fistula* which is usually associated with esophageal atresia and will be discussed with the anomalies of the esophagus

Laryngeal stenosis is an anomaly which can cause obstruction of the airway due to a definite narrowing of the lumen at the level of the vocal cords. It causes a marked inspiratory

stridor. These children must be carried on prophylactic tracheostomy to ensure an adequate airway until the larynx has grown sufficient size that a plastic repair may be carried out. Narrowing of the trachea and bronchi is rare. Abnormalities in the distribution of the bronchi are usually associated with agenesis of the adjoining areas of lung. Infants may show a condition known as *laryngo-tracheomalacia* in which the normal rigidity of the trachea has not yet developed. Radiologic examination of these infants may give the impression that there is a narrowing of the trachea. Fluoroscopic examination will show an inspiratory collapse of the trachea and not a fixed narrowing of the larynx and trachea

Congenital anomalies outside the trachea, such as vascular rings, or aberrant vessels must also be considered as causes of tracheal compression (See Chapter 16)

Following are the two common causes of tracheal obstruction in childhood:

Acute Laryngotracheobronchitis.—This is an acute infection which is sudden in onset and may progress rapidly to complete respiratory obstruction. Clinically it is known as *croup* because of the characteristic inspiratory crowing with indrawing of the suprasternal areas and the lower costal margin. There is marked edema of the larynx and accumulation of secretions in the obstructed trachea and bronchi. Tracheostomy, when indicated, must be supported by antibiotics, oxygen, and high humidity.

Aspiration of Foreign Bodies.—Children are very prone to the aspiration of foreign bodies into the trachea and major bronchi. These may be small objects that are picked up and carried in the mouth, or they may be solid forms of food. The common offenders are peanuts and pieces of carrot. There may be distress at the initial time of the accident, and then the child may be comparatively symptom-free. X-ray examination will show the specific foreign body if it is radiopaque. If it is not radiopaque, the diagnostic sign may be the emphysema that develops in the affected portion of lung due to a ball-valve type of obstruction in a major bronchus. Bronchoscopic examination for diagnosis and removal of the foreign body, if present, is indicated. If the

A

B.



C.

Fig 387—Staphylococcal pneumonia in infancy

A, Bronchopneumonia of the left lung with empyema

B, Pyopneumothorax 24 hours later

C, Closed intercostal drainage established with expansion of the lung and drainage of the empyema

foreign body is unrecognized, the late complications of pulmonary collapse, abscess formation, and bronchiectasis will occur.

Lungs

Congenital lesions vary from abnormalities of lobar segmentation to complete agenesis of one lung or partial agenesis of various lobes. In unilateral agenesis there is overexpansion of the normal lung with marked displacement of the mediastinum and heart. The degree of embarrassment of the venous return to the heart and respiratory exchange may be fatal. Isolated or multiple *congenital cysts* may occur, and these represent abnormal formation in the lungs. Some may communicate with a bronchus, and if there is a flap-valve mechanism present, they may expand to enormous size.

Mucoviscidosis is a congenital defect in the enzyme mechanism of the mucus-secreting glands. The bronchial secretions are thick and tenacious and accumulate in the pulmonary tree. As a result these children are subject to repeated bouts of respiratory infections and usually succumb to a final attack of pneumonia.

Congenital atelectasis is seen in the newborn infant. Areas of lung may fail to expand due to obstruction by secretions or aspiration of fluids during birth. Careful suctioning of the trachea after birth will prevent this complication. If left untreated pneumonia will ensue.

Pneumothorax is seen as a spontaneous result of the rupture of a pleural bleb from interstitial emphysema, or may be the result of overenthusiastic resuscitation. If respiratory distress indicates an expanding pneumothorax, it must be aspirated immediately. Persistent tension pneumothorax must be controlled by an intercostal catheter connected to an underwater drainage system.

Acute Infection of the Lungs and Pleura.—Infants are very susceptible to staphylococcal infection of these tissues. The pathologic process is one of bronchopneumonia and/or septicemia, with the formation of localized abscesses throughout the parenchyma. The onset and progress of the disease may be very rapid and dramatic. Infection may dominate the picture, with evidence of septicemia. Pleural

effusion may occur as a reaction to the underlying lung infection, and if this effusion becomes infected, an empyema develops. Rupture of an abscess through the pleura allows the formation of a bronchopleural fistula and a pyopneumothorax. The accumulation of air may lead to a tension pyopneumothorax. Here the mechanical upset in the function of the mediastinal structures rather than the infection may kill the child. These children must be treated strenuously with antibiotics and watched for the sudden onset of increasing pulse rate, dyspnea, and cyanosis which indicate possible development of tension pyopneumothorax. This must be confirmed by immediate x-ray examination. A pyopneumothorax must be controlled by intercostal drainage to an underwater seal (See Fig. 224). Any infant with an empyema should have a thoracentesis. If smear of the fluid demonstrates the presence of gram-positive cocci, a prophylactic intercostal drainage should be performed. This is a safeguard against the development of a tension pyopneumothorax and also allows the drainage of infected material from the pleural space. Loculated areas of pus may develop as a later complication. As the lung is then adherent to the thoracic cage, local aspiration is effective. In the infant it is seldom necessary to do an open drainage of the chest. Another late complication may be the development of large bullae or pneumatoceles which must be followed carefully by frequent radiologic studies; these usually subside.

GASTROINTESTINAL SURGERY

ESOPHAGUS

Introduction

While the general principles of abdominal surgery provide the basis for the definitive corrective procedures carried out on the gastrointestinal tract in infants and children, it should be pointed out that meticulous attention must be given to the supportive therapy in the preoperative, operative, and postoperative care of these patients. The three principles of early diagnosis, adequate supportive therapy through out, and the application of planned and carefully carried out corrective surgical procedures play an important role in the lowering of

morbidity and mortality. The associated anomalies that may appear as complications of the presenting condition must be sought, and as most of these conditions are only relative emergencies, the time taken to obtain a comprehensive preoperative picture in any infant is well worth while. On the list of acute abdominal emergencies admitted to the surgical service of a children's hospital, the irreducible inguinal hernia takes first position and is followed by cases of acute appendicitis and intussusception.

Congenital Atresia of the Esophagus With and Without Tracheo-esophageal Fistula

The Anomaly.—In the 4-5 mm. human embryo, the esophagus first appears as a short

canalization, accompanied by its elongation, gives rise to the normal esophagus which joins hypopharynx to the stomach. Arrest in development or aberration from a normal development gives rise to five anomalies in this region and four of these five types have as the prime anomaly an atresia of the esophagus. The first and most common is one in which there is a blind upper esophageal pouch and a fistula between the distal esophageal segment and the posterior aspect of the trachea, usually just above the carina. This type of anomaly is present in about 88% of cases. The second type, which occurs in about 5% of cases, comprises a blind upper esophageal pouch and a blind lower esophageal segment without a tracheo-esophageal fistula being present. In about 3% of cases there is a congenital fistula

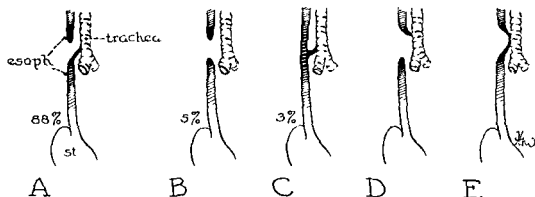


Fig. 388—Congenital atresia of the esophagus with or without tracheo-esophageal fistula

A, Esophageal atresia with a blind upper segment and the lower segment communicating with the trachea.

B, Esophageal atresia without tracheo-esophageal fistula

C, Tracheo-esophageal fistula without esophageal atresia

D, Esophageal atresia with a blind lower segment and the upper segment communicating with the trachea.

E, Esophageal atresia with both segments communicating with the trachea

ring of tissue which separates the hypopharynx from the stomach, and prior to its elongation, the developing trachea and esophagus comprise a single lumen tube. This single lumen becomes divided into two lumina by the ingrowth of two lateral septa which fuse to form the trachea ventrally and the esophagus dorsally. The last part of the septum to be fused is the most caudal portion in the region of the carina. Conversion of the esophagus to a solid rod of cells and its subsequent re-

between the esophagus and the trachea, but no esophageal atresia is present. The remaining two types, which are exceptionally rare, are, first, an upper esophageal component that communicates with the posterior aspect of the trachea and is accompanied by a blind lower esophageal pouch, and, second, an upper and lower esophageal segment that communicate with the posterior aspect of the trachea.

Complicating Factors.—Many of these infants are premature, and this appears to be

a serious complication to satisfactory treatment. Many of these infants have major associated congenital anomalies of the heart, the gastrointestinal system, the genitourinary tract, and the central nervous system. Delay in the diagnosis and persistent attempts at feeding these infants give rise to pulmonary complications. Aspiration pneumonia, characteristically located in the right upper lobe, is the common finding. If a fistula is present between the lower esophageal segment and the posterior aspect of the trachea, the regurgitation of acid chyme into the tracheobronchial tree gives rise to a very formidable type of pneumonic process.

aroused, an attempt to pass a nasogastric tube should be made. One must be aware of the possibility that the catheter which has been introduced into the blind upper pouch may curl on itself and give the impression that it has entered the stomach. The confirmation of the diagnosis is obtained by the injection of less than 1 ml of Lipiodol down the catheter and the visualization under fluoroscopy of the blind upper pouch. The intestinal tract should be carefully visualized for the presence of gas within its lumen, and if none is present no fistula exists between the trachea and the distal esophagus. However, the diagnosis of a tracheo-esophageal fistula *without* esophageal



FIG. 389.—Esophageal atresia with tracheo-esophageal fistula. X-ray examination with radiopaque medium filling the proximal blind pouch of esophagus. Gas in the abdomen indicates that there is a communicating fistula between the trachea and lower esophagus. There is evidence of atelectasis in the right upper lobe (arrows).

Diagnosis.—This should be relatively simple to make if the condition is kept in mind. In the newborn infant the presence of excessive salivation and the inability to take feedings should immediately suggest the possibility of this defect. Once the suspicion has been

atresia is one that is fraught with difficulties and indeed may not be made before operative intervention reveals the fistula. In some cases the tracheo-esophageal fistula will be evident when Lipiodol is put down the lumen of the esophagus and appears in the tracheobronchial

tree. In other cases, one may have to resort to bronchoscopy in order to establish a diagnosis, and this may be facilitated by the placing of methylene blue in the esophagus, which may be seen to appear in the trachea.

Treatment.—The early diagnosis of the condition as outlined is of prime importance. The infant should be transported in a semi-upright position, which facilitates the removal of the excess saliva from the blind upper pouch and tends to diminish the amount of regurgitation of acid chyme into the tracheo-bronchial tree. While early definitive treatment is important, a few hours' delay to give adequate supportive preoperative care and initiate therapy for the pneumonic process which may be present is time well spent. The greatest advance has been the acceptance of the fact that the continuity of the esophagus should be established and the fistula, if present, tied off in a one-stage operation. The surgical procedure of choice is a right-sided thoracotomy through the 4th interspace and a transpleural approach to the esophagus. The azygos vein is isolated and cut between ligatures. The mediastinal pleura is incised longitudinally, and the upper esophageal pouch is then isolated and dissected free as far cephalad as possible. There is little reason for concern about the blood supply of this proximal segment. The tracheo-esophageal fistula is then identified, clamped, and amputated with an adequate cuff on the tracheal side to allow closure. End-to-end anastomosis of the esophageal segments may be accomplished at this time, but the greatest problem at this stage is the distance separating the two segments of esophagus. Every effort must be made to effect an anastomosis without tension. If the lower esophageal segment appears to be short and extensive dissection is required, it is better to bring the stomach up into the thorax to assure a tension-free anastomosis, and if the precarious blood supply to the lower esophageal segment appears to be inadequate, then primary anastomosis between the upper esophageal segment and the stomach may be indicated. Two methods of anastomosis are recognized. The method proposed by Haight is an end-to-end anastomosis, where all layers of the distal segments are sutured to the mucosa of the upper segment, and the cuff of

muscularis of the upper segment is brought down to cover this anastomotic line. The other method is an end-to-end anastomosis using a single layer of interrupted fine black silk sutures, each stitch of which includes all layers of both segments.

Postoperative Care.—Early postoperative feeding of these infants is of prime importance, and this may be carried out by means of a gastrostomy feeding regime. The gastrostomy may be instituted at the time of the primary operative procedure or done under local anesthesia 24-48 hours later. The gastrostomy serves a dual purpose, for in many of these infants where postoperative esophageal stricture occurs, a retrograde bouginage can be carried out through the gastrostomy on an outpatient basis without anesthesia. Systemic antibiotics should be administered and periodic posterior pharyngeal suction carried out. Gastrostomy feedings can be started in 48 hours. As posterior pharyngeal suction produces less and less, and it is obvious that the infant is swallowing saliva, oral feedings may be undertaken at about the 10th postoperative day when the restitution to integrity of the esophageal segments has been accomplished. At about 3 weeks the esophagus should be examined with Lipiodol and preventive or corrective measures taken to eliminate any serious esophageal stricture at the site of the anastomosis, if this is demonstrated. Periodic follow-up examinations should be carried out for the first 6 months of life, and then at less frequent intervals thereafter. The general principles of therapy in the patient with esophageal atresia with tracheo-esophageal fistula apply to the other anomalies of the esophagus.

Results of Therapy.—The results of therapy would seem to depend primarily on the rapidity with which a diagnosis is made and the precautionary care given before the arrival of the infant for surgery. Prematurity, which occurs in about one in four of these infants, presents a formidable challenge and is accompanied by a high mortality rate; it is a possible indication for a staged procedure. The severe associated congenital anomalies play their role in raising the mortality statistics. In competent hands, it would appear that the mortality rate, including all complicating fac-

from left to right and a palpable pyloric mass which is most readily felt in the right upper quadrant at the lateral border of the rectus. Where there has been great dilatation and hypertrophy of the stomach itself, the tumor mass may be felt lower down on the right lateral side of the abdomen. In unusual or atypical cases, confirmation of the diagnosis may be obtained by the use of a barium swallow, during which a persistently stenosed and elongated pyloric canal is visualized. Nutritional edema is not present in these cases



Fig 391.—Congenital hypertrophic pyloric stenosis. Barium meal outlines the narrowed pyloric canal (arrows). The pyloric tumor projects into the duodenum causing a mushroomlike cap.

Differential Diagnosis—Pyloric stenosis must be differentiated from pylorospasm in which the symptoms vary from day to day, and relief is obtained by the administration of phenobarbital and atropine. Other types of intestinal obstruction of congenital origin usually give rise to vomiting in the first or second day of life and these can be differentiated from pyloric stenosis where vomiting rarely occurs before the first week or 10 days of life. In the intestinal obstructions related to malrotation of the gut, with volvulus or the presence of peritoneal bands, the vomitus usually contains bile. Poor feeding patterns instituted by the parent can produce vomiting which usually responds to correction in the feeding regime.

Preoperative Care.—While the performance of a pyloromyotomy is of prime importance in the correction of this condition, the preoperative preparation of the patient for surgery must be dealt with carefully. This condition is rarely a surgical emergency, and, indeed, the more precarious the baby's condition, the longer the operation should be delayed. Replacement of fluid by intravenous therapy, in particular the administration of chloride and potassium, must take precedence over the operative procedure. Blood transfusion preoperatively is rarely needed, but where the hemoglobin is low or the plasma proteins appear to be decreased, blood or plasma may be given in the amounts of 20 ml./kg. body weight. Feedings may be continued during the preoperative period but should consist mainly of clear fluids comprising glucose, water, and normal saline. Adequate wound healing is sometimes a problem in these debilitated infants, and the administration of large doses of vitamin C and the correction of low plasma protein levels should be encouraged.

Operation.—The surgical approach to the hypertrophied pylorus may be either through (1) a right upper rectus-splitting incision or (2) a transverse subcostal incision. The latter is preferred. The subcostal incision is made one fingerbreadth below the right costal margin and deepened so that the transversus abdominis muscle and peritoneum are entered as one layer. The opening and closing of the peritoneal cavity are facilitated by making the incision over the liver. The liver is retracted upward and the pylorus delivered through the incision. The blood supply to the pylorus is seen to come from the upper and lower surfaces, and an avascular area between the termination of these two blood supplies is chosen for the incision. One third of the muscle mass is incised with a scalpel in the long axis of the pylorus, then by means of a hemostat the circular muscle fibers are widely separated until the submucosa bulges to the level of the serosa. The utmost care must be taken in the separation of these muscle fibers distally to avoid penetration into the duodenum, and an adequate incision must be made on the antrum of the stomach if uniformly good postoperative results are to be obtained. There is usually

little trouble with bleeding, and after the pylorus has been replaced in the peritoneal cavity the stomach is swept up into the left upper quadrant of the abdomen. In these malnourished infants healing is poor and closure of the abdomen must be meticulously accomplished.

Postoperative Care.—In most cases feeding may be begun 4-6 hours postoperatively, and

the first few offered feedings should be glucose and water. When these are tolerated, the appropriate formula can be administered, and the only important qualification is that it should not be offered in large amounts. Small, frequent feedings are better tolerated for the first few days, and continuation of intravenous therapy should be carried out until adequate amounts of formula are being taken by mouth.

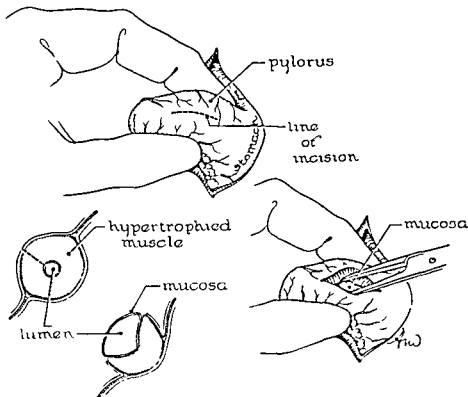


Fig 392.—Fredet Ramstedt operation for hypertrophic pyloric stenosis

DUODENUM AND SMALL INTESTINE

Congenital Atresia of the Intestinal Tract

This includes atretic areas from the first portion of the duodenum, through the jejunum and ileum, and on into the colon. Atresia, by definition, is the absence of a lumen. This may take the form of an internal diaphragm, or there may be discontinuity of the bowel with the proximal portion ending in a blind sac. The accepted embryologic theory for the development of these atretic areas is that during the 2nd or 3rd month of intrauterine life a failure of recanalization of the rapidly proliferating epithelium occurs and gives rise to an atresia. However, if recanalization takes place and there is a residual narrowing of the lumen, congenital stenosis is the result. Atresia may occur singly in the intestinal tract or in

conjunction with other atretic areas elsewhere. The ileum is the most common site for this abnormality of development, with the duodenum in second place. Ileal atresia is twice as common as duodenal atresia, and duodenal is about twice as common as jejunal atresia. The isolated colonic atresia is an extremely rare lesion. The gross picture of the intestinal tract in atresia is a striking one. Proximal to the level of obstruction the bowel is greatly dilated and may measure 4-5 cm. in diameter, whereas distal to the obstruction the bowel may measure 1 cm. in diameter. With the distention of the proximal portion of the intestinal tract there is concomitant thinning of the bowel wall, and marked ischemia may be present. Indeed, perforation often occurs. Associated with atresia of the small intestine there is always present an "unused colon,"

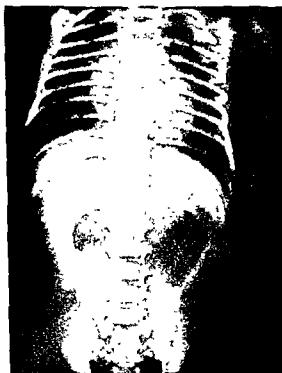


Fig 393

Fig 393—Duodenal atresia. Plain film of the abdomen showing marked distention of the stomach and duodenum and no gas in the distal bowel.



Fig 394

Fig 394—Ileal atresia. Plain film of the abdomen showing distended loops of proximal bowel and no gas in the distal colon or rectum.

which has been designated by some as a micro-colon. This distal colon, however, is normal in all respects and may be dilated to normal size and function with little difficulty. The absence of stratified squamous epithelial cells in the meconium of a newborn baby (Farber's test) is a diagnostic aid in cases of intestinal atresia, that is, the ingested vernix caseosa which is suspended in the amniotic fluid cannot pass an atretic area in the intestinal tract. While it is true that obstructions of the large bowel are better tolerated than those of the small bowel and those of the low small bowel better than those of high small bowel, nevertheless all these babies will show symptoms on the first day of life since this intrinsic obstruction of the intestinal tract has been present for such a long period of time. With the exception of the rare atresia of the duodenum which is proximal to the entrance of the ampulla of Vater, the vomiting of all these infants will be bile-stained, and this finding is one of the signs that necessitate the utmost urgency in diagnosis and treatment. The gross appearance of the meconium in these infants may be misleading, and the amount of abdominal distention present may depend on the level of the atresia and the adequacy of the decompression of the proximal bowel by the vomiting. Diagnosis may be confirmed by x-ray examination of the abdomen and the ingestion of radiopaque material. Barium is not recommended because of the dangers of regurgitation and aspiration and the inspissation of the barium in the intestinal tract. However, an enema in these infants, using Lipiodol, is of benefit in that a malrotation of the bowel can be ruled out and the presence of an "unused colon" visualized.

Choice of Operative Procedure.—A long right rectus incision centered on the umbilicus is best, since it gives ready access to the whole gastrointestinal tract. For the atretic area which is diaphragmatic in nature, excision of the segment of bowel containing the diaphragm, with anastomosis, is the procedure of choice. If this is difficult, an enterocenterostomy should be performed to circumvent the obstruction. For the duodenal atresia below the ampulla of Vater and for upper jejunal atresias, duodenoduodenostomy is recommended. Gastrojejunostomy should be avoided and is perhaps indi-

cated only in those lesions that are above the level of the ampulla of Vater. For the other atretic areas in the intestinal tract, three approaches may be considered: (1) a long side-to-side isoperistaltic anastomosis, (2) a double-barreled Mickulicz type of enterostomy, which after 4-5 days may have the spur crushed with a specially designed crushing clamp, or (3) an end-to-end anastomosis, where possible, after decompression of the proximally distended loop and excision of the proximally dilated and atonic portion of the proximal bowel and distention of the distal collapsed portion of the intestine with normal saline solution, air, or mineral oil. A one-layered anastomosis of interrupted Connell type sutures with No 5-0 silk on an atraumatic needle is preferred.

Congenital Stenosis of the Intestinal Tract

While the embryology of a congenital stenosis of the intestinal tract is similar to that of an atresia, as mentioned above, stenosis is in fact a separate entity. In this connection, the stenoses of the duodenum are twice as common as those of the ileum. As in the case of an intestinal atresia, an isolated stenotic area in the colon is rare, and multiple areas of stenosis in the intestinal tract are also not commonly found. There is a compromise of the lumen of the intestine. A diaphragm or tissue may remain in which there is a small perforation that measures 2-3 mm. in diameter. In other cases the aperture may be larger, and there may be only slight compromise to the lumen. Proximal to each area of narrowing there is dilatation of the bowel, and if the obstruction is of sufficient duration there may be hypertrophy as well. If the lumen is extremely narrowed dilatation is great, but in contradistinction to atresia, perforation of the intestine occurs uncommonly proximal to the stenotic area. The age of onset of symptoms varies with the severity of the stenosis and, when severe, may occur in the first week of life. In the marked forms of intestinal stenoses the symptoms may be indistinguishable from those of intestinal atresia, whereas in the milder cases the symptoms may be only occasional vomiting, intermittent abdominal pain,

3 A normally rotated intestine where the cecum and ascending colon are unattached posteriorly and are excessively mobile

Complications which can arise as a result of these arrests in rotation are as follows:

1. Duodenal obstruction
2. Volvulus of the midgut
3. Reversed rotation
4. Internal hernia

Volvulus of the Midgut

With nonrotation of the intestine, a condition exists in which a long portion of the intestinal tract is suspended from a mesentery with an extremely short base. Under these conditions the entire length of the midgut may rotate in a clockwise manner about the axis of the superior mesenteric artery. When the midgut has rotated through two or more

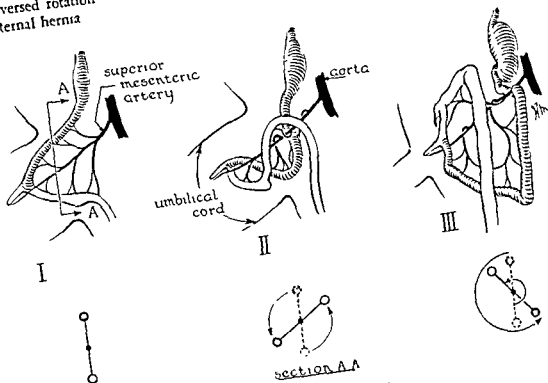


Fig 395—Rotation of the gut.

Duodenal Obstruction

When the cecum lies in close proximity to the duodenum it may be fixed in this position by peritoneal bands which arise in the region of the cecum or ascending colon and insert into the posterior abdominal parietes to the right of and below the third portion of the duodenum. Pressure of the cecum, itself, or of these posterior peritoneal bands, gives rise to duodenal obstruction which is usually high grade but incomplete. Great dilatation of the stomach and proximal duodenum occurs and this is associated with bile-stained vomitus. This condition is easily remedied by cutting the band or freeing the cecum and allowing it to attain a position in the left upper quadrant of the abdomen, where it should be allowed to remain.

right angles about this axis, congenital volvulus exists. The pathologic principles apply here as to volvulus elsewhere. Here we have a closed loop, with the obstruction lying proximally at the region of the duodenojejunal junction and distally somewhere in the region of the middle of the transverse colon. Because the volvulus occurs around the origin of the superior mesenteric artery and vein, venous engorgement, edema, and infarction of the whole of the midgut may be imminent. Volvulus of the midgut gives signs and symptoms of a high small bowel obstruction, with vomitus which is bile-stained, dehydration, and electrolyte imbalance. The amount of abdominal distention is variable. It may be limited to the epigastrium or it may be generalized, depending on whether or not gas has entered

or is forming within the involved loop Barium enema will show the cecum in the epigastrium or the right upper quadrant of the abdomen, and this, associated with signs and symptoms of a high small bowel obstruction, will give a clue to the diagnosis

Treatment is laparotomy after adequate pre-operative decompression and preparation The volvulus is reduced and the cecum must be freed and allowed to migrate into the left side of the abdomen Where gangrenous bowel is present it must be resected and anastomosis accomplished It is now well known that the newborn infant will tolerate the resection of a large amount of small intestine, and this excision of gangrenous bowel should be carried out in every case The abdomen is closed in layers and a 1/4" Penrose drain placed in the peritoneal cavity Meticulous postoperative care is required in these infants, and protracted diarrhea may pose a difficult problem which can usually be overcome with a careful feeding regime and the use of a fat-free formula

Reversed Rotation

Reversed rotation of the midgut loop may present a puzzling problem for the surgeon who laparotomizes an infant whenever this condition exists The embryogenesis lies in a return of the postarterial loop of the midgut to the peritoneal cavity before the prearterial loop has entered, and clockwise rotation of 90 degrees allows the colon to come to lie behind the superior mesenteric artery The duodenum will cross in front of the transverse colon and the superior mesenteric artery If this anomaly of rotation is associated with an imperforate anus or Hirschsprung's disease, the performance of a transverse loop colostomy may be difficult unless this anomaly is understood

Internal Hernia

Internal or intra-abdominal hernias may occur at several sites and give rise to intestinal obstruction and may appear in any part of the mesentery Herniation through a defect in the mesentery in the region of the terminal ileum is the most common variety in childhood Other sites include those through the ascending mesocolon and the sigmoid meso-

colon and the extremely rare variety which transgresses the foramen of Winslow. Internal hernias seen in infancy and childhood also include the mesentericoparietal types. Hernias of this group are congenital in origin and may arise from a defect in the mesentery or from a defect in fusion of the ascending or descending mesocolon with the posterior abdominal parietes The paraduodenal variety occurs in the region of the junction of the duodenum and jejunum, where the duodenum emerges from its retroperitoneal position Two potential fossae are present, one which develops on the left and faces to the right and the other which develops on the right and faces to the left These have been called, respectively, left and right mesentericoparietal hernias. (See Fig. 378)

Symptoms and Signs of Intra-abdominal Hernia.—When these intra-abdominal herniations give rise to symptoms they are the same as those associated with intestinal obstruction, i.e., nausea, vomiting, crampy abdominal pain, and dehydration, and where the incarcerated intestine becomes deprived of its blood supply, melena may appear A palpable mass is sometimes present, and auscultation of the abdomen may reveal high-pitched bowel sounds. Roentgenologic examination of the abdomen will reveal dilated and gas-filled loops of intestine above the site of obstruction, and there may be a sentinel loop In the mesentericoparietal variety the radiologic picture of the intestinal pattern may be such as to show the small bowel collected into these peritoneal spaces.

Treatment.—The treatment is to release the intestinal obstruction present, reduce the hernia, and repair the defect in the base of the mesentery Resection of the nonviable intestine is carried out Reduction of this hernia in the infant is not usually difficult, but it must be remembered that in the left mesentericoparietal hernia the neck of the sac contains the inferior mesenteric artery, while in the right mesentericoparietal hernia the neck of the sac is in close proximity to the superior mesenteric artery The presence of these two important vascular structures in the vicinity of the hernial neck precludes the random incision of the neck to release the entrapped intestinal contents Laparotomy is undertaken for the mani-

festations of intestinal obstruction, and the diagnosis of internal hernia, if suspected preoperatively, may be confirmed only at the time of laparotomy. A thorough understanding of the sites and the mechanisms of these hernias that occur in infancy and childhood should be part of the armamentarium of every surgeon who undertakes operation in this age group

Omphalomesenteric Duct Apparatus

Embryology.—In the human embryo, at the beginning of somite formation at about the age of 16 days, the primitive gut lined by

testinal portal and becomes delineated as the foregut. Similarly, at the caudal end of the embryo an entodermally lined sac which comprises the embryologic hindgut remains in communication with the yolk sac through the posterior intestinal portal. When the embryo contains about 14 somites, or is about 22 days of age, there is a region of the primitive gut that lies between the foregut and the hindgut which is destined to be included within the embryo but as yet has no floor and communicates with the yolk sac. This region is known as the embryologic midgut. As the foregut and hindgut increase in length, at the expense of the midgut, the midgut remains in

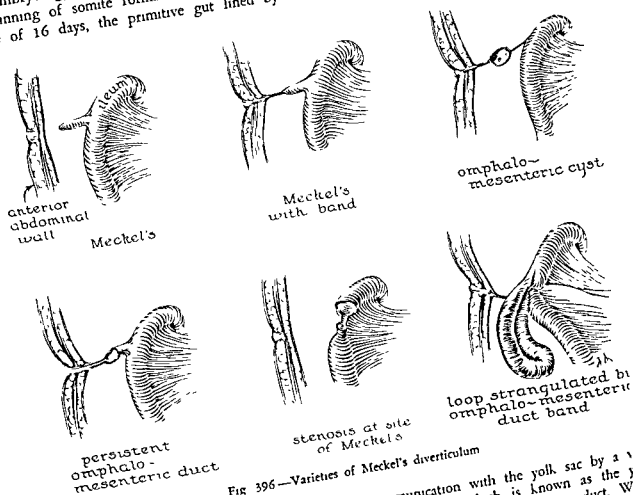


Fig 396—Varieties of Meckel's diverticulum

entoderm has a large communication with a similarly lined sac which is known as the yolk sac. The first portion of the primitive gut to be incorporated within the body of the embryo is that which lies under the head process, and with the forward growth of the head an entodermally lined tube communicates with the yolk sac through the anterior in-

communication with the yolk sac by a very narrow canal which is known as the yolk stalk or the omphalomesenteric duct. While the yolk sac is spoken of as a vestigial structure, morphologically it is of considerable size in the young embryos, and as the umbilical cord is formed, the yolk stalk is embedded in the substance of the cord, and the

small yolk sac is carried out to be caught between the amnion and the growing placenta. Our concern, then, in the discussion of the omphalomesenteric duct apparatus is not the yolk sac per se as it is extraembryonic but the varieties of form that a partial persistence of the yolk stalk may take. A sacculaton on the antimesenteric border of the ileum represents a partial persistence of the yolk stalk, and is known as a *Meckel's diverticulum* when it ends in a blind pouch.

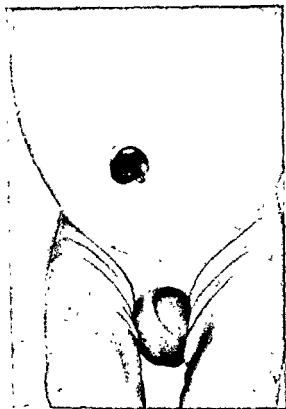


Fig 397—Patent omphalomesenteric duct. The duct is patent from the umbilicus to the ileum and has prolapsed slightly onto the abdominal wall.

Persistent Omphalomesenteric Duct

The omphalomesenteric duct extends from the primitive yolk sac to the antimesenteric border of the ileum. This duct may fail to obliterate and presents as a fistulous opening in the depression of the navel. It may secrete a thin mucuslike material which causes maceration of the umbilicus. Because of its communication with the ileum, contents of the small intestine may issue from the umbilicus.

Cauterization of the ostium of this fistula is futile, and careful probing or the injection of the fistula with Diodrast or Lipiodol will show the nature of the communication. The treatment of choice is excision with repair of the bowel wall at one end and excision of the fistula from the umbilicus at the other.

Meckel's Diverticulum

Meckel's diverticulum is one of the commonest anomalies of the intestinal tract. It has been stated that it is present in 2% of people, is 2" long, and is found 2' proximal to the ileocecal valve. In some series its incidence is closer to 3% and its length varies from 1/2-3". It may arise from 12-36" proximal to the ileocecal valve. It is not often a source of trouble, but when pathologic changes ensue they are likely to be serious ones. The

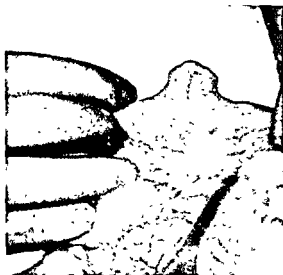


Fig 398—Meckel's diverticulum.

mucosa of a Meckel's diverticulum varies greatly in its histologic picture and may include gastric, colonic, or ileal type mucosa, or pancreatic tissue, within its substance. If symptoms arise from a Meckel's diverticulum they are most likely to do so in the first 2 years of life, and fully 50% of patients fall within this age group. The complications include peptic ulceration with perforation or hemorrhage, obstructive symptoms from intussusception or volvulus, and inflammatory symptoms with or without perforation.

premedicated and prepared for the operating room which is in readiness should reduction be impossible

4. That 10-12" of the terminal ileum be visualized at the time of the reduction

5. That cessation of symptoms should follow the hydrostatic reduction

Objections advanced to reduction by barium enema are as follows:

1 That it is an unsatisfactory procedure for patients with an ileoileal intussusception

2 That it may be incomplete, particularly in those intussusceptions which are ileoileocolic

3 That the viability of the intestine cannot be determined

4 That an etiologic factor such as an invaginated Meckel's diverticulum or a pedunculated polyp cannot be removed at the time of the correction of the condition

The proponents of the reduction by barium enema state that the mortality and morbidity rates are low and that the dangers associated with the operative treatment in young infants are avoided. The recurrence rate after hydrostatic pressure reduction is somewhat higher, reaching 6% in some series as opposed to 2% for operative reduction. It has been pointed out by the proponents of the non-operative treatment that the postoperative mechanical obstruction from adhesions is less common in the conservative treatment and they recommend operative intervention for cases in the older age group, where an etiologic cause is more frequently demonstrable, and for recurrent cases, where one may more likely find a precipitating factor. Each method appears to have merit, and when properly applied for the individual case the mortality rate has been reduced from the initial 50% to less than 1%, an accomplishment which has only been superseded by the operative treatment of congenital hypertrophic pyloric stenosis.

COLON, RECTUM, AND ANUS

Embryology

The development of the rectum and anus is complicated by its close association with the developing urogenital system. In the 5-week embryo, the urogenital system and the hindgut open into a common cavity, the cloaca, which is closed off from the exterior by the cloacal

membrane. As growth progresses a urorectal septum of mesoderm descends toward the cloacal membrane, defining the urogenital system anteriorly and the hindgut posteriorly. The diminishing communication between the urogenital system and the hindgut is known as the cloacal sinus. When the urorectal septum reaches the cloacal membrane it forms the perineum and divides the cloacal membrane, to leave the urogenital membrane anteriorly and the anal membrane posteriorly. The urogenital membrane perforates at about the 7th to 8th week. The anal canal develops externally to form the proctodeum. This structure is surrounded by mesodermal elements which give rise to the external anal sphincter. The hindgut and the proctodeum meet at the anal membrane, which perforates at about the 9th week to form the anorectal canal.

Congenital Anomalies of Rectum and Anus

Arrest or abnormal development during the critical period from the 6th to 9th week gives rise to the various anomalies of the rectum and anus

Anal Stenosis.—Perforation of the anal membrane occurs, but the anal canal remains narrowed

Imperforate Anus.—This is of varying degree which may be classified into three main types:

1 *Membranous.* There is persistence of the anal membrane

2 *Absence of anal canal.* There is failure of development of the canal, and the rectum ends as a blind pouch at a distance from the skin. This is the common form of malformation occurring in 80% of cases

3 *Presence of anal canal.* The anal canal is normal, but the rectal pouch is a blind sac. Some distance separates the proctodeal cul-de-sac from the blind rectal pouch

Associated Fistulas.—Persistence of the cloacal sinus gives rise to fistulas between the urogenital system or perineum and the hindgut which are present in most anomalies of the rectum and anus and must be searched for carefully. The rectoperineal fistula is common to both sexes. In the male the rectum may communicate with the trigone of the bladder, i.e., a rectovesical fistula, or with the prostatic

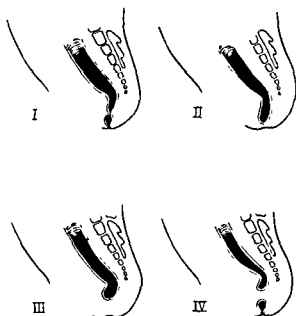


Fig 404—Congenital stenosis and types of imperforate anus. Treatment in I and II is a simple dilatation or a cruciate incision in the anal diaphragm. Treatment in III and IV requires considerable mobilization of the proximal bowel. (After Gross.)

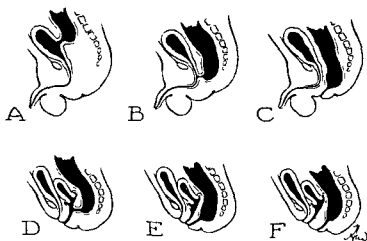


Fig 405—Imperforate anus with associated fistula. (After Gross.)

Male

- A, Rectovesical fistula
- B, Rectourethral fistula
- C, Rectoperineal fistula

Female

- D, Rectovaginal fistula
- E, Rectofossa navicularis fistula
- F, Rectoperineal fistula

or membranous urethra, i.e., a rectourethral fistula. In the female the rectum may end in the posterior vaginal wall, commonly in the lower third, to give a rectovaginal fistula. Rectovesical and rectourethral fistulas are rare in the female but do occur.

Clinical Features.—*Anal stenosis* causes partial obstruction, with obstipation or the appearance of ribbonlike stools. *Imperforate anus* is usually associated with complete intestinal obstruction. However, a rectoperineal or rectovaginal fistula may be large enough to modify the clinical picture to that of partial obstruction. The passage of meconium rules out a complete obstruction. The presence of meconium in the urine indicates the presence of a rectourinary fistula.

Plain lateral films are then taken with the child inverted, or lying on his back with the thighs flexed on the abdomen, and a radiopaque marker placed at the anal dimple. The distance between the marker and the air shadow in the rectum can be measured. This procedure is most important in evaluating the malformation and determining the surgical approach to its correction.

At operation the perineum may be explored with a syringe and needle before the approach is decided. If meconium is aspirated, Lipiodol may be injected into the rectum to outline the blind sac. X-ray will then show the true degree of the defect. Malformation of the rectum and anus has a high incidence of associated anomalies, particularly of the upper urinary

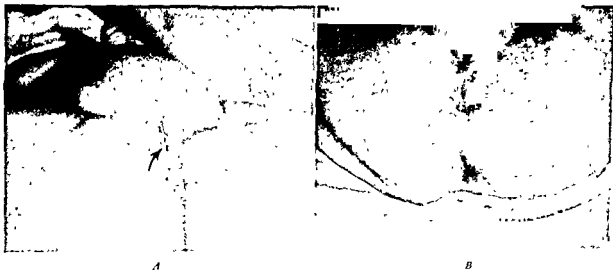


Fig. 406—A, Imperforate anus in a male with a small midline perineal fistula
B, Imperforate anus in a female

The perineum of the child must be examined carefully. The absence of the anus is easily noted. In the membranous imperforate anus, the anal membrane appears black and bulging due to the presence of meconium. A perineal or vaginal fistula will be evident by the passage of meconium at an abnormal site. Examination of the anal canal will reveal a stenosis or the persistence of the anal membrane at the end of a normal anal canal. X-ray examination of the abdomen confirms the diagnosis and is used to determine the degree of the defect between the rectum and anus. If the examination is delayed 12-24 hours, gas in the intestinal tract will have reached the

tract. Severe defects of the ureters or kidneys should be ruled out before operation by intravenous pyelography.

Treatment.—Anal stenosis is treated by repeated dilatation, which can be done by the parents at home. The membranous type of imperforate anus is relieved by a cruciate incision of the membrane. Dilatations are then started and repeated at regular intervals as in anal stenosis.

The imperforate anus with no anal canal may be approached by two methods which are determined by the distance between the rectum and the skin of the perineum.

1. *Perineal Approach*—This is used if the defect is less than 2 cm. Careful dissection of the perineum is carried out in the midline, deep to the anal dimple, until the blind rectal pouch is found, mobilized, and brought through the external anal sphincter and sutured to the skin without tension.

2. *Abdominoperineal Approach*—This is used if the rectum is high in the pelvis. In small infants a transverse colostomy may be performed as an interval procedure until the child has reached a weight of 10 kg. The

a fistula to the urinary tract must be treated immediately because of the hazard of an ascending urinary infection. A formal abdominoperineal repair is indicated, but a transverse colostomy may again be used as a temporizing measure. Low rectovaginal and rectoperineal fistulas are repaired by the perineal approach, lifting the fistula over the perineum and bringing the bowel down through the anal sphincter. Frequently these fistulas may be dilated to allow adequate gastrointestinal function. Repair may then be delayed past the neonatal period and performed as an elective procedure at an older age.

Congenital Megacolon (Hirschsprung's Disease)

Congenital megacolon, although rather well documented in the literature prior to the publication of Hirschsprung in 1888, is now known as Hirschsprung's disease, and while many theories have been advanced as to its etiology, it has only been in recent years that a definitive theory has been universally accepted as to the precipitating etiologic factor. There is a congenital absence of the ganglion cells in the myenteric plexus of Auerbach situated in the pelvic colon, which may extend cephalad up the colon for varying distances. In the affected area there is absence of peristaltic activity, and this region acts as a physiologic obstruction to the forward propulsion of gas, fluid, and feces. The normal proximal colon becomes dilated and hypertrophied, while the affected segment, although patent, remains of normal caliber. With this understanding of the causation of the condition, surgical therapy has kept pace and now offers definitive cure for these patients. With vigilance and cognizance of the condition, the diagnosis is being made more frequently in the neonatal period. With the realization that the underlying etiology is an absence of ganglion cells in the myenteric plexus of the rectum, the recto-sigmoid, and sigmoid, attention has been withdrawn from the dilated and hypertrophied bowel which occurs proximally to this area, and emphasis has been placed on the site of physiologic obstruction that results from the absence of adequate peristalsis in this narrow segment of colon. Primary or congenital



Fig 407—Imperforate anus. Plain film, with patient inverted in the buttocks up position, showing gas in the rectal pouch at a distance from a marker placed in the perineum.

definitive procedure mobilizes the sigmoid and rectum down to the levator ani muscles. A perineal dissection is then made and the bowel pulled through the levator ani muscles and external anal sphincter and anchored to the skin.

The high incidence of fistulas associated with imperforate anus requires most careful dissection of the blind pouch of the rectum to identify possible fistulas. The presence of

megacolon must be differentiated from secondary megacolon which may be present proximal to an anal stenosis or strictures or present in those children who withhold stool for one reason or another. The onset of symptoms may appear shortly after birth. It has been pointed out that in children who have undergone laparotomy for intestinal obstruction in the newborn period, where no mechanical block is found, congenital megacolon should be strongly suspected and colostomy performed. Histologic section of the colon reveals the absence of ganglion cells, which

Palpation or visualization of the fecal masses in the greatly enlarged colon is often possible, and peristaltic waves may be seen through the thin, tense anterior abdominal wall. Rectal examination, while inconclusive in the newborn infant, is frequently of diagnostic significance in the older child, where the rectum appears to be normal in diameter, and in those children with sigmoid impaction a large bolus of stool may be felt with the examining finger. While constipation is the rule, diarrhea may occur when liquid feces pass around the impacted mass of stool. Many of these children

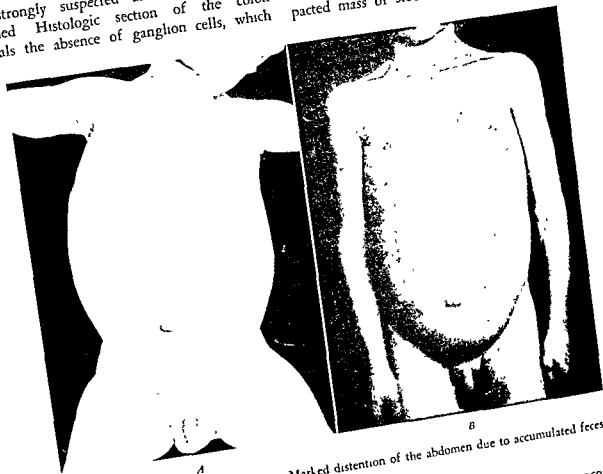


Fig 408.—Congenital megacolon. Marked distention of the abdomen due to accumulated feces. A, In an infant. B, In a young child.

appears to be an all-or-none phenomenon. In very small infants with the onset of symptoms shortly after birth, it would appear that there is a more severe degree of aganglionosis and that this defect may be carried well proximal to the sigmoid colon. There is a sex ratio, 9 males to 1 female. Persistent constipation with enlargement of the abdomen, elevation of the leaves of the diaphragm, anorexia, and evidence of malnutrition may be present

with Hirschsprung's disease show concomitant megaloblasts and megaloureters, presumably from the same neurologic deficit, and it is important to investigate the urinary tract. X-ray examination of the abdomen is often helpful in confirming a diagnosis, and a markedly dilated colon containing large amounts of gas and feces should arouse suspicion. Careful examination of the large bowel with limited amounts of barium will reveal that no me-

life to adolescence. Unlike multiple polyposis or the isolated polyp in the adult, there is little tendency to malignant degeneration. The patient may present with (1) bleeding per rectum, (2) prolapse of a polyp through the anus, or (3) intussusception. Rectal and sigmoidoscopic examination will demonstrate their presence in the lower bowel. Once the diagnosis is suspected, a barium enema must be given to rule out polyps at a higher level



Fig 410—Rectal polyp outlined by barium after evacuation

Postevacuation films after a barium enema are essential to demonstrate the polyps. Other causes of bleeding per rectum in the child must be excluded (1) fissure-in-ano, (2) Meckel's diverticulum, (3) intussusception, (4) intestinal duplication, and (5) hemorrhagic diseases.

The treatment of the isolated polyps in children is removal by snare and cauterization of those polyps that can be reached by proctoscope. Polyps high in the sigmoid or proximal intestine must be removed by abdominal exploration with ileotomy or colectomy over the site of the individual polyp.

BILIARY SYSTEM, LIVER, AND PORTAL HYPERTENSION

Obstructive Jaundice in Infancy

The persistence of jaundice in the infant past the newborn period poses a difficult problem in differential diagnosis. Conditions of surgical importance are those causing obstructive as distinguished from nonobstructive jaundice, by the presence of acholic stools and dark bile-stained urine. (See Chapter 19.) Obstruction to the flow of bile in the infant is caused by (1) inspissated bile pigments, (2) congenital atresia of the bile ducts, and (3) neonatal hepatitis. Mucous plugs in the common duct and extraluminal compression of the biliary system may be additional but rare causes. Unless the obstruction is relieved there may be irreversible hepatic changes and biliary cirrhosis.

Inspissation of bile pigments in the bile ducts and canaliculi occurs with excessive hemolysis and is seen in erythroblastosis fetalis (Rh sensitivity) and icterus precox (ABO sensitivity). Hematologic tests for erythrocyte fragility, infant and maternal blood group incompatibility, and antibody determinations will isolate cases of obstructive jaundice due to increased hemolysis. These conditions should be anticipated by antenatal blood studies of the mother and exchange transfusion of the infant after birth. Most cases of bile pigment obstruction will respond to expectant therapy with the use of cholagogues. Severe blockage of the bile ducts may require laparotomy and washing out of the bile plugs.

Unfortunately, in obstructive jaundice due to congenital atresia of the bile ducts or neonatal hepatitis, the clinical picture is identical and laparotomy examinations will not give a clear differential diagnosis. A steadily rising bilirubin is suggestive of biliary atresia, but the values may fluctuate widely in either condition. Other tests of liver function are even more confusing. Twenty per cent of the anomalies found in congenital atresia of the bile ducts can be treated surgically (see congenital atresia of the bile ducts, Chapter 20). It is of utmost importance that a definite diagnosis be established before the progressive changes of biliary cirrhosis occur. Experience has shown

that in most cases of obstructive jaundice, in infants will succumb to the effects of biliary cirrhosis, even if the obstruction is relieved early. For this reason a diagnostic laparotomy is required at or prior to 1 month of age. The procedure is not extensive. A small incision is made over the liver, sufficient to allow inspection of the subhepatic area. An absence of the gall bladder indicates that there is a probable extrahepatic biliary atresia. If the gall bladder is present it is catheterized and a cholangiogram obtained. At the same time a liver biopsy is taken. The incision is then closed, and if the gall bladder has been catheterized, the tube is left in place for drainage and used for repeated cholangiographic studies if necessary. Correlation of the cholangiogram and the pathologic findings of the liver biopsy will furnish a correct diagnosis. Liver biopsy will establish the diagnosis of intrahepatic biliary atresia, for which there is no treatment, extrahepatic biliary obstruction manifested by the presence of some degree of biliary cirrhosis, or the hepatocellular changes of neonatal hepatitis. Cholangiogram will demonstrate the site of the extrahepatic biliary obstruction. An early definitive approach may then be planned for a proved case of extrahepatic biliary atresia, and the child with neonatal hepatitis has not been subjected to a prolonged surgical exploration of the biliary system. This technique has proved the prognosis in those cases of biliary atresia that are amenable to surgical correction.

Portal Hypertension in Childhood

Our experience with 21 patients operated upon for portal hypertension (1949-1956) is given in brief below to indicate the peculiarities and difficulties encountered in the handling of this entity in a children's hospital (See also Chapter 19).

Etiology.—Unlike the portal hypertension which manifests itself in adult life, our cases have been confined to those classified as subhepatic or extrahepatic portal obstruction, and rarely have we seen hepatic cirrhosis as the precipitating factor. The causes of extrahepatic portal block have been arranged in an interest-

ing classification which includes (1) trauma, (2) postoperative mesenteric vein thrombosis, (3) systemic infections, (4) neonatal omphalitis, and (5) congenital absence of intrahepatic portal venous radicals.

Neonatal omphalitis is the most common cause of portal hypertension in childhood. The infection spreads up the lumen of the umbilical vein, which runs in the free edge of the falciform ligament, and an accompanying septic thrombosis passes from the umbilical vein to the portal vein via intercommunications. Recanalization creates a condition which is known as *cavernomatous degeneration or transformation of the portal vein*, the result of which is an extrahepatic portal block and an inflammatory reaction at the porta hepatis. Blockage of the flow of blood in the portal vein gives rise to portal hypertension and is manifested by hemorrhage from esophageal varices and splenomegaly. Massive hematemesis in a previously normal child or the design of increased portal pressure. In some cases anemia, thrombocytopenia, or leukopenia (hypersplenism) may suggest radiographic and esophagoscopic studies, with the visualization of esophageal varices. In a child the commonest cause of massive hematemesis is bleeding from such varices, as the other conditions encountered in adults are uncommon.

Treatment.—When severe hypersplenism exists the treatment is splenectomy. In children over 5 years of age, splenorenal anastomosis should be attempted unless the splenic vein is hopelessly inadequate in caliber. In extrahepatic portal obstruction, portacaval anastomosis is often impossible because of cavernomatous transformation of the portal vein.

We have performed a transesophageal ligation of varices in seven patients with extrahepatic portal block for recurrent hemorrhage. One patient had a previous portacaval anastomosis, two a splenorenal anastomosis, and all a splenectomy. Only two of these patients have rehemorrhaged. Results of the shunting procedures in childhood have been disappointing. Perhaps in extrahepatic obstructions, splenectomy alone, accompanied by transesophageal ligation of varices, will become an accepted procedure. Clatworthy has attempted

end-to-end anastomosis of the superior mesenteric vein to the cut proximal inferior vena cava in cases where other previous anastomoses have failed

We have reduced portal pressures from over 600 mm of saline to under 300 mm by means of shunting procedures on several occasions in children and have had recurrent bleeding in all cases. There is no evidence that these small caliber venovenous anastomoses remain patent in children.

PERITONEUM, OMENTUM, AND MESENTERIES

Primary Peritonitis

Primary peritonitis is a condition which is found most commonly in infancy and early childhood. The peritonitis does not arise from an inflammatory focus within the peritoneal cavity but is probably carried there during bacteremia. It was once thought that the most common organism was the pneumococcus and that this condition occurred almost exclusively in females, the infection being carried to the peritoneum by way of the fallopian tubes. It is now known that the most common organism is the hemolytic streptococcus and that the sex incidence is about equal. More than two thirds of the patients are under 5 years of age. The illness usually begins with an upper respiratory tract infection, which is rapidly followed by severe abdominal pain and by vomiting which may progress to dehydration. Diarrhea is not uncommon in the early stages, but as the peritonitis progresses constipation or obstipation is the rule. The picture is one of extreme toxicity with high fever and rapid pulse. The abdomen is distended, doughy to palpation, and diffusely tender without localized tenderness. Rectal examination reveals diffuse pelvic tenderness. The white blood count is elevated and ranges from 30,000-60,000, 80-90% being polymorphonuclear leukocytes. The presence of pneumonia in infants and young children must be excluded by x-ray, and this may be exceedingly difficult because pneumonia and primary peritonitis may occur simultaneously in the very young infant. This condition must be differentiated from a generalized secondary peritonitis, the commonest source of which is a ruptured ap-

pendix. Acute appendicitis is rare under the age of 2 years and uncommon under the age of 3 years. The progression of the abdominal symptoms of an acute appendicitis, with periumbilical pain and localization to the right lower quadrant before the onset of generalized peritonitis, is the usual picture, while in primary peritonitis the tenderness is diffuse from the beginning. The association with an upper respiratory tract infection, the onset with chill and high fever, the severe toxicity, and the markedly elevated white count all help to make an adequate diagnosis which is only possible in two thirds of the cases, necessitating confirmation by limited laparotomy. The contraindications to limited laparotomy are (1) indications, where the association of primary peritonitis is common and antibiotic therapy is proving satisfactory, and (2) marked toxicity and sepsis from some generalized condition in which the peritonitis is only a part. On opening the peritoneal cavity the odor of the contents should be carefully noted, and if not feculent, suspicion should be aroused. A smear of the peritoneal exudate should be examined immediately. If the streptococcus or pneumococcus is demonstrated, the diagnosis is confirmed. As much exudate as possible should be aspirated through the small incision and a Penrose drain inserted into the pelvis. The postoperative care after this diagnostic laparotomy is of utmost importance. Penicillin is the antibiotic of choice and should be given in large doses until all the acute symptoms have subsided and then continued in diminished dosage for several days. Adequate sedation must be used. The infant does best in an atmosphere of high oxygen content. Continuous Wangenstein suction with a Levin tube in the stomach should be used until the bowel sounds are well heard and the infant has passed feces and flatus. Nutrition should be maintained by the intravenous route and blood or packed red blood cells administered to combat the anemia which is so often present in these infants with overwhelming peritonitis. Concentrated human serum albumin can be employed for the correction of hypoproteinemia. The drain into the peritoneal cavity should be mobilized in 48 hours and then gradually shortened day by day until completely removed.

It is extremely important to recognize this condition in the young infant and child. Failure to suspect it or extensive laparotomy to find a source of primary infection within the peritoneal cavity is accompanied by increased morbidity and mortality. The instillation of 1 million units crystalline penicillin and 1 Gm streptomycin into the peritoneal cavity is a procedure which may be useful. With the advent of antibiotic therapy and its institution from the very beginning of this condition, together with a limited laparotomy and adequate postoperative care, the mortality rate from primary peritonitis has been considerably diminished.

Omental Cysts

Embryology.—These thin-walled unilocular or multilocular cystic masses which appear in the omentum are thought to arise from developmental anomalies of the lymphatics within this structure. They are lined by a single layer of endothelial cells and filled with a clear serous fluid. Enlargement occurs slowly except when sudden hemorrhage into the loculated structures of the cyst causes rapid change in size.

Signs and Symptoms.—These children usually present with a history of a gradual enlargement of the abdomen, and on physical examination a soft palpable mass may be felt anywhere in the abdominal cavity, depending on the site of the cyst at the time. A space-occupying lesion can be visualized by x-ray, and if it lies in the anterior portion of the abdominal cavity, an omental cyst may be suspected. A further diagnostic aid is the use of pneumoperitoneum, and x-ray studies with this double contrast medium will outline the cystic mass.

Treatment.—The treatment of choice is simple excision. The smaller omental cysts can be readily dissected from between the leaves of the omentum. The larger varieties may require resection of most of the omentum to ensure removal of the entire cyst.

Mesenteric Cysts

Definition.—These are smooth, thin-walled masses which lie between the leaves of the

mesentery of the small bowel or between the layers of the mesocolon.

Etiology.—It is thought that these cysts develop from ectopic rests of lymphatic tissue, which continue to secrete and enlarge because of lack of communication with the normal lymphatic channels. They are most commonly found between the leaves of the mesentery of the jejunum or the ileum but may occur between the layers of the mesocolon throughout its entire length. They are soft and the contents may vary greatly in volume. Some, occurring between the leaves of the mesentery of the small intestine, contain chyle and are known as *chylous cysts*. Other mesenteric cysts contain a serous fluid similar to that of plasma in concentration and composition.

Diagnosis.—These children usually present with a painless, slowly increasing growth of the abdomen. The cysts may cause intermittent crampy abdominal pain and occasional vomiting, or rarely give rise to acute intestinal obstruction when the cystic mass is saddle-shaped, resting on the adjacent bowel and obstructing its lumen. In most cases a soft fluctuant swelling can be palpated within the abdomen which appears to be rather freely mobile. X-ray examination may reveal a space-occupying lesion displacing intestinal loops, and pneumoperitoneum may afford better visualization.

Treatment.—In many instances it is possible to dissect the cyst from between the layers of the mesentery without damage to the arterial or venous supply to the intestine. Sometimes, because of the size of the cyst and its proximity to the intestine, it may be necessary to resect the adjacent intestine with the cyst in order to preserve the blood supply to the bowel.

ABDOMINAL WALL

Congenital Absence of the Abdominal Musculature

This is a condition which has been reported with increasing frequency in recent years. The three main groups of associated anomalies are found in the genitourinary tract, the gastrointestinal tract, and in the extremities. The genitourinary anomalies that occur most fre-

quently with the absence of abdominal musculature comprise hydroureter, hydronephrosis, and an enlarged bladder. The etiology of this condition is obscure. There is a strong preponderance in the male sex, and because of this, the theory of a primary genitourinary tract obstruction as an etiologic factor is proposed by Spumme since such obstructions are more commonly found in the male infant. Another, almost constant finding is failure of the

is extremely variable and depends on early diagnosis and correction of the associated anomalies.

Neoplasms of the Abdominal Wall

Neoplastic masses may appear in any portion of the anterior abdominal wall, and the majority are present at birth. The most common mass is a *hemangioma*, which may be capillary or cavernomatous in type and is benign. The treatment is surgical excision and repair of the defect in the anterior abdominal wall. *Lipomas* of the anterior abdominal wall frequently associated with lipomatous masses elsewhere, are common. They are characteristic in appearance and present a typical roentgenologic picture. The treatment is simple excision.

The Desmoid Tumor.—The desmoid tumor of the rectus sheath is characteristically found anterior to the sheath and occurs more frequently below the level of the umbilicus. It presents a firm, asymptomatic, ovoid mass which is not well circumscribed and is attached deeply to the rectus sheath. This tumor tends to recur locally and, for this reason, primary excision should be complete.

The Umbilicus

Omphalocele

Definition.—During the 5th to 10th week of intrauterine life, a discrepancy between the size of the developing intra-abdominal structures and the size of the celomic cavity which contains them results in the physiologic herniation of the midgut, sometimes accompanied by a portion of the liver, into the base of the umbilical cord. If the structures of the midgut remain too large, or the celomic cavity is too small to contain them, this physiologic herniation may persist as an *omphalocele*. Normally, the structures of the midgut are returned to the peritoneal cavity in a definitive manner at about the 10th week of intrauterine life. The hernia in the base of the umbilical cord is characteristic: (1) the two umbilical arteries and the umbilical vein may be seen coursing over its surface, and (2) the pro-



Fig. 411—Congenital absence of abdominal musculature

descent of the testes into the scrotal sac. Whatever the cause, the disturbance in embryogenesis must occur before the 10th week of fetal life. The constant physical findings are those associated with absence of the abdominal musculature and flaring of the lower rib cage. The abdominal wall is soft and the abdominal organs can be readily palpated. The prognosis

truded abdominal contents may be visualized through the thin, translucent avascular covering of the pouch, which consists only of peritoneum and amniotic membrane. The size of the sac may be quite variable but on the average measures 10 cm in diameter. The size of this sac is not related to the size of the opening at the umbilicus through which the intestinal contents protrude. Because of the perishable nature of the covering membranes and their avascularity, this condition becomes one of the most urgent observed in the delivery room. Indeed, it has been said that these babies should be transferred from the arms of the obstetrician to the arms of the surgeon. During



Fig 412—Omphalocele. A large herniation into the umbilical cord through a comparatively small defect at the umbilicus.

The operative correction can be approached in two ways: (1) If the pouch and its contents are small, the pouch can be emptied by the replacement of the abdominal viscera into the peritoneal cavity, excision of the skin at the base of the pouch carried out, and repair of the anterior abdominal wall accomplished in a one-stage procedure. (2) However, if the omphalocele is large this may present a formidable problem. It may be necessary to accomplish the repair in a two-stage procedure: (a) Excision of the pouch is made down to the point where fresh skin edges can be obtained with closure of these freshened skin edges and subcutaneous tissue over the protruding mass of abdominal viscera. In these large omphaloceles there is little risk in burying the covering of peritoneum and amniotic membrane under the skin if it has been carefully cleansed with a bland detergent. Post-operative support by the use of a binder or an elastic bandage can be instituted, and this gentle pressure is maintained until the peritoneal cavity is of sufficient capacity to contain the abdominal viscera. After 10-12 weeks it is usually possible to pick up the covering skin and see that the abdominal contents can remain intraperitoneally. (b) At this time a second stage is undertaken in which the defect in the anterior abdominal wall is repaired and the redundant skin excised and closed.

Cyst of the Umbilical Cord

Throughout any length of the umbilical cord large collections of Wharton's jelly may occur and distend it in a cystic manner. The complications of this event are twofold: (1) a large cyst may interfere with delivery of the baby, and (2) a cyst may appear at the base, complicating the tying of the cord at birth. If there is little space between the proximal portion of the cord and the anterior abdominal wall, the vessels of the cord must be tied distal to the cyst, but because of the nature of the cyst covering, its avascularity, and its tendency to early drying, surgical removal should be attempted as soon as possible. *Treatment* is surgical excision of the cyst and ligation of the two umbilical arteries and the vein on the abdominal side of the structure.

the first few hours of life the sac remains moist and will permit a certain amount of gentle handling, but later, with drying and because of the avascularity, evisceration and infection will occur. It is important to realize that the persistence of this midgut hernia is almost always associated with some degree of failure of rotation of the intestine. Definitive corrective surgery is carried out most easily before the contents of the pouch have become distended with gas and fluid. There is no place for watchful waiting in the treatment of this condition.

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organism is most sensitive. Warm, moist saline compresses should be applied to the local area, and when abscess formation occurs, an attempt should be made to drain these localizations of pus. With the advent of peritonitis the prognosis is extremely grave and the mortality rate in this condition can only be lowered by instituting the treatment that has been advocated for the therapy of a primary peritonitis.

Urachal Cyst and Sinus

In early embryonic life, the urinary bladder extends from the umbilicus down to the bladder neck. As caudal migration of the urinary vesicle occurs, a remnant of its umbilical attachment may persist which may be patent throughout, communicate both with the umbilicus and the dome of the bladder, and manifest itself by the periodic excretion of variable amounts of urine at the umbilicus. The urachal remnant may be obliterated at the umbilicus and at its attachment to the bladder to form a urachal cyst. This presents as a midline swelling in the lower abdomen. Diagnosis of the persistent urachus is accomplished by means of a cystogram, while the anterior extraperitoneal position of the midline mass of the urachal cyst is diagnostic. The treatment of both these conditions is surgical excision at the earliest moment, which is readily performed before infection has supervened.

HERNIA

(See also Chapter 29)

Umbilical Hernia

This is a common condition in infancy and childhood. It occurs at a point where there are muscular and fascial defects at the umbilicus. Unlike the omphalocele, it is never a surgical emergency. The peritoneum is closed and forms a sac which in this case is covered by skin and subcutaneous tissue. The hernial ring is usually firm, fibrous, and circular in shape and is formed by the fusion of the anterior and posterior rectus sheaths. This condition may be confined to the region of the umbilicus itself or may also appear above and below the umbilicus in the form of a supraumbilical or infraumbilical herniation. These weaknesses

can be detected in the early months of life and are readily observed in infants who have prolonged periods of crying or straining. They become more prominent when the patient assumes an upright position and vary in size and content. Their diameter ranges from 1-4 cm, and the contents may comprise omentum or omentum with a single loop of small intestine. The symptoms are usually mild. Incarceration is a rare complication and strangulation practically never occurs.

Treatment.—

CONSERVATIVE APPROACH.—It is important to realize that the vast majority of small umbilical hernias in infants will obliterate themselves as the child grows and, for this reason, do not require treatment. In the larger umbilical hernias, however, closure may be facilitated by the application of an adhesive

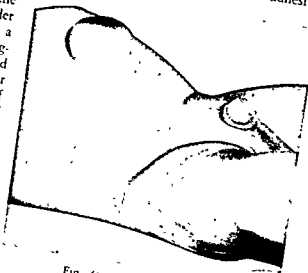


Fig 413—Umbilical hernia

strapping over the region of the umbilicus under sufficient tension to give a vertical crease in the region of the umbilicus. If no improvement has occurred after 4-6 months of strapping it is futile to continue this therapy. Strapping probably plays no part in the cure of hernia in patients over the age of 6 months.

SURGICAL APPROACH.—The indications for umbilical herniorrhaphy depend on the age of the patient and the size of the neck of the sac. The umbilical hernial ring, 1 cm. or less in diameter, will usually close spontaneously. If the hernia is present at 2 years of age it

Persistent Granulation Tissue at the Umbilicus

Within the first few weeks of life and after the cord has separated, a small mass of granulation-like tissue may appear in the depths of the umbilicus. This mass may discharge a serosanguineous fluid and cause maceration of the skin of the newly healed umbilicus. This is usually a temporary condition which responds to cauterization with silver nitrate, but sometimes this conservative therapy is unsuccessful and surgical excision is necessary. In some of our cases this tissue sent for histologic section has revealed the presence of an intestinal-like mucosa which accounts for its continual secretion and resistance to conservative treatment.

Omphalitis

Omphalitis, which by definition means *inflammation of the navel*, is an all-inclusive term. Inflammatory conditions may involve one or more of the several structures, and these inflammations vary in their severity and in the specific type of complication which may result. The infection may be localized and remain so, or it may spread in the lymphatic drainage of the periumbilical region. The process may begin at the umbilicus and ascend the lumen of the umbilical vein or it may propagate in the lumen of the unobliterated umbilical arteries. The organisms most commonly found are the staphylococcus and the hemolytic streptococcus, but mixed infections are not uncommon, and that caused by an anaerobic streptococcus may be particularly difficult to treat.

Localized Omphalitis.—Localized superficial infections in the region of the umbilicus rarely give rise to serious complications and respond well to local treatment.

Migrating Infections of the Umbilicus.—Infections about the umbilicus spread in several definite ways. If the infection is complicated by lymphangitis it may give rise to inguinal adenitis or, spreading upward in the fascial planes, it may develop into a localized abscess of the abdominal wall. If the organism is an anaerobic streptococcus, an extensive panniculitis may supervene which may give rise to desquamation and sloughing of the

skin over the anterior abdominal wall above the umbilicus. This appears first in the regions where the bony structures of the sternum and ribs underlie the spreading infection. The breakdown of tissue may be rapid, but the initial inflammatory reaction about the umbilicus may have subsided by the time the abdominal musculature is laid bare. The treatment consists of warm, moist saline compresses which are changed frequently to accomplish an effective débridement of the dead and dying tissue. Cleansing with hydrogen peroxide facilitates this, and the use of zinc peroxide which releases nascent oxygen may prove beneficial. Once the infection has been brought under control, re-epithelization is usually rapid and proceeds centrally into the defect. Large skin defects require a thin Thiersch graft.

Infections Spreading in the Lumen of Umbilical Vessels.—This type of spreading omphalitis carries the highest morbidity and mortality along with serious and far-reaching consequences. While the lumen of these vessels usually obliterates shortly after birth, they may remain patent for several weeks. As a result of this, umbilical artery or vein infection may give rise to a bacteremia with all its consequences. If the infection becomes localized along the path of the umbilical artery or vein, it may give rise to an extraperitoneal abscess and this, in turn,

ward course in the lowermost edge of the falx-form ligament and its communication with the portal vein and the inferior vena cava via the ductus venosus, inflammation may result in portal vein thrombosis. Such a thrombus organizes and becomes partially recanalized, giving rise to a condition which is known as *cavernomatous degeneration of the portal vein*, with an extrahepatic portal vein obstruction and portal hypertension as a possible complication.

Because of the extreme importance of omphalitis in the newborn infant, aseptic precautions should be carried out in the handling of the umbilical cord. If infection occurs, identification of the organism should be obtained, and the infant should be treated with massive doses of penicillin and the appropriate broad-spectrum antibiotic to which the

the cord have all enlarged to accommodate the hernia, and this is definitely discernible. The child will not tolerate invagination of the scrotum by the examining finger as will the adult, as the scrotum has not developed sufficiently to allow this procedure. The reduction of a hernial mass confirms the diagnosis.

Differential Diagnosis.—An inguinal hernia can be differentiated from (1) hydrocele, (2) acute inguinal adenitis, and (3) cryptorchidism with the testis in the canal. The hydrocele is firm, will not reduce, and transilluminates. Also it is possible to define the upper limits of the cyst outside the external ring. However, the presence of a hydrocele does not rule out an associated hernia. An acute inguinal adenitis may simulate an incarcerated hernia, as these nodes overlie the small inguinal canal of the child. The progressive symptoms of incarceration are not present, and there is usually evidence of inflammation. The child must be watched very closely to determine the true diagnosis. The partially descended testicle can be readily recognized by its absence from the scrotum.

Treatment.—A patent processus vaginalis or potential hernial sac may disappear in the first few months of life. However, once a hernial mass has become apparent, it is very rare for it to disappear. Small hernias in infants under 1 year may regress. If the hernia is large or the child is aged over 1 year, the processus will not obliterate spontaneously. Once the diagnosis has been made corrective surgery should be done. With adequate facilities for pediatric anesthesia and surgery, infants tolerate the procedure well and there is little risk. It is done as an elective operation, but the parents must be fully aware of the possibility of incarceration in the infant, which then presents an emergency situation. Intestinal obstruction and gangrene of the testis may be in infants. The operation may be delayed until after the child has passed the neonatal period and is thriving. In our hospital this is set arbitrarily at 3 months, or when the infant weighs 5 kg. The use of a truss as a specific treatment has been discarded.

Operation.—The operation for hernia in children is directed at removing the patent

processus vaginalis. Unless the hernia is unusually large the walls of the inguinal canal are normal and intact. A transverse incision is made in the skin crease over the canal. The external oblique is opened to the level of the subcutaneous inguinal ring to expose the cord. The hernial sac is then defined and dissected to the deep inguinal ring, where it is ligated and excised. The cord is returned to the canal ring by suturing the conjoined tendon to Poupart's ligament (Bassini repair). The external oblique, Scarpa's fascia, and the skin are closed in layers.

With this type of inguinal herniorrhaphy, limited to simple excision of the sac, the recurrence rate is less than 2%, which is more acceptable than a 10% incidence of testicular atrophy found in infants who have had a more extensive type of hernial repair. Recurrence is associated almost entirely with the emergency treatment of incarcerated hernias.

Postoperative Care.—Feedings are started in 4 hours, and the patient discharged on the 2nd postoperative day.

Diaphragmatic Hernia

Defects in the diaphragm, which give rise to hernias in infants and children, are almost entirely congenital in origin. However, traumatic hernias must be considered in compression injuries of the chest and abdomen.

Embryology.—The diaphragm develops as a muscular partition between the pleural and abdominal cavities. The central portion is formed by the ventral septum transversum and the dorsal mesentery. At this stage the pleural cavity on each side still communicates with the abdominal cavity through the pleuroperitoneal canals. This defect is then closed by a double serosal layer contributed by the peritoneum and pleura. Muscular elements of central and dorsal portions and, to a lesser degree, the body wall develop in this layer to form the muscular diaphragm with its central tendon. During its development the diaphragm incorporates or surrounds the contents of the dorsal mesentery—the foregut, the aorta, and the inferior vena cava. (See also p. 772 and Fig 374.)

should be repaired. Those with a fascial defect larger than 2 cm., and particularly those that contain bowel, are increasing in size, or give rise to occasional symptoms, should be treated by surgical correction.

Operation—There is no place in the surgical repair of the umbilical hernia in infancy for a procedure that does not preserve the normal umbilical depression. A periumbilical crescentic incision is made through the skin and subcutaneous tissue, and through this incision the hernial sac is isolated. The sac is then inverted into the peritoneal cavity and the medial aspects of both rectus muscles are approximated in the midline by a vertical row of interrupted sutures. Because of the vascularity in the periumbilical region and its tendency to produce a serous collection, the dome of the protruding skin should be sutured to the anterior rectus sheath. An occlusive dressing with lateral compression is applied. The older method of subcutaneous ligation of the hernial sac through three small incisions is not recommended. The occurrence of umbilical hernia is more common in female than in male infants, and because of the increase in the size of the abdomen with pregnancy, it is important that these larger hernias be treated by a surgical approach in the young female.

Inguinal Hernia

Inguinal hernias seen in children are congenital remnants of the processus vaginalis, which may remain patent to give an indirect, complete, or scrotal hernia. Persistence of a portion of the processus may give rise to an incomplete hernia, a hydrocele of the testis or tunica vaginalis, an encysted hydrocele of the cord in the male, or a cyst of the canal of Nuck in the female. As the right testicle descends after the left, the processus on the right closes at a later stage, which probably explains the higher incidence of right inguinal hernia. The experience of Gross is that 60% occur on the right, 15% are bilateral, and 25% occur on the left side. The presence of a left inguinal hernia demands a careful examination of the right side, as a bilateral hernia may be present. Ninety per cent of the hernias occur in the male. (See Fig 360)

Clinical Features and Diagnosis.—Typically it becomes apparent after a few months and may present as a bulge at the external ring or descend into the scrotum. The contents are small bowel, cecum, or sigmoid, as the omentum has not developed sufficiently to reach into the hernial sac. In females, the contents are very frequently an ovary and fallopian tube because of the shallow pelvis and the association of the round ligament of the uterus with the inguinal canal. The parents' observation of a swelling in the groin may be the only specific indication of a hernia. In older children with large hernias there may

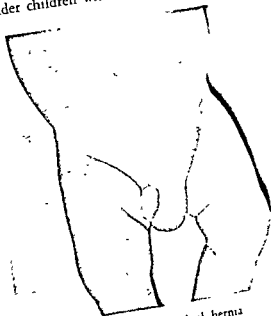


Fig 414—Left inguinal hernia

be local discomfort. The chief hazard is incarceration, which is most common in the first year of life and may be the first evidence of a hernia. The swelling is tense, and the child is in obvious pain. If it has persisted for some time, vomiting occurs, and the condition progresses to strangulation of the bowel and intestinal obstruction.

Inspection is the first examination for hernia. Increasing the intra-abdominal pressure by allowing the infant to cry or by having the older child stand will frequently produce the telltale bulge. Gentle palpation of the spermatic cord by rolling it under the finger tip will demonstrate a definite thickening on the suspected side. Even if the sac is empty the structures of

the cord have all enlarged to accommodate the hernia, and this is definitely discernible. The child will not tolerate invagination of the scrotum by the examining finger as will the adult, as the scrotum has not developed sufficiently to allow this procedure. The reduction of a hernial mass confirms the diagnosis.

Differential Diagnosis.—An inguinal hernia can be differentiated from (1) hydrocele, (2) acute inguinal adenitis, and (3) cryptorchidism with the testis in the canal. The *hydrocele* is firm, will not reduce, and transilluminates. Also it is possible to define the upper limits of the cyst outside the external ring. However, the presence of a hydrocele does not rule out an associated hernia. An *acute inguinal adenitis* may simulate an incarcerated hernia, as these nodes overlie the small inguinal canal of the child. The progressive symptoms of incarceration are not present, and there is usually evidence of inflammation. The child must be watched very closely to determine the true diagnosis. The *partially descended testicle* can be readily recognized by its absence from the scrotum.

Treatment.—A patent processus vaginalis or potential hernial sac may disappear in the first few months of life. However, once a hernial mass has become apparent, it is very rare for it to disappear. Small hernias in infants under 1 year may regress. If the hernia is large or the child is aged over 1 year, the processus will not obliterate spontaneously. Once the diagnosis has been made corrective surgery should be done. With adequate facilities for pediatric anesthesia and surgery, infants tolerate the procedure well and there is little risk. It is done as an elective operation, but the parents must be fully aware of the possibility of incarceration in the infant, which then presents an emergency situation. Intestinal obstruction and gangrene of the testis may be in infants. The operation may be delayed until after the child has passed the neonatal period and is thriving. In our hospital this is set arbitrarily at 3 months, or when the infant weighs 5 kg. The use of a truss as a specific treatment has been discarded.

Operation.—The operation for hernia in children is directed at removing the patent

processus vaginalis. Unless the hernia is unusually large the walls of the inguinal canal are normal and intact. A transverse incision is made in the skin crease over the canal. The external oblique is opened to the level of the subcutaneous inguinal ring to expose the cord. The hernial sac is then defined and dissected to the deep inguinal ring, where it is ligated and excised. The cord is returned to the canal. It is rarely necessary to decrease the internal ring by suturing the conjoined tendon to Poupart's ligament (Bassini repair). The external oblique, Scarpa's fascia, and the skin are closed in layers.

With this type of inguinal herniorrhaphy, limited to simple excision of the sac, the recurrence rate is less than 2%, which is more acceptable than a 10% incidence of testicular atrophy found in infants who have had a more extensive type of hernial repair. Recurrence is associated almost entirely with the emergency treatment of incarcerated hernias.

Postoperative Care.—Feedings are started in 4 hours, and the patient discharged on the 2nd postoperative day.

Diaphragmatic Hernia

Defects in the diaphragm, which give rise to hernias in infants and children, are almost entirely congenital in origin. However, traumatic hernias must be considered in compression injuries of the chest and abdomen.

Embryology.—The diaphragm develops as a muscular partition between the pleural and abdominal cavities. The central portion is formed by the ventral *septum transversum* and the dorsal mesentery. At this stage the pleural cavity on each side still communicates with the abdominal cavity through the pleuroperitoneal canals. This defect is then closed by a double serosal layer contributed by the peritoneum and pleura. Muscular elements of central and dorsal portions and, to a lesser degree, the body wall develop in this layer to form the muscular diaphragm with its central tendon. During its development the diaphragm incorporates or surrounds the contents of the dorsal mesentery—the foregut, the aorta, and the inferior vena cava (See also p 772 and Fig 374.)

Classification of Congenital Hernia of the Diaphragm.—The types are (1) posterolateral, (2) anterior, and (3) hiatal. Commonly the *posterolateral hernias* represent a persistence of the pleuroperitoneal canal in which there is free communication between the pleural and abdominal cavities. Less frequently the defect has been bridged by the serosal layer so that the hernia presents with a peritoneal sac. A congenital weakness or defect in the diaphragm anteriorly at its attachment to the sternum gives rise to a *retrosternal hernia*. These hernias may or may not present with a peritoneal sac. Rarely the defect also involves the diaphragmatic portion of the pericardium so that the herniation is into the pericardial sac. *Congenital hernias of the esophageal hiatus* may present with or without a peritoneal sac. The hiatus may be abnormally large and allow the stomach to herniate through the opening with a sac of peritoneum, which rarely may contain intestine. The diaphragm and the stomach both develop considerably cephalad to their final locations. If there is a disproportionate migration caudad, a portion of the stomach may develop above the diaphragm to give rise to a thoracic portion of stomach or what has been called a congenitally short esophagus. This type of anomaly may show a persistence of a segmental blood supply to the thoracic portion of the stomach from the aorta.

Clinical Features.—Symptoms are referable to the respiratory, circulatory, and digestive systems, and the severity will depend on the amount of abdominal contents filling the thorax. In some infants respiratory embarrassment requires prompt emergency treatment. Persistence of episodes of dyspnea and cyanosis will suggest the condition. Repeated pulmonary infections with failure to thrive is sometimes part of a prolonged history. Vomiting may be present. There may be a lag of the affected hemithorax with or without audible bowel sounds. The abdomen appears scaphoid. Plain films of the chest establish the diagnosis, and the use of radiopaque materials in this examination is unnecessary and contraindicated.

The hiatal hernia may cause symptoms in infancy. It should be suspected where there is persistent vomiting, particularly if the vomitus

is coffee ground in nature or frank hematemesis occurs. A definite diagnosis can be made only by careful x-ray examination and the demonstration of a portion of the stomach lying above the diaphragm. To differentiate this from *chalasia* or cardiac incompetence, gastric rugae must be demonstrated above the hiatal opening. Unless treated early, children

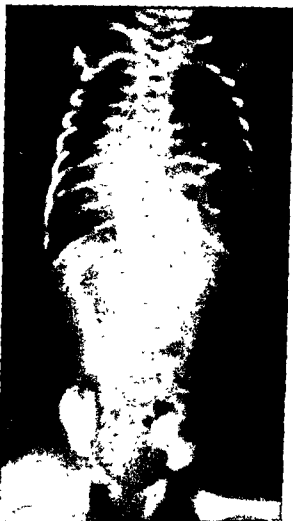


Fig. 415.—Posterolateral diaphragmatic hernia. Plain film of the chest and abdomen showing bowel in the left thorax displacing the heart and mediastinum to the right. The abdomen has decreased bowel shadows.

showing regurgitant symptoms will develop the complication of esophageal stricture. The presence of esophagitis or stenosis must be excluded by x-ray and esophagoscopy examination.

Treatment.—Surgical repair of the defect of the diaphragm after reduction of the hernia

Repair of hiatal hernia is approached from above through a left thoracotomy. The technique used is that described by Allison, which restores the action of the diaphragmatic crura at the esophagogastric junction



Fig 117—Hiatal hernia. A portion of the cardia of the stomach is above the diaphragm. There is an accompanying secondary stenosis of the esophagus.

WRINGER INJURIES

(See also Chapter 39)

With the decrease in the number of hand-operated clothes wringers and the popularity of electrically operated roller type wringers, the inquisitive child has become the victim of the *wringer injury*. It is our impression that with the advent of spin-dry and vacuum type clothes wringers on washing machines, these injuries are decreasing in frequency, but they still present a problem in a children's hospital.

The Injury.—The injury is almost exclusively confined to the upper extremity and may extend from the finger tips to the axilla. The severity depends upon the tightness of the rollers, the size of the arm, the duration of time the arm is between the rollers, and the length of the arm which is crushed. The injury may be intensified by the rollers being held up by an elbow which is maintained at right angles or by the parent attempting to extract the child without releasing or stopping the rollers. This may result in increased abrasive action in the antecubital fossa, the whole length of the arm, or damage to the shoulder joint or brachial plexus. An ipsilateral type of traumatic asphyxia has been seen on two occasions.

These injuries are likely to be misjudged by the uninitiated. Most wringer injuries are seen shortly after the accident and may appear deceptively benign. There may be slight swelling, ecchymosis, and minor abrasions, most marked in the antecubital fossa or axilla. If the thumb is abducted when the fingers are caught there may be an avulsion type laceration at the base, but fractures occur in only 1% of cases and are usually not displaced. All patients should be x-rayed. These injuries may look worse in 48-72 hours than on initial examination.

Treatment.—Because in the injuries seen early the initial treatment is so important, these patients are admitted to hospital with the exception of the obviously minor ones where rollers have been under little tension and the extent of crushing limited peripherally. The involved tissue should be cleansed with a bland detergent (aqueous Zephiran) and the ecchymotic and abraded areas covered with petrolatum gauze, over which a crimped roller gauze bandage is wrapped from finger tips to beyond the proximally crushed area. This should be covered with a cotton crepe bandage and well reinforced with 1" adhesive tape. The arm should then be elevated. In 24 hours the arm should be redressed with sterile precautions, and subcutaneous collections of blood aspirated or released through cutaneous incisions. Recently, intramuscular trypsin, streptokinase, and streptodornase have found a place in the treatment. The above therapy prevents subsequent skin loss in questionable areas, but where skin is lost it should be re-

placed by means of a Thiersch graft. When collections of serum and blood occur, systemic antibiotics should be administered. Skin contractures in the axilla and antecubital fossa should be anticipated and immobilization with the arm in abduction and the elbow in extension instituted. Lesions seen early and treated adequately leave little disfigurement. Those seen early and inadequately treated, or seen several days or weeks after injury, may show contractures, disfigurement, and limitations of function.

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Film References

Title	Running Time	Sound or Silent	Obtainable From
The Development of the Gastrointestinal Tract Part I—Embryology, Part II—Surgical Conditions	66 min 22 min	Silent Color	National Medical & Biological Film Library Canadian Film Institute 172 Wellington St Ottawa, Ont

Film References—Cont'd

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Acute Intussusception in Infants	20 min.	Sound Color	National Medical & Biological Film Library Canadian Film Institute 172 Wellington St Ottawa, Ont
The Diagnosis and Surgical Treatment of Congenital Hypertrophic Pyloric Stenosis (Brings out the most important features of diagnosis and technique of the Fredet-Rammstedt pyloromyotomy operation) (1952) (By Edward J Donovan, M D, New York)	12 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn
End-to-End Anastomosis of Esophagus for Congenital Atresia With Tracheo-esophageal Fistula (1952) (By George H Humphreys II, M D, New York)	31 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn
Operative Treatment for Hirschsprung's Disease (Demonstrates the author's technique for treatment of Hirschsprung's disease) (1950) (Orvar Swenson, M D, Boston)	15 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn
Acute Laryngo-tracheobronchitis (A clinical study of acute respiratory obstruction in children) (1952) (Dr H E McHugh, Montreal)	40 min	Silent Color	Chas E Frosst Co 350 Selby St Montreal, Quebec
Hernias in Infants and Children (1956) (Colin C Ferguson, M D, and Norman P Merkeley, M D, Winnipeg)	31 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn
Surgical Treatment of Pectus Excavatum The Surgical Repair of Funnel Chest (1955) (Colin C Ferguson, M D, Winnipeg)	12 min	Silent Color	Dept of Surgery University of Manitoba Winnipeg 3, Man

Chapter 31

Genitourinary System

John T MacLean, M.D

In genitourinary surgery it is essential that fundamental facts be recognized as such. These facts, combined with experience, provide a basic concept of the subject. It is for this reason that the various facts will be presented under seven main divisions, and these in turn will be applied to each area, e.g., kidney, bladder, etc. Following are the main divisions:

- 1 Anatomy and embryology
- 2 Normal physiology
- 3 Congenital abnormalities
- 4 Infections
- 5 Tumors
- 6 Trauma
- 7 Foreign bodies

Pyogenic infections, tuberculosis, and calculi will be discussed separately.

A carefully elicited history, physical examination, and urine analysis are often as valuable as the most expertly performed instrumental investigation.

A method of eliciting a history and examining a patient is as follows:

On admission to hospital:

- a Complete history
- b Urinalysis and blood count
- c Kidney function tests (PSP and NPN)
- d Roentgenograms and cystoscopy if indicated

Outline of History or Case Report

- 1 Chief complaint
Secondary complaints in order of importance

- 2 History of present illness; onset, giving symptoms in chronologic order as much as possible, looking for points in history referable to the different systems in order, as in the physical examination

Frequency:

- Day, night, on exercise
- Effect of cold or dampness
- Duration of symptoms

Pain:

- Site of pain
- Radiation to loin, genitalia, bladder, perineum, or penis
- Character—sharp—dull—aching, throbbing, requiring morphium
- Duration
- Number of attacks

Hematuria:

- Color and character: bright red clots—old or new
- Time of appearance—before, during, or after urination
- Following exercise or intercourse
- Duration

Dysuria:

- Time: on exercise, before, during, or after urinating
- Radiation to penis or elsewhere
- Duration

Alteration of urinary stream:

- Gradual or sudden narrowing of stream
- Retention of urine, complete or incomplete
- Number and duration of attacks
- Catheterization or other instrumentation employed

GENITOURINARY SYSTEM

Hesitancy in onset; force

If the facts do not appear in the history, note the absence of:

- a Hematuria
- b Passing of sand, gravel, or calculus
- c Chill
- d Fever

Physical examination

- a General
- b Local—genitourinary system

Kidneys

On examination note.

- a Tenderness in costolumbar angle
- b Kidneys palpable (bimanual examination)
- c Tenderness of kidney to palpation
- d Mass in renal region—description of size, extent, and mobility; ballottable or movable into loin; describe what you see, feel, and perceive, note any tenderness, rigidity, or splinting of the muscles of the anterior abdominal wall in the renal region

Ureters.

Examine for tenderness along course of ureters

Describe site and extent of tenderness and any masses palpated

Bladder and urethra

Inspect suprapubic area, describe visible masses as to size, shape, and extent, and by palpation any tenderness present, the consistency of any mass, and whether it is indurated or not, define the exact area of the mass by percussion

Urethral meatus.

Inspect meatus for size, discharge (smear). evidence of acute or chronic inflammation, indurations (meatal chancre)

Examine by external palpation the course of the urethra from the meatus to its disappearance in the perineum, note indurations and fistulas

Explore the urethra with soft rubber catheter (Fr No 14)

Note any obstruction found, the site, and whether it can be passed

Use gentleness at all times so as to avoid making a false passage

Record residual urine and bladder capacity in milliliters or ounces

Penis

Examine for scars, evidence of inflammation, rash

Scrotum.

Inspect for evidence of inflammation, induration or eruptions; describe any masses felt, their consistency, transmission of light, impulse on coughing, and whether they extend into the inguinal canal

Testes:

Describe size, consistency, induration, and whether any inflammation is present; note if testicular sensation is normal

Epididymes:

Describe size, consistency, tenderness

Vasa:

Describe indurations, nodules, tenderness

Venous of pampiniform plexus:

Varicocele, note if any present

Rectal examination:

- a Note if sphincter tone is normal
- b Describe prostate as to size, shape, consistency, nodules, fluctuation, and fixity to surrounding tissues
- c Carcinoma of rectum—note whether present or absent

Seminal vesicles:

Palpate for induration, nodules, tenderness

Laboratory Work

Routine on admission

- 1 Routine specimen of urine gross appearance, specific gravity, pH; sugar, albumin and microscopic examination
- 2 PSP renal function
- 3 Wassermann
- 4 NPN
- 5 Creatinine
- 6 Red and white counts, hemoglobin

Bacteriologic examination of urine specimen

Sterile specimen for culture

Prepare stained smears, including methylene blue, Gram's stain and Ziehl-Neelsen

Urethral discharge

Smears: methylene blue

Gram's stain

Prostatic fluid

Microscopic

Smear: methylene blue

Gram's stain

Dark-field examination

Serum from sores; puncture of inguinal nodes

Other Procedures Including Operative Procedures

*Intravenous Pyelogram Series**Cystoscopy*

Urethral urine specimens for

(a) Macroscopic, (b) Urea, (c) Volume,

(d) Microscopic, (e) Culture, (f)

Guinea pig inoculation

Retrograde pyelography

Biopsy with cystoscopic forceps

marking in the various transverse or horizontal levels. These are the xiphisternal junction (between T9 and 10); transpyloric plane (between L1 and 2); umbilicus (middle of body of L4); anterior superior spine (between L5 and S1)

The upper pole of the kidneys lies halfway between the horizontal line at the end of the body of the sternum and the transpyloric plane 2" from the midline, i.e., at the disc between the 11th and 12th thoracic vertebrae. The lower pole lies halfway between the transpyloric plane and the intertubercular line 3" from the midline, i.e., at the disc between the

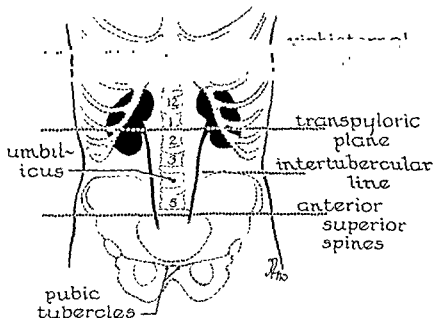


FIG. 418.—Surface anatomy of kidneys

Resectoscope.

Biopsy with resectoscope loop

Special tests

Mosenthal

Urea clearance

Guinea pig inoculation of 24-hour urine specimen

3rd and 4th lumbar vertebrae. The left kidney is very slightly higher than the right.

The ureter is marked out by a line drawn from the hilus of the kidney to the pubic tubercle.

A capsule of fat surrounds each kidney, and it in turn is enclosed between two fibrous layers derived from the fascia transversalis. The anterior layer is continuous with that of the opposite side and therefore passes in front of the inferior vena cava and the aorta. The posterior layer passes behind the kidney to the vertebral column. Above, the two layers unite

KIDNEYS

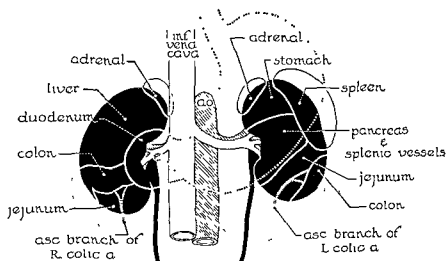
Anatomy

Surface Anatomy.—The kidneys can be marked out on the surface of the body by first drawing the vertebral column and then

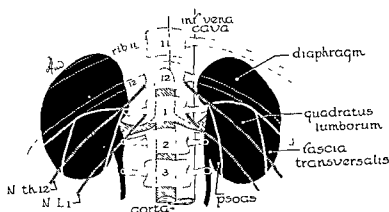
Below, the two layers are open. In nephroptosis, the kidney descends between these two layers. A perinephric abscess likewise extends downward upon the psoas muscle between these two layers. Nephroptosis may be corrected by stripping the capsule off the upper half of the kidney and attaching the lower pole to the 12th rib. It may also be corrected by suturing the anterior layer of the perirenal

fascia to the posterior abdominal wall, thereby forming a basket under the kidney.

Venous blood from the three paired glands on the right side (adrenal, kidney, and testicle) empties separately into the inferior vena cava; on the left side, that from all three into the renal vein. The right adrenal is covered by the "bare area of the liver." The lymphatics from the right adrenal empty directly into the portal



Anterior Relations



Posterior Relations

Fig. 419—Anterior and posterior relations of kidneys

ANTERIOR RELATIONS OF THE KIDNEYS

<i>Right Side</i>	<i>Left Side</i>
1 Adrenal	1 Adrenal
2 Duodenum	2 Stomach
3 Liver	3 Spleen
4 Colon	4 Pancreas
5 Ascending branch of right colic artery	5 Colon
	6 Ascending branch of left colic artery

POSTERIOR RELATIONS OF THE KIDNEYS

1 Diaphragm
2 12th rib
3 Psoas muscle
4 Quadratus lumborum
5 Fascia transversalis
6 12th thoracic nerve
7 1st lumbar nerve
8 Tips of transverse processes of 1st, 2nd, and 3rd lumbar vertebrae

system. Those of the left side are connected with the lumbar nodes and pass downward to the groin and aortic nodes and upward to the deep cervical nodes and the skull

Embryologic Development of Kidney.—In the human being there are three stages of development of the kidney. The pronephros, which develops during the 2nd week of

pronephros and development of the mesonephric tubules occur simultaneously, the latter replacing the former.

At a later date the metanephros develops and becomes the permanent kidney. The excretory portion is developed from the caudal end of the nephrogenic cord (at the level of the 1st and 2nd sacral segments).

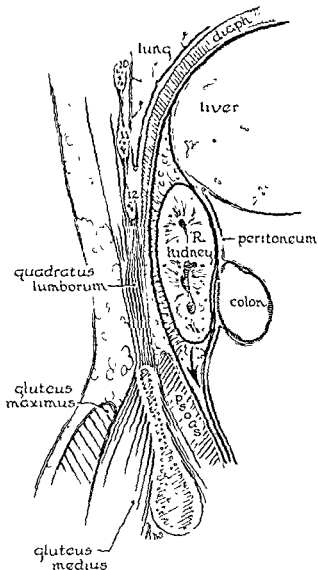


Fig 420 —Perirenal fascia The adrenal gland is medial to section shown

embryonic life on each side of the vertebral column, forms the wolffian body, the caudal portion of which is tubular and extends distally to connect with the cloaca. About the 3rd week of embryonic life, degeneration of the

The ureteric bud arises from the postero-medial wall of the mesonephric duct and eventually forms the entire tubular collecting system. The cranial end of the ureteric bud enlarges, comes into contact with the meta-

nephros (excretory portion) which forms a cap around it, and then by a series of subdivisions forms the major calyces which divide to form the minor calyces. Thus each major calyx and in turn each tubule has a cap of metanephric tissue which becomes more highly differentiated into excretory glomeruli and convoluted tubules.

nution of the body curvature during development. During ascent of the kidneys their intrinsic blood vessels receive their blood supply from lateral splanchnic stem arteries which arise from the aorta at increasingly higher levels until the final renal artery is reached. Failure of ascent results in congenital pelvic kidney and abnormal vascular supply.



Fig. 121.—Retrograde pyelogram with presacral air insufflation, showing cyst at upper pole of right kidney, confirmed at operation.

Congenital Anomalies of the Kidneys

Polycystic Kidneys.—The renal glomeruli may, in part, fail to join the collecting tubules. If such closed glomeruli secrete urine, cysts are formed. Marked degrees of this condition give rise to *congenital polycystic kidney*. Minor degrees account for isolated or solitary cysts.

Congenital Pelvic Kidney.—During its development, the metanephros undergoes a relative change of position which is usually called the "ascent" of the kidney. This is partly the result of cephalad growth of the ureter and blastemal cap but is due in part also to dimi-

Aberrant Renal Arteries.—These are the result of the persistence of the blood supply from lower levels than normal.

Horseshoe Kidney.—This is due to fusion of the lower poles of the kidneys during their ascent.

Double Ureter and Triple Ureter.—These are due to early division of the ureteric bud. Double ureter may be either unilateral or bilateral, complete or incomplete.

Fetal Lobulation.—This may persist in adult life.

Congenital Absence of One Kidney.—This is the result of the failure of the mesonephric

duct (wolffian duct) to produce a corresponding renal bud

Aplastic or Anaplastic Kidney.—This is due to failure of the metanephrogenic blastema to develop

Rotation of the Kidney.—This may be (1) absent (nonrotation), (2) incomplete, (3) excessive, (4) reversed

The malformation of the kidney, as such, rarely causes trouble, unless it is complicated by secondary pathologic changes. This finding has been supported by the large number of anomalous kidneys discovered at autopsy in patients who were urologically asymptomatic during life

Only 17% of individuals who have a congenital anomaly reach adult life without de-

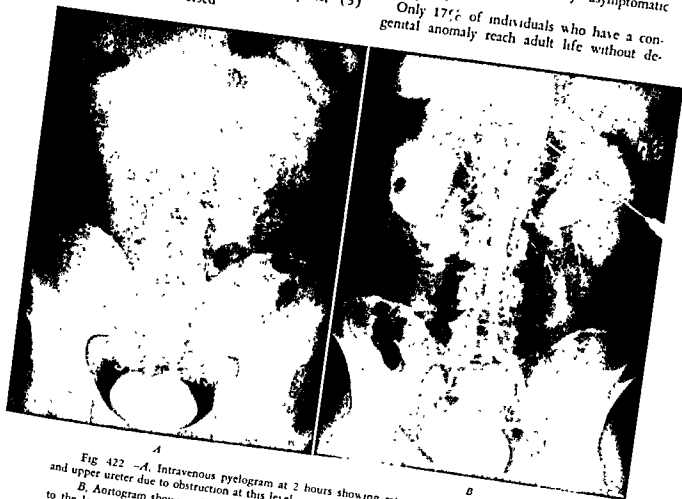


Fig. 422 —A, Intravenous pyelogram at 2 hours showing retention of dye in left renal pelvis and upper ureter due to obstruction at this level
B, Aortogram showing that the obstruction is due to an aberrant renal artery crossing the ureter to the lower pole of the left kidney. This was confirmed at operation. An intrinsic lesion was also found in the ureter, as is frequently the case

Symptoms in the Congenital Anomalies

The symptoms are listed in Table 30

	PERCENTAGE	APPROXIMATE FRACTION
Asymptomatic	17%	1/5
Pain	79%	4/5
Frequency	52%	1/2
Hematuria	25%	1/4
Calculi	10%	1/10
Gastrointestinal	32%	1/3

veloping symptoms referable to the malformation. It would seem to be the rule rather than the exception for the patient with an anomaly to develop symptoms that soon bring him to the urologist. Approximately 30% will have symptoms referable to the gastrointestinal tract.

Practically any urologic symptoms may be encountered in individuals with congenital anomalies. In most instances, the symptoms

GENITOURINARY SYSTEM

TABLE 29
EMBRYOLOGY AND CONGENITAL ABNORMALITIES

	NUMBER	SIZE	FORM	SITE	STRUCTURE
Kidney	Supernumerary Duplication Solitary	Infantile Hypoplasia Hypertrophy	Lobulated Horseshoe L-shaped Sigmoid Bifid	Rotation Ectopic Crossed ectopic unilateral fused Extrarenal Intrarenal Ectopic orifice	Cystic
Renal pelvis	Reduplication	Hydronephrotic	Valves Diverticulum Twists and kinks		
Ureter	Duplication complete incomplete	Megaloureter Stricture Ureterocele (Arterial and venous)		Low origin High origin Ectopic	Urachal cyst
Blood supply	Aberrant vessels	Hypertrophy	Diverticulum Persistent cloaca		
Bladder	Absence complete incomplete Duplication	Hypertrophy	Valves Cysts Diverticulum		
Urethra	Absence complete incomplete Duplication	Hypertrophy	Hypospadias Epispadias Hermaphroditism		Cryptorchid Ectopic
Penis	Absence complete incomplete Duplication	Hypertrophy Rudimentary penis			
Testicle	Anorchism Monorchism Polyorchism Microorchism Synorchism	Infantile			

Persistent anomalies of opposite sex are as follows *Male*—hydatid morgagni; utriculus masculinus *Female*—Gartner's duct

TABLE 30
INCIDENCE OF 471 CONGENITAL ANOMALIES OF THE KIDNEY AND URETER IN 18,460
CONSECUTIVE UROLOGIC ADMISSIONS TO ROYAL VICTORIA HOSPITAL

CONGENITAL ANOMALY	NO OF CASES IN SERIES	PERCENTAGE IN SERIES	CLINICAL INCIDENCE	CLINICAL INCIDENCE REPORTED IN LITERATURE
Solitary kidney	13	2.76	1,420	1,600
Hypoplasia of kidney	35	7.43	1,527	1,600
Abnormal form of kidney	58	12.31	1,318	--
Polycystic kidney	37	7.86	1,497	1,250
Horseshoe kidney	24	5.10	1,766	1,644
Ectopic kidney	19	4.03	1,972	1,000
Malrotation	47	9.98	1,392	1,195
Duplication of renal pelvis and ureter	97	20.70	1,190	--
Triple renal pelvis and ureter			--	4 cases reported
Abnormality of pelvis and calyces	79	16.67	1,234	--
Abnormalities of the ureter	15	3.18	1,231	1,500
Valve in ureter		--	--	4 cases reported
Aberrant vessels	47	9.98	1,392	1,313

are due to acquired disease. There are many cases, however, in which there is no evidence of renal or ureteral disease, but the complaints are renal or ureteral in location. The symptoms may be produced by the following:

1. Irritation of adjacent nerves
2. Increased peristalsis of a small, asymmetric portion of the kidney pelvis
3. Relative narrowing of the lumen at the point of bifurcation and poorly coordinated ureteral peristalsis
4. Physiologic hypertrophy on the normal side, or in the solitary kidney itself

Gastrointestinal symptoms may be produced by a distended renal pelvis sending impulses through the renal digestive reflex arc via the celiac plexus.

The associated pathologic processes encountered in congenital anomalies are due to malposition, malrotation with ureteral obstruction, and resultant hydronephrosis. Infection occurs in 80% of cases. Almost every known type of secondary renal disease has been found in anomalous kidneys. The commonest associated pathologic findings are pyelonephritis (60%), hydronephrosis (45%), and calculus (20%).

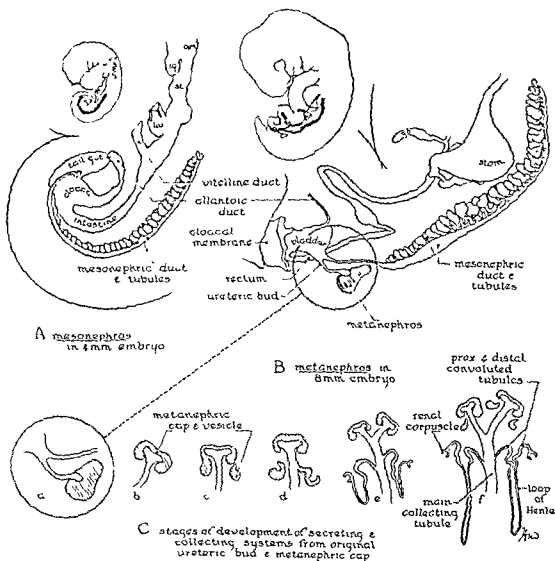


Fig. 423.—Mesonephric and metanephric tubules in the embryo showing development of collecting system (After Shikama)

Thirty per cent of all the patients with a congenital anomaly of the urinary tract will require a major operative procedure, and of these, 50% will require a nephrectomy

Physiology of the Kidney

Renal Circulation.—Each kidney is supplied by a renal artery derived from the aorta. The artery enters the hilus of the kidney between the renal vein which is anterior and the

(intralobular arteries) are given off which supply the cortex, while other branches extend downward into the medulla.

The veins accompany the arteries and form the same pattern. Each renal vein empties into the inferior vena cava.

It is now considered that there are three possible circulations within the kidney. The first is that just described and represents the normal circulation. The second, or inner, circulation, as described by Trueta and his asso-

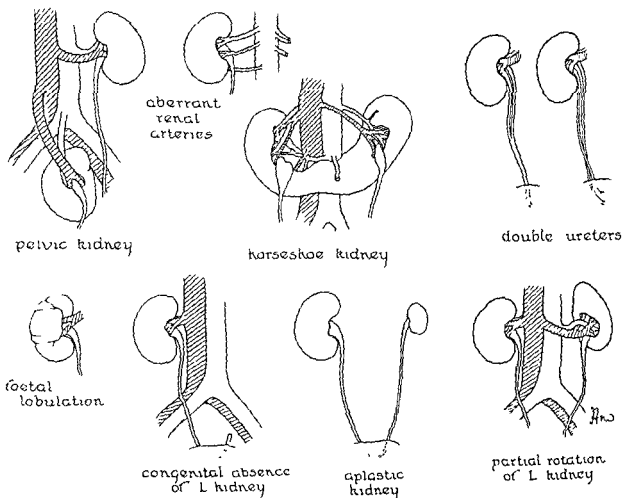


Fig. 424—Congenital abnormalities of the kidney and ureter (Modified after Grant)

renal pelvis which is posterior. Upon entering the kidney, or just prior to entry, the artery divides into several branches. These branches, in turn, give off interlobar arteries which run radially between the pyramids to the zone between the cortex and medulla, where they branch to form the arcuate arteries which run in a series of arcades between the two zones. From the arcuate arteries radial branches

ciates, consists of the passage of blood through the interlobular artery, the glomeruli lying in the deepest zone of the cortex (juxtamedullary glomeruli), arterial components of the vasa recta in the medulla, the venous components of the vasa recta, and the interlobular vein. Trueta and his associates claimed that during conditions of stress, blood might be "shunted" through this inner circulation, re-

sulting in blanching and relative ischemia of the cortex. There is no proof to date that this occurs clinically.

The third circulation within the kidney has been postulated by Barrie. He has described smooth muscle bundles which run longitudinally and parallel with the arcuate arteries, but on only one side of these arteries. They branch when the arteries branch, but are not continued onto the cortical arteries. Barrie has

believed that this mechanism is not found in normal kidneys but only in kidneys that have become diseased.

Glomerular and Tubular Function.—Renal function can be adjusted to meet the varying chemical demands placed upon it. By this means the composition of body fluids is maintained at levels consistent with normal cellular activity. Patients with impairment of renal function can maintain well-being only as long

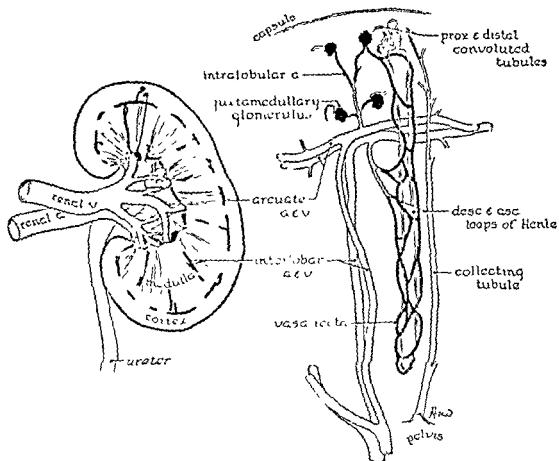


Fig. 425 Renal circulation

found a mesh of sinusoids or vacuoles in these muscle fibers. The sinusoids at one end have open communications with the arteriolar coil and at the other end drain into one of the larger veins. The function of this system is unknown, but Barrie believes that it may be regarded as an arteriovenous shunt, which acts as a decompression mechanism when the arterial circuit is overloaded. Barrie further be-

lieves that this mechanism is not found in normal kidneys but only in kidneys that have become diseased.

as they can be protected from stresses that tend to seriously alter the body chemistry. The normal volume of renal blood flow is approximately 1,000 ml. per minute (one-fifth of the cardiac output), so that in 5 minutes the total blood volume has passed through the renal circulation

Filtration pressure is normally 25 mm. Hg. It is the net effective pressure of two opposing

forces. On the one side of the scale is a positive pressure of 75 mm Hg within the capillaries, which tends to force water out of the capillaries. Opposing this there are pressures that tend to prevent filtration, as shown in Table 31.

Glomerular filtration ceases and anuria develops when the systolic blood pressure drops below 60 mm Hg. If hypotension persists for only a short while, the process may be reversed, but if prolonged, it becomes irreversible.

out the operation. In most cases an almost ischemic operative field is obtained. They regard the hypotension as perfectly harmless. They state that at pressures below 75 mm Hg, "secretion of urine is much diminished and may even cease, but while effective ventilation with oxygen is maintained as in the technique described, viability of the renal epithelium is not endangered when the systolic pressure rises to the level necessary for filtration, secretion of urine starts." In their series of 44 patients, 3 showed a reduction

TABLE 31

POSITIVE PRESSURE FAVORING FILTRATION	PRESSURES OPPOSING FILTRATION	NET EFFECTIVE FILTRATION PRESSURE	FILTRATE
75 mm Hg derived from arterial side	1 Osmotic pressure of plasma proteins, 30 mm Hg 2 Interstitial pressure, 10 mm Hg 3 Pressure necessary to move fluid down tubule, 10 mm Hg	25 mm Hg	120 ml of fluid per minute in tubule, all except 1 ml of which is reabsorbed by the tubule

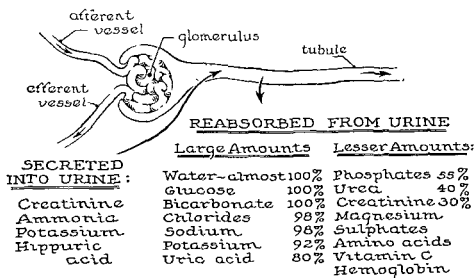


Fig. 426—Tubular secretion and reabsorption

The work of Griffiths and Gillies of Edinburgh directly opposes this concept of "filtration pressure" which has been held for many years. They produced a total sympathetic block with the use of intravenous Pentothal and a spinal anesthetic of 150-250 mg. of procaine which is allowed to extend up to and involve the first thoracic area. The blood pressure drops off to zero and is unrecordable through-

in the output of urine during the first 36 hours after operation.

Tubular Secretion and Reabsorption.—The glomerular filtrate is identical in composition to that of blood plasma without the protein. It therefore follows that to alter the composition of the filtrate to what is eventually excreted as urine, the tubules have three main functions

1. Reabsorption of very large quantities of water. Of every 125 ml of filtrate formed every minute, 124 ml are reabsorbed (Homer Smith).

2. Secretion of creatinine, ammonia, potassium, and hippuric acid

3. Reabsorption of certain threshold substances necessary to maintain the chemical equilibrium of the body, including acid-base balance. There is some difference of opinion as to the exact amounts reabsorbed, but the figures given by Allen appear acceptable. Water is almost completely absorbed, glucose entirely, sodium and chlorides about 98%, and potassium approximately 92%.

One should note that the entire function of the remainder of the urinary tract (from the tip of the pyramids onward) is to conduct the urine to the exterior (Davis). If there is obstruction to the normal outflow of urine, the brunt of the back pressure destructive changes eventually occur in the kidney so that urea, creatinine, and such may be retained in excess (azotemia), while other substances such as sodium, chloride, and potassium may be lost in excess due to lack of normal reabsorption. The resultant chemical imbalance may prove fatal.

Nerve Supply of Kidney, Pelvis, and Ureter.—The kidney capsule is supplied with sympathetic nerve fibers from T12 and L1. Likewise the pelvis and ureter have a sympathetic nerve supply from the T11 and 12 and 1st lumbar segments. In the latter case the autonomic ganglia are situated in the adventitia of the ureter. So far as is known, the parenchyma of the kidney has no nerve supply. Overdistention of the renal pelvis, or back pressure and distention of the whole kidney, with consequent stretching of the capsule, causes pain. However, the parenchyma may be completely destroyed as in tuberculous caseation, without pain, since the parenchyma lacks a nerve supply. Parasympathetic fibers from the vagus enter the kidney, but their function is unknown.

The renal plexus which surrounds the renal artery consists of an intricate network of nerve fibers derived from the aorticorenal ganglia, middle and inferior splanchnic nerves, inter-

mesenteric nerves, and fibers from the lumbar sympathetic chain. Surgical stripping of the renal artery, as sometimes carried out in selected cases of hypertension, is usually not a complete denervation.

The origin of the impulse which initiates ureteral contraction is obscure. Some workers believe that the impulse originates in the central nervous system. Others believe that the ureters can function automatically and initiate their own muscular contractions. Most agree, however, that the usual stimulus for ureteral peristalsis is the production of urine by the kidneys and consequent distention of the ureteral wall. Peristalsis is increased by increased excretion of urine, by increased intra-abdominal pressure, and by ureteral obstruction.

Lymphatics of the Kidneys.—There are three sets of lymphatics associated with the kidneys: (1) The superficial group of lymph channels lies in the superficial or perinephric fat surrounding the kidney. (2) The subcapsular group lies beneath the capsule. (3) The third or deep group of lymphatics lies within the substance of the kidney, surrounding the tubules and blood vessels in the interstitial tissue. They drain into larger channels at the hilus of the kidney and empty into adjacent aortic lymph nodes. They all intercommunicate freely, thus assisting the spread of infection through the kidney.

In retrograde pyelography, injection of excessive amounts of contrast media into the renal pelvis may cause a backflow of the dye into the deep (intrarenal) lymphatic system or veins. This is known as *pyelovenous backflow*.

Chyluria.—Normally the fat particles which enter the intestinal lymphatics give the lymph fluid a milky appearance (chyle). This passes upward through lymphatic channels and eventually into the main lymphatic duct. In some conditions such as filariasis, the main lymphatic channels may become occluded. The chyle backs up and may be forced by ever-mounting pressure into the deep lymphatics of the kidney and thence by further pressure into the renal pelvis, where it is excreted in the urine. This is known as *chyluria* and is the reverse process of pyelovenous backflow.

Orthostatic Albuminuria

Orthostatic albuminuria is a condition in which albumin is found in the urine without any pathologic condition in the kidneys. It is usually due to lordosis causing some pressure on the renal vein, resulting in delayed emptying.

Orthostatic albuminuria may be tested for by producing exaggerated lordosis in one of two ways: (1) by placing a pillow under the small of the back when the patient is lying flat, or (2) by having the patient bend backward 10 degrees for 10 minutes while standing with his hands on hips.

The following criteria are required before a diagnosis of orthostatic albuminuria is made:

- 1 No past history of renal or cardiovascular disease
- 2 No elevation of blood pressure
3. No WBC, RBC, or casts in the urine, except intermittently and in small numbers
4. Normal kidney function (PSP, urea clearance test, urine concentration tests)
- 5 Normal blood chemistry (NPN, urea clearance, total protein, normal albumin-globulin ratio)
- 6 Negative plain x-rays and normal intravenous pyelograms
7. Absence of albumin in the urine secreted when in the horizontal position

Hydronephrosis

Hydronephrosis is a dilatation of the renal pelvis, with stagnation of urine caused by an obstruction to the outflow of urine. The obstruction is nearly always mechanical, but on rare occasions may be due to neurogenic dysfunction. Hydronephrosis may affect one or both kidneys, it may be congenital or acquired and may be infected or not infected.

Primary Renal Atrophy.—Sudden complete obstruction of the ureter is thought by some to produce primary atrophy of the kidney without a preceding hydronephrosis. This belief is, however, not borne out by either experimental or clinical observation. It occurs so rarely that it may be disregarded.

Hydronephrosis With Secondary Renal Atrophy.—The usual sequence of events following obstruction to the outflow of urine

is as follows. Dilatation of the renal pelvis or renal pelvis and ureter above the site of the obstruction occurs, whether this is at the ureteropelvic junction, in the ureter, or at the entrance of the ureter into the bladder. As the back pressure is increased, the walls of the ureter and pelvis become progressively distended. Urine continues to be formed. Absorption of urine from the pelvis occurs while the kidney still continues to secrete urine. The contents of the hydronephrotic sac thus change continuously. Absorption of the fluid in the hydronephrosis is thought to take place either through the tubules or by pyelovenous backflow, or by both.

Eventually the combination of increased intrapelvic pressure, renal ischemia, and disuse results in degeneration and atrophy of the kidney.

Hydronephrosis develops more rapidly in a high obstruction than it does in a low obstruction. The rate of urine formation, however, remains the same whether fluids are forced or withheld completely.

Symptoms.—The symptoms are those of the underlying pathologic process, i.e., obstruction. There may be no symptoms at all or a dull ache in the back. If the hydronephrosis becomes infected, there will be chills, fever, and frequently pus in the urine.

Treatment.—Remove the underlying cause of the hydronephrosis if possible. If this is not possible, or if the disease in the kidney is far advanced, remove the kidney.

Anuria

Anuria is the cessation of urine formation. Oliguria is decreased secretion of urine. The causes of anuria are listed below. The post-renal causes are due to obstruction to the outflow of urine. They are usually classified under anuria because no urine is obtained. In these cases urine is formed, so that it is not a true anuria.

Causes of Anuria

I. Prerenal (Circulatory)

1. Occlusion of the main vessels of both kidneys by embolism or by the external pressure of a tumor

2. Reduction of blood volume due to
 - a Dehydration, e.g., diarrhea, vomiting, excessive perspiration
 - b Hemorrhage
3. Retention of fluids in the tissues (generalized edema)
 - a Advanced cardiac decompensation
 - b Retention of salt in the tissues
4. Endocrine form—due to dysfunction of the pituitary, thyroid, or adrenal gland—these forms rare
5. Low blood pressure, as in shock
6. Reflex anuria—or inhibitory form
 - a Inhibition of uninvolved kidney by obstruction in opposite kidney
 - b Vascular spasm of main vessels following splanchnic stimulation as in abdominal surgery, distention, etc.
 - c Peripheral irritation, e.g., passing sounds, catheterization, ureteral calculus, etc
 - d Anesthesia, especially spinal
 - e Severe painful stimuli
11. Renal—Intrinsic Renal Disease—(Excretory) (Occur on the basis of inadequate secreting renal tissue)
 - 1 Acute infectious nephritis, glomerulonephritis, suppurative nephritis
 2. Extensive destruction of kidney tissue bilaterally, e.g., advanced tuberculosis, polycystic kidney disease

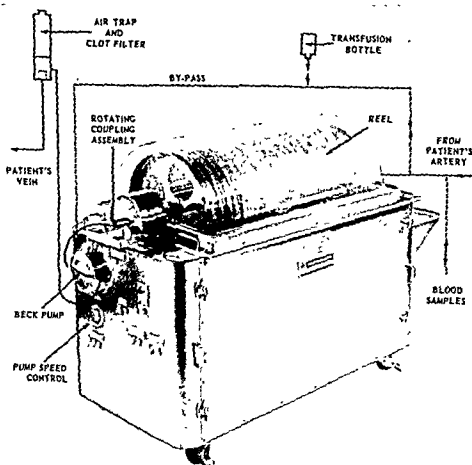


Fig. 427.—Flow of blood through the artificial kidney. The blood is forced by arterial pressure through Tygon tubing into the dialyzing membrane wound on the reel. It is worked to the left by the rotation of the reel. The rinsing fluid continuously bathes the membrane. The blood as it leaves the machine is pumped through an air trap and clot filter, back to a vein. A shunt line is provided to by-pass the machine. There is also provision for transfusion equipment.

3. Acute nephrosis, e.g., poisoning by heavy metals, mercury, bismuth, arsenic, lead
4. Acute toxic nephritis
 - a. Toxic products from damaged liver
 - b. Pre-eclamptic toxemia
5. Post-partum cortical necrosis
6. Following removal of solitary kidney

III. Postrenal—Obstructed Outflow of Urine (Eliminatory)

- 1 *Intrinsic*—blockage of one or both ureters by blood clot, calculi, stricture, neoplasm of pelvis or ureter, sulfonamides, tumor of bladder, enlargement of prostate
- 2 *Extrinsic*
 - a Accidental injury or ligation of ureters during gynecologic or other operations
 - b Retroperitoneal growths and malignant pelvic tumors
3. Composite or Mixed (Includes those cases where more than one of the above factors are present)
 - 1 Injuries to kidney and ureter
 - a Injury or avulsion of a single kidney
 - b. Bilateral renal rupture
 - 2 Lower nephron nephrosis—which may occur after crushing injury to muscle, non-traumatic muscular ischemia, burns,

transfusion reactions with incompatible blood, heat stroke, malaria, toxemia of pregnancy, alkalosis, sulfonamide anuria and following transurethral prostatic resection

Treatment of Anuria

Treatment of the prerenal and renal causes of anuria:

1. *Prevention*—maintain an adequate blood volume and adequate blood pressure.
2. *Active treatment*—try to restore the blood pressure and blood volume to normal by means of blood transfusions, vasoconstrictors, and adrenal cortical extracts. Renal decapsulation may be indicated

Treatment of postrenal causes of anuria: Remove obstructive cause if possible.

Treatment of lower nephron nephrosis: In lower nephron nephrosis the kidney normally tends to recover spontaneously between the 5th and 9th day of the disease. During this period it is important to limit fluids to 1,500 ml. daily, i.e., the amount lost through respiration and perspiration. If the anuria persists and the kidney fails to recover spontaneously, the artificial kidney may be used to wash the waste products from the blood

TABLE 32
TREATMENT OF LOWER NEPHRON NEPHROSIS

FIRST 24 HOURS (CONSERVATIVE TREATMENT)	AFTER 24 HOURS	5TH TO 9TH DAY
<ol style="list-style-type: none"> 1. Treat shock if present 2. Good nursing care 3. Posture, especially for good drainage of chest 4. Indwelling urethral catheter to collect all urine 5. Antibiotics 6. Digitalization if necessary 	<ol style="list-style-type: none"> 1. Cystoscopy with retrograde pyelography to ensure accurate diagnosis 2. Limit fluid intake of 1,500 ml of 5-10% glucose in water, plus an extra amount equal to the amount of urine excreted if any 3. Do not give sodium chloride during this period 4. Maintain electrolyte balance, correct acidosis by giving ½ molar sodium lactate intravenously or 1 ½% sodium bicarbonate 	<p>This is time that spontaneous recovery most likely to occur; if, however, patient becomes excessively toxic, treatment with artificial kidney recommended</p>

DURING RECOVERY

(when diuresis has become established)

1. Increase fluid intake beyond basic 1,500 ml daily, by an amount equal to amount of urine excreted in 24 hours
2. Measure 24-hour output of chloride in urine and replace this gram for gram
3. Frequent blood chemistry estimations; compensate for any deficiencies present

Bilateral Diffuse Cortical Necrosis

This lesion is defined by Duff and Moore as a pathologic entity of unknown cause manifested by extensive uniform necrosis of the cortex of both kidneys. There are two theories of origin: (1) that it is due to toxins, as in pregnancy; (2) that it is due to ischemia causing vascular occlusion. Clinically there is oliguria, anuria, and pain, usually epigastric, radiating along the ureters. Edema of the dependent parts occurs. The outcome is usually fatal, however, a very few patients do recover.

Sulfonamide Anuria

The use of the sulfonamides either as a prophylactic or therapeutic measure entails some risk.

The sulfonamides are excreted by the kidneys, being concentrated as much as three hundred times in the urine by tubular absorption. There is no relationship between the blood level of the sulfonamide and the patient's ability to tolerate the drug.

Any patient receiving the sulfonamides would be given enough fluids to ensure a 4-hour urinary output of at least 1,500 ml. Sodium bicarbonate may be given along with sulfonamide.

Sulfonamide anuria can be prevented by limiting the administration of sulfonamides and controlling the pH of the urine. The blood and urine levels of the drug, the presence of microscopic hematuria, and the possibility of sensitivity to the drug should all be borne in mind when using this type of therapy.

When anuria does occur, treatment should follow a definite plan. The drug must be discontinued. Cystoscopy is done to exclude the existence of mechanical obstruction to the drainage of urine and to wash each kidney pelvis, thereby removing any crystals and blood clot present. If mechanical obstruction of the ureter exists, and it is impossible to catheterize either renal pelvis, pyelotomy must be performed.

Renal Hypertension

(See Chapter 19, Liver and Portal Hypertension.)

Renal Rickets

Renal rickets is a disease of childhood characterized by the symptoms of polyuria, polydipsia, retardation of growth, deformity of growth, and delayed sexual development. A genu valgum develops. With the increased softening of the metaphyses, extreme degrees of distortion and deformity may be produced.

The kidneys show some degree of deficiency which may be pronounced as the disease advances. Chronic pyelonephritis is frequently present and calculus formation is a frequent complication. The specific gravity of the urine is fixed at a low level.

There are two types of bone changes which may occur.

1. Rachitic changes at the ends of long bones.
2. Generalized osteoporosis with loss of distinction between cortex and medulla.

The blood phosphate is elevated and the blood calcium lowered.

The cause of renal rickets is unknown. One theory is that it is primarily a renal disease, due to inability of the kidney to excrete an adequate amount of phosphates. A second theory is that renal rickets is an endocrine disease, caused by primary overactivity of the parathyroid glands with or without associated pituitary dysfunction.

There is no known treatment for this disease. Death from renal failure usually occurs before the age of 20 years.

RENAL INJURIES

The kidney is protected by its mobility and retroperitoneal position. Injuries are caused by external violence, such as a fall, kick, or blow on the back or side, by crushing injuries, or by penetrating wounds. Eighty per cent are due to blows on the back or side. The kidney responds like a ball of fluid to the force of an impact, transmitted in all directions.

The pathologic changes that occur are shown in Fig. 428 and are tabulated as follows.

1. Tears of the fatty capsule only. Hemorrhage into the perirenal tissues may be absorbed, organized, or encysted.
2. Contusion of the kidney. This is not serious unless it becomes infected.

3 Rupture of the parenchyma, usually posterior surface, with resultant pulping of the kidney. Hemorrhage may be marked or even fatal. Hematuria is always present. Nephrectomy may be considered.

4. Rupture of the parenchyma and capsule. The hemorrhage is greater than when the capsule is not torn.

5. Rupture of the parenchyma, capsule, and pelvis. The vessels of the renal pedicle are usually torn, and hemorrhage may be fatal. There may be urinary extravasation with infection.

Diagnosis.—Diagnosis is based on (1) history of injury, (2) variable degree of shock, (3) hematuria, (4) localized tenderness and rigidity, (5) a palpable mass, which may or may not be present.

The foregoing changes shown in Fig 428 describe the pathologic lesion which may be present in the kidney. It should be noted that clinically the extent or degree of injury is not nearly as precisely defined. If a patient has a complete rupture of the vascular pedicle, he obviously will live only a few minutes. Consequently upon the arrival of an injured

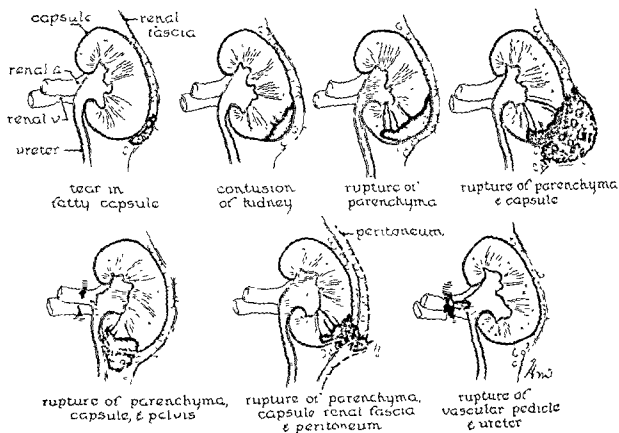


Fig 428—Injuries of the kidney

6. Rupture of the parenchyma with rupture of the peritoneum. This is a rare but serious type which occurs chiefly in children.

7. Rupture of the vascular pedicle and tearing of the ureter. The pedicle is sheared off. Death occurs promptly from hemorrhage.

8. Rupture of the liver or spleen may be associated with rupture of the kidney.

patient in the hospital, the immediate decision to be made is whether the injury is major or minor. The effect of the perirenal fascia in limiting the spread of the extrarenal hematoma should also be noted (Figs. 420 and 428).

Treatment.—

1. *Immediate.* The following supportive measures are most important: (a) treatment of

shock, (b) adequate blood transfusion. The blood pressure, pulse, and degree of hematuria must be carefully followed.

2 If patient is recovering, (a) continue conservative treatment, (b) give urinary antiseptic daily, (c) do an intravenous series as soon as possible after the patient is out of shock. This will give an approximate idea of the degree of damage to the kidney and establish the presence or absence of the other kidneys. If the bladder is included in the x-ray films it will also show whether there is an extravasation of dye from a rupture of the

If the patient continues to improve a cystoscopic with retrograde pyelograms and a differential PSP test may be carried out several days later. The PSP test is particularly valuable for defining in legal terms the degree of damage to the kidney, and it will indicate the degree of progression of the fibrosis in the ruptured kidney (see below).

3. If condition continues to deteriorate after initial therapy, i.e. pulse rate steadily increases or blood pressure cannot be maintained in spite of continuous transfusion, (a) prepare for nephrectomy and carry out exploration of the kidney, (b) make certain the opposite kidney is present before operating—this may be done by intravenous pyelography, (c) when the kidney is exposed and attempts to control the bleeding are unsuccessful, immediate nephrectomy should be done.

Complications.—

1. Sepsis—usually prevented by giving urinary antiseptics or antibiotics
2. Secondary hemorrhage
3. Fibrosis of part or the whole kidney resulting in scar formation with partial or complete loss of function

Prognosis.—

1. Seventy per cent recover without requiring an operation, even though critically ill on admission
2. A secondary nephrectomy for a nonfunctioning kidney may be required months after the initial injury.

NEPHROPTOSIS

Nephroptosis of the kidney is descent of the kidney beyond the normal 4 cm. dia-

phragmatic excursion. Reference to Fig. 420 will indicate how this may occur. It is present in approximately 1% of men and 6% of women. It is much more common on the right side because of the weight of the liver and the fact that the renal fossa is shallower on this side. The kidney is normally held in place by the fatty capsule and surrounding fascia, the vascular pedicle, and the support supplied by surrounding organs. In a thin person, and in one in whom there is a decrease of muscle tone, the additional trauma of a twist or fall, or the added weight of an overloaded colon, may be sufficient to cause ptosis of the kidney. Ptosis frequently causes kinking of the ureter and back pressure with resultant hydronephrosis.

The symptomatology varies considerably. There may be no symptoms referable to the ptosis, but in 66% of cases there is pain in the lumbar region, due to improper drainage, which gradually increases as the day wears on and is relieved by recumbency, which permits the kidney to fall back into place and thus improves drainage. The symptoms are occasionally entirely nervous because of a reflex from the kidney through the celiac axis. The patient then complains of epigastric pain, nausea, and vomiting. In some cases the patient complains of acute attacks of violent pain like that of renal colic. These attacks are called *Dietl's crises* and are attributed to kinking of the ureter or to twisting of the kidney pedicle. The symptoms may be entirely gastric (22%), in type, the patient being irritable and easily fatigued. It is of the utmost importance to select cases for operation carefully. It is only those patients who have pain in the back, increasing as the day advances, and relieved by lying down who should be subjected to operation. The accidental finding of ptosis of the kidney without any of the above symptoms is definitely not an indication for surgery. Similarly, the emotionally unstable type of individual with innumerable complaints will not obtain a good result from nephropexy, even though the corrected anatomic position of the kidney is excellent, the patient will continue to complain of pain. Generally speaking it is advisable to treat these patients with a well-fitting elastic girdle with attached

In the patient selected for operation, nephropexy may be done by one of several procedures. Decapsulation of the upper two thirds of the kidney with a suture passed through the lower pole and tied over the 12th rib is the operation most used. The denuded area of the kidney becomes firmly adherent to the undersurface of the diaphragm, peritoneum, and posterior abdominal wall. An alternative procedure is to expose the kidney and use the perirenal fascia to form an apron, which is stitched down to the muscles of the posterior abdominal wall with interrupted sutures, thus forming a basket beneath the lower pole of the kidney. If cases are properly selected as indicated above, and if the operation is well performed, the results are excellent. The patient obtains complete relief of pain, and recurrence of the ptosis does not take place. If, however, all patients with nephroptosis are operated upon without selection, the results will not be uniformly good.

TUMORS OF KIDNEY

Tumors of Children

Wilms' Tumor (Synonym: Embryoma or Adenomyosarcoma)

Clinically, Wilms' tumor or embryoma represents 20% of all tumors occurring among children. The age incidence is from birth to 10 years, 75% occurring in the first 5 years of life. In the later decades a period when other tumors are more common, one rarely encounters a Wilms' embryoma. Sex is not a factor. These tumors are extremely malignant. With early recognition, a 5-year cure may be effected in 25% of cases. A few cases have been reported in which the patient was alive 10 years or more after nephrectomy.

Origin.—The origin of mixed renal tumors is not clear. Most observers agree that the tumors are embryonal, in that they arise in the region of the developing kidney.

Three Theories of Origin—

1. The tumor is due to inclusions of Wolffian tissue which have become displaced and which persist among the cells of the developing kidney or metanephros.
2. The tumors arise from aberrant cells of the myotome and sclerotome, the apparent

mixed character being explained by the varying constituents that enter the ultimate formation.

3. The tumors are not due to inclusions from extrarenal sources, but they are true kidney, the embryonic tissue persisting and becoming metamorphosed into cellular structures of various types.

Pathology.—

Gross.—The Wilms' tumor is a large, solid, grayish white, encapsulated tumor of variegated histology. It is usually separated from the kidney by a layer of tough connective tissue. The capsule of the tumor blends with that of the kidney and with this membrane.

Microscopic.—The tumors are composed of a variety of tissues. The microscopic picture varies according to the predominating type of tissue present. In addition to simple epithelial and mesenchymal components, the presence of muscle elements indicates that tissue from the myotomes is included. The small papillary buds seen in some of the glandular components of the tumor may be abortive glomerular structures.

Signs and Symptoms.—

1. Presence of swelling in the upper abdomen—most cases.
2. Painless—usually.
3. Occasionally painful—usually late—due to pressure or to tension on peritoneum associated with weakness, pallor, fever, gastrointestinal symptoms due to bowel displacement, hematuria (10%), and hypertension.

Diagnosis.—History and signs and symptoms as above, plus deformity of the pyelograms.

Differential Diagnosis.—

1. Other kidney tumors and hydronephrosis.
2. Polycystic kidney disease.
3. Retroperitoneal tumors separate from the kidney.
4. Adrenal neuroblastoma (preoperative differential diagnosis may be impossible).
5. Splenic enlargements.
6. Tumors of liver.
7. Omental and pancreatic cysts.
8. Ovarian tumors.

Treatment.—Preoperative radiation followed by nephrectomy offers the best results.

TABLE 33
CLASSIFICATION OF TUMORS OF THE KIDNEY

A Children—Wilms' embryoma			
B Adults			
(a) PARENCHYMA			
Tissue of Origin		Common Name	Synonym
1 Epithelial		Benign Adenoma	
		Malignant Hypernephroma	Grawitz tumor
		Carcinoma	Hypernephroid carcinoma
2 Connective tissue		Benign { Fibroma Lipoma Leiomyoma	Adenocarcinoma
		Malignant Sarcoma	
3 Wilms' embryoma		Embryoma	
		Adenomyosarcoma	
(b) RENAL PELVIS		Papillary { Papilloma Papillary carcinoma	
		Solid type { Squamous cell carcinoma Adenocarcinoma	
(c) CAPSULAR TUMOR		{ Lipoma Fibrolipoma Fibrosarcoma Liposarcoma	

The tumor usually shrinks with radiation therapy followed by radiation is an alternative method of treatment.

Prognosis.—Although the tumor can be removed successfully, the likelihood of recurrence is so great that the prognosis is poor. Metastases may occur even before one suspects the presence of a primary tumor.

Tumors of Adults

- 1 Tumors of Renal Parenchyma
 - 2 Tumors of Renal Pelvis
- Forms 0.5% of all tumors in adults

Tumors of Renal Parenchyma

Tumors of Epithelial Origin—

BENIGN—Adenoma

Small adenomas, varying in size from 2 mm-1 cm. or larger, are found occasionally at post mortem in arteriosclerotic kidneys. They occur in the form of multiple discrete, whitish nodules located just beneath the renal capsule or scattered throughout the renal parenchyma. They often occur bilaterally. Histologically, they are made up of irregular groups of epithelial cells ranged around lumina which are regular in outline. Papillary projections of epithelial cell masses into such a lumen are a

frequent observation. The epithelium, however, is present only in a single layer. The resemblance to normal renal tubules is so close as to render differentiation difficult.

MALIGNANT.—Carcinoma (Synonyms: Grawitz tumor, hypernephroma; hypernephroid carcinoma).

Primary malignant tumor of the kidney is almost always unilateral.

In 1883 Grawitz proposed the theory that these tumors arise from aberrant adrenal tissue. Most people today believe that the tumor is a renal carcinoma arising either from adult tubules or from islets of nephrogenic tissue which have persisted in the renal cortex. A single renal tumor often exhibits mixed features.

Gross Pathology of Renal Carcinoma (Hypernephroma).—The striking variegated yellow and red of its gross appearance result from the high lipid content of the neoplastic cells and the hemorrhages which are almost invariably present.

Microscopic Pathology.—The clear cells which are prominent in microscopic sections of these tumors bear a vague resemblance to the cells of the adrenal cortex, which led

Grawitz to believe that they were derived from adrenal rests. Many hypernephroid tumors are not composed entirely of clear cells. Granular cells and mixed cell variants are also recognized. Furthermore, not all hypernephroid tumors are malignant, and the differentiation between adenoma and adenocarcinoma is often

looking growths may metastasize widely through the renal vein. It is therefore important to examine the vessels grossly and microscopically.

Metastases.—Metastases occur by direct extension of the growth through the capsule or into the renal vein, with showers of em-



Fig 429.—Wilms' embryoma. Intravenous pyelogram showing large shadow in region of left kidney with only superior calyces filled.

difficult. The size of the tumor is not a criterion of its degree of malignancy. If there is obvious invasion of the pelvis or frank cellular anaplasia, then the diagnosis of malignancy is simple. However, encapsulated and benign-

bolus into the blood stream. Spread takes place in this manner to the lungs, brain, and long bones. Pathologic fracture of a bone may occur before the primary tumor is recognized.



Fig. 430 —Wilms' embryoma. Gross specimen from another case.

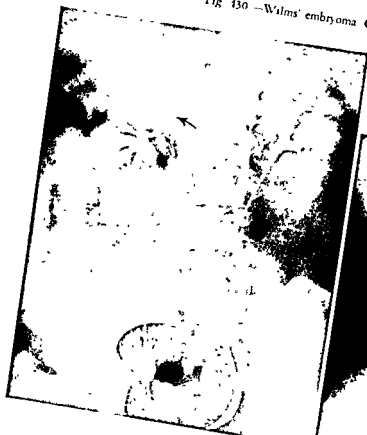
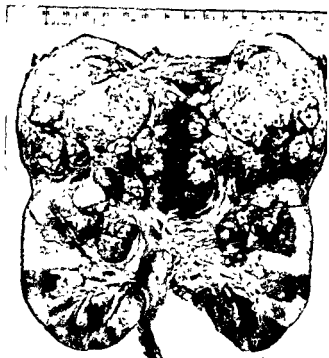


Fig. 431—*A*, Pyelogram showing gross deformity in upper portion of right kidney, typical of either kidney tumor or cyst
B, Retrograde pyelogram showing enlarged left kidney with filling defect in lower pole, typical of kidney tumor



A



B

Fig 432 —A, Retrograde pyelogram showing enlarged right kidney with compression of entire pelvis and calyces

B, Gross specimen of carcinoma of upper pole of kidney

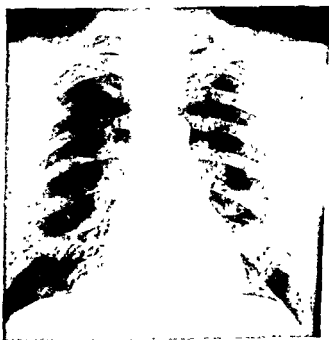


Fig 433 —X-ray of chest showing cannon ball metastases from carcinoma of kidney.

Prognosis.—The prognosis is poor. Without surgical intervention a fatal outcome is inevitable within approximately one year. In cases seen before metastases are present, surgical removal offers a distinct hope of cure. The outcome depends upon an early diagnosis and complete removal of the tumor. In the majority of cases the disease is well advanced before the patient consults a doctor.

Connective Tissue Tumors.—

BENIGN —

1. *Fibroma.* Two forms occur, the first as small whitish nodules which rarely cause symptoms and are usually discovered at autopsy and the second as large cortical growths which produce symptoms and require surgical treatment. They are rare but may occur at any age, even in children.

2. *Lipoma.* Small multiple masses of lipoid tissue may be present beneath the renal capsule or in the cortical substance. They are rarely larger than 1 cm in diameter.

3. *Leiomyoma.* Leiomyoma is a tumor composed of plain muscle. It may have fibrous tissue mixed with it so that the tumor is in reality a fibroid and identical to a fibroid in the uterus. It rarely reaches sufficient size to produce symptoms.

Sarcomatous degeneration may occur in any of the above tumors but is rare.

4. *Hemangioma.* Hemangiomas are occasionally encountered. Unless they attain considerable size and cause pressure or other symptoms, they are of no clinical importance. Hematuria does not occur unless the tumor erodes the renal pelvis. In the differential diagnosis of essential hematuria or occult hematuria, a minute angioma which may be so small that it is easily overlooked at operation is a possibility to be considered.

MALIGNANT —

Sarcoma is very infrequent in adults. Fifty per cent occur between the ages of 40-60 years. The histopathologic structure varies considerably, but all are highly malignant and invariably terminate fatally regardless of treatment. Spindle cell sarcoma is the most common type, with fibrosarcoma, leiomyosarcoma, and other forms being very rare.

The symptomatology, diagnosis, and treatment are the same as for other parenchymal

Signs and Symptoms of Renal Tumors.—

1. *Hematuria* is the most constant finding and may be the earliest symptom except in Wilms' tumor in children, in whom a mass is usually the first sign. Bleeding is always intermittent and seldom profuse. A single hemorrhage may be followed by months or even years without recurrence.

2. *Pain* in the kidney is the initial symptom in 20% of cases and is present in a large percentage of the late cases. It may be caused by stretching of the renal capsule by the expanding tumor, or it may result from pressure by the mass on neighboring nerves, viscera, or ureter, i.e., hydronephrosis.

3. Although hematuria and pain are the commonest symptoms, loss of weight, weakness, gastrointestinal disturbances, fever, cachexia, and anemia may occur. These are usually late symptoms.

Diagnosis.—The diagnosis of renal tumor is made on (1) history, signs, and symptoms, (2) deformity shown in the pyelogram, usually a filling defect.

Prognosis.—Success in the treatment of renal tumors depends upon their early recognition. The over-all prognosis is poor.

Treatment —

1. Prompt nephrectomy with postoperative radiation is the treatment of choice.

2. In inoperable cases with considerable bleeding, Dicumarol may be given to diminish coagulation and minimize the colic caused by the passage of blood clots.

3. Ligation of the ureter may be carried out if the tumor is inoperable and the kidney is not infected.

Tumors of Renal Pelvis

In a 40-year period at the Royal Victoria Hospital, from 1915-1954, there were 207 patients with kidney tumor operated upon, of which 15 had primary carcinoma of the renal pelvis. In an 8-year period in the Department of Veterans Affairs Hospitals in Canada there were 164 patients with renal tumor operated upon or found at autopsy, of which 18 had primary carcinoma of the renal pelvis.

In the total series of 371 cases, 33 were carcinomas of the renal pelvis, giving an incidence of 7%.

CLASSIFICATION OF TUMORS OF RENAL PELVIS AND URETER

Tissue of Origin

I Epithelial

Gross { Papillary 75%
Solid 25%

Microscopic (Whitlock, McDonald, and Cook 1954)

- 1 Papillary carcinoma, Grades 1, 2, and 3
- 2 Papillary and infiltrating carcinoma, Grades 2, 3, and 4
- 3 Nonpapillary infiltrating carcinoma, Grades 2, 3, and 4 (including transitional cell carcinoma, squamous cell carcinoma, and adenocarcinoma)

Note 1 Structural type (i.e., papillary or non-papillary)
2 Presence or absence of infiltration
3. Cell type transitional or squamous
4 Degree of anaplasia

II Mesodermal

Benign fibroepithelial polyp

- Myoma
- Lipoma
- Neurofibroma
- Angioma

Malignant

- Fibrosarcoma
- Myosarcoma
- Lymphoblastoma

III Endometriosis

IV. Metastatic

Carcinomas of the renal pelvis arise from the epithelium which is similar morphologically to that of the ureter and urinary bladder. The epithelium of the renal pelvis is stratified and does not exhibit keratinization or cornification. It is therefore called transitional in type. Certain stimuli, such as chronic infection and stones, may cause the epithelium of the bladder, ureter, and pelvis of the kidney to cornify (leukoplakia), while under other circumstances, such as in extrophy of the bladder, cystitis, ureteritis, and pyelitis glandularis, the epithelium assumes a glandular form. These changes are precancerous conditions.

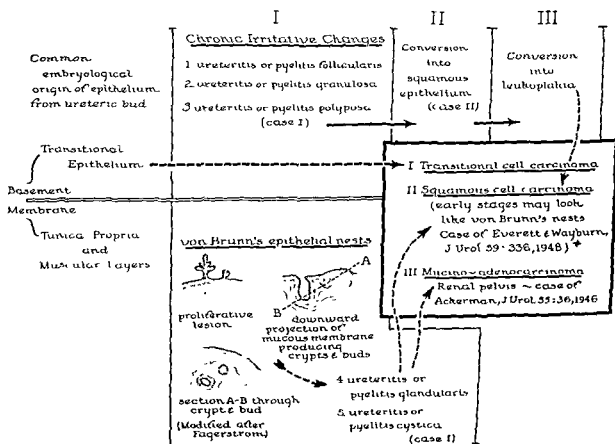


Fig 434—Diagram showing changes that may occur in metaplasia in the urinary tract. MacLean and Fowler have reported a case of squamous cell carcinoma of the renal pelvis and one of mucinoadenocarcinoma

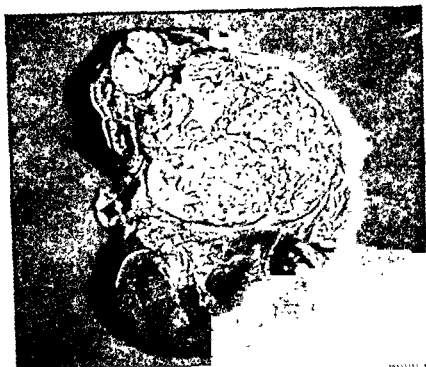


Plate 37.—Renal Carcinoma in Upper Pole of Kidney.

Courtesy Marshall, Victor F.: The Diagnosis of Genito-Urinary Neoplasms, American Cancer Society, Inc.

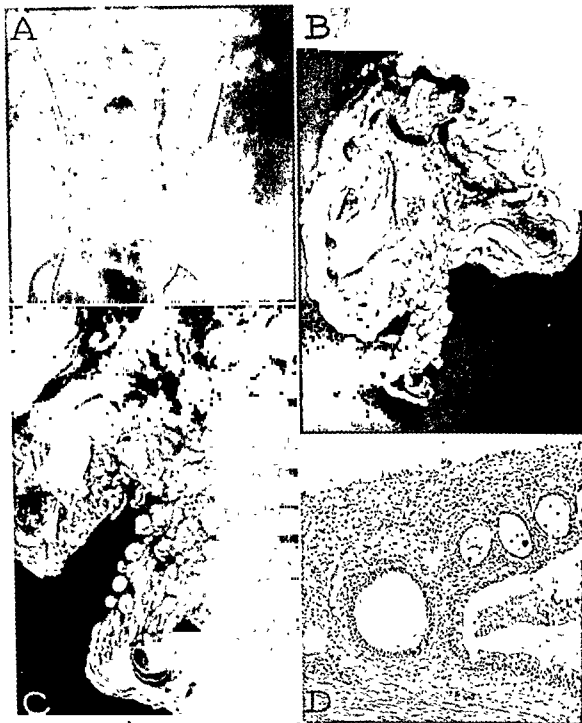


Fig 435 —A, Retrograde pyelogram showing typical filling defects in the left ureter caused by cysts. Note partial staghorn calculus in left ureter.

B, Gross specimen showing staghorn calculus in situ and grapelike cluster of cysts in ureter and pelvis.

C, Close-up view of gross specimen showing cysts.

D, Microscopic section of ureteritis cystica.

(A, C from MacLean, J. T. *Brit J Urol* 26, 127-138, 1954.)

Papilloma and Papillary Carcinoma.—These tumors are usually multiple, appearing as villous growths which are very vascular and bleed easily. They may only involve a small area or be extensively distributed over the mucosa of the pelvis, ureter, and bladder.

Microscopically the papilloma consists of branching processes containing minute blood vessels, covered with many layers of transitional epithelium. The tumor cells are cuboidal or cylindrical. Round cell infiltration is present in the stroma. The structure is typical and uniform. Though usually benign, it has a tendency to become malignant. Once the tumor invades and infiltrates the submucosa and connective tissue, it becomes a true papillary carcinoma.

Hematuria is an early symptom because of the marked vascularity of the papillary processes.

Metastases occur in the ureter and bladder in over 50% of cases by direct seeding. It is

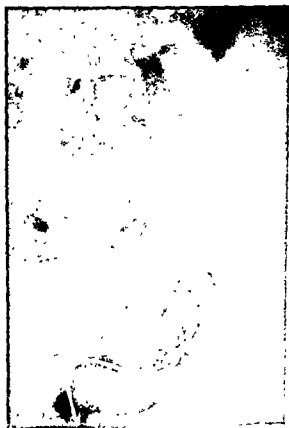
therefore imperative that the entire ureter be removed along with the kidney.

These growths may cause various degrees of hydronephrosis, depending upon the site and degree of obstruction.

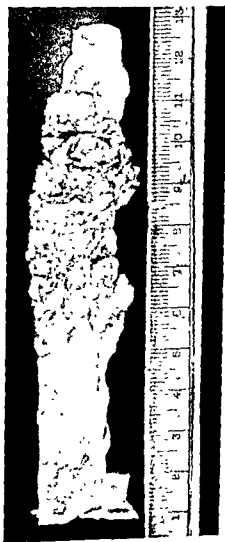
Nonpapillary Tumors.—

1. *Adenocarcinoma* is a large, infiltrating, highly malignant type of tumor which metastasizes rapidly to any part of the body. It is regarded as being a far-advanced papillomatous growth which has lost its papillary character and has assumed an alveolar or scirrhous appearance.

2. *Squamous cell carcinoma* is a highly malignant tumor which has an insidious onset and runs a rapid and fatal course. It is rare



A.



B.

Fig. 436—A, Pyelogram showing catheter curled up in left lower ureter.
B, Gross specimen of case in A, showing tumor of mid-ureter

Chronic infection, leukoplakia, and calculus are frequently associated with squamous cell carcinoma of the renal pelvis and are believed to play an important role in its causation.

The squamous pelvic tumor is a grayish, indurated, infiltrating growth which extends over a limited portion of the mucosa. It is large, flat, and ulcerated, but occasionally tends toward a papillary form.

Microscopically the squamous qualities are pronounced. There is usually cornification and pearl formation. These tumors rapidly invade the surrounding structures, including the kidney. The prognosis is poor. There has been no 5-year cure reported.

Signs and Symptoms.—These are the same as in other tumors. Hematuria is the most frequent symptom.

Differential Diagnosis.—The conditions to be excluded in the differential diagnosis are renal tuberculosis, renal calculus, pyelonephritis, and tumors of the renal parenchyma.

Treatment.—Treatment is nephroureterectomy, plus the treatment of any seeding present in the bladder.

PRIMARY CARCINOMA OF THE URETER

Ureteral tumors closely resemble bladder tumors in their pathologic characteristics.

At the Royal Victoria Hospital, in the 30-year period from 1925-1954, inclusive, there were 4 patients with primary carcinoma of the ureter operated upon. In a 22-year period from 1932 to 1954, there has been one case of primary carcinoma of the ureter found in 10,223 successive autopsies.

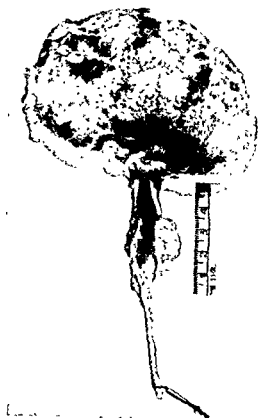
There are two main groups: (1) papillary—more common and (2) nonpapillary.

They occur most commonly in the 4th to 6th decades and are more common in the lower third of the ureter.

Although ureteral tumors are quite similar to bladder tumors pathologically, metastases from ureteral tumors occur earlier and more



A.



B.

Fig. 437—A, Ureterogram showing complete obstruction to the passage of dye beyond the level of the 3rd lumbar vertebra.

B, Gross specimen of case in A, showing solid tumor in upper ureter at site of obstruction.

frequently. This may be due to the insufficient barrier offered by the thin wall of the ureter as compared to the thick wall of the bladder and to the richer lymphatic drainage of the ureter

Signs and Symptoms of Tumors of the Ureter.—Hematuria is the commonest symptom. Pain along the course of the ureter may occur. A mass along the course of the ureter may be palpable

Diagnosis.—The diagnosis is made on the history of physical signs and deformity shown in the pyelograms or ureterograms.

Treatment.—This consists of nephroureterectomy where possible. If this is not possible, intraureteral radium or deep roentgen therapy may be used

Prognosis.—The prognosis in ureteral tumors is good if they are diagnosed early.

PYOGENIC INFECTIONS OF THE URINARY TRACT

Normally the urinary tract is sterile in both the male and the female, with the exception of the terminal 1 cm of the urethra. When infection occurs repeatedly or persists, there is usually urinary stasis due to some degree of obstruction to the normal flow of urine. The obstruction must be relieved in order to cure the infection. Where there is paralysis of the bladder, as in the paraplegic patient, repeated and recurrent attacks of infection are common.

The infection may reach the urinary tract from the following

1. Blood stream—hematogenous
2. Lymphatics—lymphogenous
3. Direct extension from other organs, e.g., abscess
4. The exterior—(a) trauma and (b) gonococcus

When the kidney is attacked, its function is impaired and the organ is permanently damaged. Infections of the skin, bone, intestine, throat, teeth, and cervix may act as foci of infection for the urinary tract.

Pyogenic infections of the urinary tract are due to invasion of the kidney, ureter, or bladder by the organism itself rather than by a toxin. The organism causes an inflammatory reaction in the interstitial tissue, whereas the toxin acts on the glomerulus producing a glomerulonephritis.

Diagnosis.—Certain criteria must be met to establish an accurate diagnosis. Pyogenic organisms must be present. Failure to examine the specimen for organisms is the commonest cause of error in diagnosis. The specimen examined should be freshly voided in the male and be a catheterized specimen in the female. It should be collected in a sterile container.

If organisms are found, they may then be assumed to come from the patient. The prognosis depends largely upon the etiology. In a male patient, if there is any doubt as to whether a urethritis may be producing the pus in the urine, a three-glass urinalysis should be done.

Three-Glass Urinalysis.—The patient is asked to void into three separate containers. The first containing 1-2 ounces represents urethral washings. The second also containing 1-2 ounces is discarded. The third specimen may therefore be regarded as truly representing the conditions in the bladder. If the first specimen shows 40 pus cells per high power field and the third specimen only 2 or 3, the patient obviously has a urethritis. If, however, the third specimen shows just as many pus cells as the first specimen, then the patient must have an infection either in the bladder or at a site higher up in the kidney.

Bacteriology.—The infecting organisms in the order of frequency are as follows:

- | | | |
|--|--|------------------------------|
| 1 | <i>E. coli</i> (<i>Escherichia coli</i>) | 42% |
| 2 | Other gram-negative bacilli | 30% |
| Roughly 70% are due to gram negative bacilli | | |
| a | <i>Aerobacter aerogenes</i> | (<i>B. coli aerogenes</i>) |
| b | <i>Pseudomonas aeruginosa</i> | (<i>B. pyocyaneus</i>) |
| c | <i>Pseudomonas fluorescens</i> | (<i>B. fluorescent</i>) |
| d | <i>Proteus vulgaris</i> | (<i>B. proteus</i>) |
| 3 | <i>Staphylococcus pyogenes</i> | 12% (20%) |
| a | Micrococci | 8% |
| 4 | Mixed organisms | |
| 5 | Other types | |
| a | Gram-positive bacilli | |
| b | <i>Streptococcus faecalis</i> (Lancefield D streptococcus) (synonym— <i>Enterococcus</i>) | |
| c | Diphtheroids | |
| d | Gonococci (<i>Neisseria</i>) | |

The bacteriology of urinary tract infections appears to be changing. Staphylococcal infections now seem to be more prevalent than *E. coli* infections in some areas of the country.

From a clinical point of view an elaborate process of staining is undesirable and unnecessary as nearly all the bacillary infections are gram-negative and all the coccal infections, with the exception of gonococci, are gram-positive. A simple methylene blue stain is therefore adequate to determine whether one is dealing with a bacillary or coccal infection.

Following are the natural groups:

1. Pyelonephritis only
2. Groups under 15 years of age
3. Groups complicated by renal calculi
4. Associated with pregnancy
5. Cystitis only
6. Cystitis in the female associated with narrowing of the urethra
7. Associated with overgrowth of the prostate
8. Occurring in fractured pelvis, ruptured bladder, and cord bladder

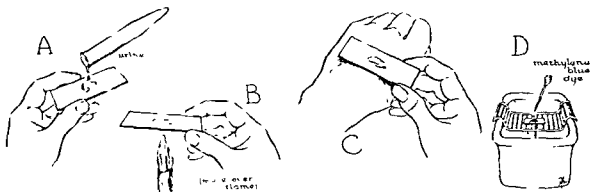


Fig. 438.—Method of preparing a methylene blue stain on urinary sediment. The slide should not be made any hotter than the back of the hand can stand, otherwise the bacteria will be destroyed.

In staining, the slide is warmed over a Bunsen burner but is not allowed to get any hotter than the back of the hand can readily stand, otherwise the bacteria are deformed or destroyed and do not take the stain properly.

The infection may reach the kidney by the blood stream, by lymphatic spread, or by regurgitation up the ureter. The kidney is permanently damaged and its function impaired. Infections of the skin, bone, intestine, throat, teeth, and cervix may act as foci of infection for the urinary tract.

Classification of Urinary Tract Infections.—Cases of urinary tract infection, either pyelonephritis or cystitis, fall into certain natural groups by virtue of the fact that the etiology, pathology, and prognosis differ greatly. Once a case is diagnosed and the group to which it belongs is determined, one can then predict with a high degree of accuracy what the prognosis will be for that particular patient.

SPECIAL FEATURES OF SOME URINARY TRACT INFECTIONS

Pyelonephritis

Acute

In acute pyelonephritis, one or both kidneys may become involved in the inflammatory process. Pyelitis alone almost never occurs, there being an associated pyelonephritis in 99% of cases. This is evidenced by the finding of an acute inflammatory reaction in the interstitial tissue similar to that found elsewhere in the body. The interstitial tissue and tubules are filled with polymorphonuclear leukocytes.

The infection may initially reach the kidney by (1) the blood stream (hematogenous), (2) the lymphatics, and (3) direct ascent up the ureter (urogenous). The latter may occur in neurogenic bladder associated with atonic, neurogenic ureters (reflux up the ureters).

It is probable that most renal infections are hematogenous.

Symptoms.—The patient usually complains of pain or a sense of fullness in the affected kidney. There is a high spiking temperature, and the patient looks toxic. Bladder symptoms of frequency and urgency are usually present when the bladder is involved.

Diagnosis.—A diagnosis is made upon the symptoms described and upon the presence of tenderness of the affected kidney to palpation or tenderness in the costovertebral angle, plus the finding of pus in the urine.

Treatment.—The treatment consists of keeping the patient flat in bed with only one pillow so as to provide better drainage for the kidneys (The kidneys have a normal excursion of approximately $1\frac{1}{2}$ vertebrae. When the patient is in the sitting position they tend to "drop" this normal amount, the ureter bending or folding in an accordion-like manner to take up the slack. Frequently the bend is at the ureteropelvic junction. When the patient is kept flat in bed, the kidney "drops" back into its normal position, and the ureteropelvic junction and ureter are straightened out, thus providing better drainage of the kidney, in spite of the fact that the pelvis of the kidney is at a slightly lower level than the bladder. This position is maintained for only a few days to a week and thus does not increase the tendency to stone formation, described as occurring in "prolonged decubitus.") This is continued throughout each 24-hour period, the patient being allowed to turn on his side to eat but not allowed to sit up. Fluids are given in large amounts. The appropriate chemotherapeutic agent or antibiotic is given. Large, hot linseed poultices are applied to the loin for the relief given if required. Rarely, a ureteral catheter has to be passed to the renal pelvis and left there for a few days to provide adequate drainage.

Chemotherapy.—There are several chemotherapeutic agents, and every effort is made to select the one effective against the organism involved. Any one drug is continued for a period of not longer than 10 days in order to avoid renal irritation. One would anticipate that by culturing the urine and determining the sensitivity to the various drugs of the organism obtained, it would be relatively easy

to eradicate the infection by using the appropriate drug. However, this frequently has not been the author's experience. For example, one may treat a patient with an *Aerobacter aerogenes* infection sensitive to Chloromycetin with large doses of Chloromycetin for a period of 4-5 days and at the end of that time obtain a culture of the same organism with the same degree of sensitivity. For this reason the author has only a moderate confidence in the *sensitivity test*. One must also bear in mind whether there still exists obstruction to the urinary flow and the fact that it is not uncommon for the bacterial flora to change completely during treatment, so that one is no longer dealing with *Aerobacter aerogenes* but some totally different organism such as staphylococcus. This in turn may be an indication for a change in therapy.

In the presence of urinary tract obstruction, calculi, or foreign body (urethral catheter, suprapubic tube, or ureteral splint), sterilization of the urinary tract cannot be effected by the usual doses of antibiotics.

The drugs most commonly used are a follows:

1. Mandelamine
2. Mandelic acid and Urotropin
3. Sulfonamides—(sulfathiazole and Gantrisin are the two most effective)
4. Penicillin
5. Streptomycin
6. Antibiotics

A. *Three antibiotics all derived from streptomycetes:*

- a. Aureomycin
- b. Chloromycetin (chloramphenicol)
- c. Terramycin

B. *Antibiotics derived from bacteria*

- a. Tyrothricin
- b. Bacitracin
- c. Polymyxin (Aerosporin)

C. Erythromycin (Iliotycin)

D. Tetracycline (synonyms: Achromycin, polycycline), a broad-spectrum antibiotic, effective against both gram-positive and gram-negative organisms

E. Furadantin—(Nitrofurantoin) effective against gram-negative organisms

Summary of effective drugs in order of preference, according to organism involved

1. Any gram negative bacillus—98% of all bacterial infections
 - a Uramand
 - Gantrisin
 - Trisulla
 - b Tetracyclin or Chloromycetin
 - c Streptomycin
 - d. Combinations of above
- 2 Any coccal infection (staphylococcus, streptococcus)
 - a. Genticillin
 - b Erythromycin
 - c Tetracycline
 - d Combinations of above
- 3 Very resistant organisms
 - A Proteus
 - a Chloromycetin
 - b Gantissin
 - c Furadantin
 - d Tetracycline and streptomycin
 - B Pseudomonas
 - Polymyxin
 - C Any other resistant organism
 - a Furadantin
 - b Combinations of the above groups

For further information see Chapter 3

Complications of Chemotherapy—It must be remembered that certain drugs carry with them the risk of injury when administered in clinical amounts. It is for this reason that antibiotics are administered for only 4-5 days at a time and other drugs for a maximum of 10 days.

Complications of Treatment—

- 1 Anaphylactic reaction, e.g., penicillin
- 2 Dermatitis, e.g., penicillin, sulfa drugs
- 3 Anuria, e.g., sulfa drugs
- 4 Paralysis and renal damage, e.g., polymyxin
5. Eighth nerve damage, e.g., streptomycin

Sequelae.—With the above regime, the acute infection usually quickly subsides. Resolution may become complete, and healing of the inflammatory process with scar tissue replacement occurs. However, one of the most characteristic features of pyelonephritis is its tendency to recur. With each successive attack

the interstitial tissue of the kidney progresses to a chronic inflammatory state, so that eventually a chronic pyelonephritis becomes established.

Chronic

Chronic pyelonephritis is a diffuse infection of the kidney or kidneys which may last for months or years. During this period, acute recurrences of the infection may take place. In the chronic stage the interstitial tissue is filled with lymphocytes, only a few polymorphonuclear leukocytes being present. Colloid



Fig. 439.—Microscopic section showing intense interstitial tissue reaction of pyelonephritis

casts are present in the tubules which are lined with atrophic epithelium. Periglomerular fibrosis is also present. With each attempt of the kidney to heal, more scar tissue forms, eventually leading to impairment of renal function. The symptoms are those of recurrent infection and pyemia. The treatment is the same as for an acute infection.

Occasionally one will find on routine pyelography a pyelogram which is typical of chronic pyelonephritis, yet the patient ...

deny ever having had any kidney trouble at any time. The pyelogram will show dilated, blunted calyces, permanently fixed in that position by the surrounding fibrosis.

Pyelonephritic Atrophy

This process of recurrent exacerbations of infection, followed by recession and the laying down of scar tissue, may eventually produce a small contracted kidney referred to as *pyelonephritic atrophy*. Renal function may be so impaired by the large amount of scar tissue present that renal failure ensues. The condition may be unilateral or bilateral.

Necrotizing Renal Papillitis

Necrotizing renal papillitis or *necrotizing pyelonephritis* is a severe type of renal infection that is encountered usually in diabetics. Latent infections in the urinary tract may serve as the foci for the development of more severe lesions at a later date. Pathologically the kidney shows multiple small abscesses in the renal pyramid about the level of two thirds of the way from the papilla to the cortex. As the abscesses coalesce, complete necrosis of the terminal two thirds of the papilla occurs. The process usually involves all the pyramids of the affected kidney. It may be bilateral.

The onset may be fulminating, in which case the patient suddenly becomes desperately ill, or occasionally the process may be slightly less rapid, although equally violent in effect. High spiking temperature, prostration, and rapid pulse are present without signs or symptoms suggestive of urinary tract involvement. The course is rapidly fatal. A correct diagnosis is rarely made before death.

Pyonephrosis

Pyonephrosis refers to an enlarged kidney containing pus. As any inflammatory condition in the kidney progresses over a prolonged period, the delicately defined contour of the calyces is lost. They become club-shaped and gradually increase in size at the expense of the cortex which is thinned out. In a well-developed case the outside of the kidney appears lobulated, the lobulations corresponding

to the thinned-out soft cortex overlying distended calyces. The products of inflammation (pus) accumulate within the kidney. The kidney eventually becomes a mere pyonephrotic sac (pus sac) and is almost if not entirely nonfunctional. If the ureter is open, there is purulent drainage from the sac.

The treatment of pyonephrosis is nephrectomy.

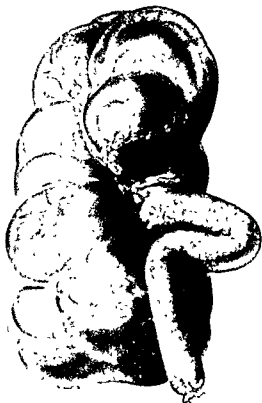


Fig. 440—Pyonephrosis

Group of Infections in Children Under 15 Years of Age

Pyogenic urinary tract infections in children show many features in common. The symptoms are more often referred to the gastrointestinal than to the urinary tract.

1. Response to treatment varies directly as the length of time the symptoms existed before treatment is started.
2. Twenty-five per cent of these patients have recurrent attacks of pyelonephritis 1 to 10 times.
3. Twenty-five per cent have an acute upper respiratory infection immediately preceding the urinary tract infection.

4. Twenty per cent have a congenital lesion in the urinary tract, e.g., aberrant renal vessel, valves in the urethra, etc.

5. Thirty per cent require a major surgical procedure such as a plastic procedure on the renal pelvis, or a nephrectomy, before a cure can be effected.

The Group Complicated by Renal Calculi

1. The infection cannot be cleared up until the calculus is removed. Only 50% succeed in clearing up their infection completely.

2. Thirty per cent develop recurrence of infection.

3. Forty per cent eventually require nephrectomy.

5. Twenty-five per cent are cured during the pregnancy.

6. Ninety per cent are cured within two weeks of emptying the uterus.

Endocrinologic Aspects of Pyelitis of Pregnancy.—The exact cause of dilatation of the ureter during pregnancy is unknown. Some observers believe it is due to the weight of the gravid uterus; others believe that it is due to hormonal factors.

During pregnancy the gonadotropic hormone reaches a maximum excretion in the urine on the 56th day, and thereafter declines rapidly. At the 3rd month of pregnancy the placenta takes over the function of producing and secreting estrin and corpus luteum hor-

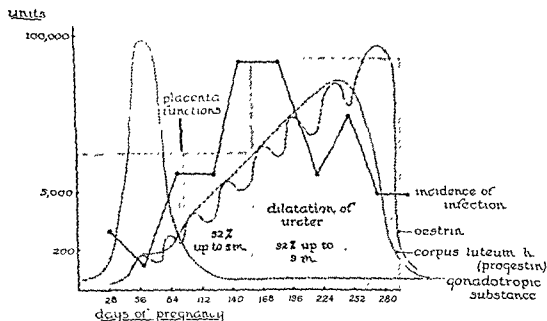


Fig. 141.—Relationship between pyelonephritis of pregnancy, hormone excretion in urine, and dilatation of ureter.

The Group of Infections Associated With Pregnancy

1. The incidence of infection diminishes with each successive pregnancy.

2. Two thirds of all the infections occur in the first and second pregnancies.

3. Eighty-five per cent of the patients can be carried to term by conservative treatment.

4. Ninety per cent of these can be delivered of living babies.

mones. This excretion in the urine increases progressively throughout pregnancy.

The incidence of dilatation of the ureter in pregnancy is 52% up to the 5th month of pregnancy, and 92% up to the 9th month. If the incidence of dilatation of the ureter is charted on the graph, it is found that its mean parallels exactly the increased excretion of estrin and corpus luteum hormones during pregnancy.

If one now charts on the graph the incidence of pyelonephritis during pregnancy, it is found that it parallels exactly the incidence of dilatation of the ureter, and both parallel the increased excretion of estrin and corpus luteum hormones in the urine as pregnancy advances.

(It has been shown in the Rhesus monkey that if the fetus is removed from the uterus and the placenta left in place, dilatation of the ureter continues. This is what one would expect, since dilatation parallels the excretion of estrin and corpus luteum hormones.)

Cystitis Only

1. Treatment consists of chemotherapy and bladder lavage. The drug of choice will depend upon the organism present.
2. Sitz baths are of some value.
3. Stricture of the urethra if present will require dilatation.
4. Prostatitis if present will also require treatment.

Cystitis in the Female Associated With Narrowing of the Urethra

Narrowing of the urethra in middle-aged women is a common finding. There is often an associated cystitis and less frequently a pyelonephritis.

Many of these women are near or have just passed the menopause.

There are four distinct entities which may occur:

1. Narrowing of the urethra with associated cystitis. The patient complains of marked frequency, urgency, and burning on micturition. On catheterization the urethra is found to be tight. There is pus in the urine. Most writers refer to this as stricture of the urethra, which is an unfortunately inaccurate term. What actually happens is that as the menopause is approached, a good many of the elastic fibers in the urethra are replaced by fibrous tissue fibers, so that the urethra loses its elasticity. Here, as elsewhere in the body, fibrous tissue fibers shrink so that the caliber of the urethra becomes relatively narrowed. This in turn produces just enough resistance and obstruction to the flow of urine to cause congestion of the trigone, and it is not long before infection also

develops. These patients respond well to dilatation of the urethra to a Fr. No. 26 sound, bladder lavage, and the proper urinary antiseptic. Stilbestrol may be of help if not contraindicated from a menstrual point of view.

2. In the same category, with the same symptoms except burning but the additional symptom of pain in the bladder, and in any age group (although most do occur near the menopause) is the patient with a markedly reduced bladder capacity, either with or without some narrowing of the urethra. A catheterized or voided specimen of urine will show only a few red blood cells but no pus. However, the basic problem here is not the narrowing of the urethra but the contracted bladder. These patients have an interstitial cystitis in which there is a fibrous adhesion of the mucosa of the bladder to the underlying layers. This was described many years ago by Hunner, who also noted the minute ulcers present. These are infrequently seen.

There have been innumerable treatments devised for this condition, including local excision and fulguration of the area most involved and cortisone therapy. All of these treatments have proved to be remarkably unsatisfactory in curing the symptoms. In the author's experience, the most satisfactory treatment has been the simplest, namely, to stretch the bladder with water under direct cystoscopic vision under general anesthesia. As the bladder is distended from its initial 3 oz. capacity, there suddenly appears a streaking of blood beneath the mucosa, as the adhesions between the mucosa and deeper layers are torn. At this point the dilatation should cease as the bladder capacity will now be between 300-500 ml. Urinary antiseptics should be given prophylactically for 4 days after the dilatation. The patient will now remain asymptomatic for a period of 3 months to 1 year, at the end of which time the dilatation should be repeated.

3. The third group of women have either the interstitial cystitis or the cystitis with the narrowing of the urethra as described, plus a cervicitis or vaginitis. Many of the vaginal infections are due to *Trichomonas* or *Monilia*. Again, unless the cervicitis and vaginitis are eliminated, the urinary symptoms will not disappear. One should bear in mind the possi-

bility of the husband being the cause of re-infection.

Care is taken to exclude an unsuspected carcinoma of the cervix. Cervical smears stained by the Papanicolaou technique are of great assistance.

4. A few of these patients, particularly in the older age group, have, in addition to a tightness of the urethra, the presence of a thickening of the bladder neck, with definitely hypertrophied prongs causing a mechanical obstruction. A resection of the bladder neck is required to remove the obstructing tissue.

Infections Associated With Overgrowth of Prostate

In men in whom the prostate hypertrophies, there develops an obstruction at the bladder neck. As the obstruction increases, the bladder fails to empty completely, so that a residual urine remains. As this urine is more or less stagnant, it is an ideal breeding ground for bacteria. Thus cystitis in a patient with an enlarged prostate is not an uncommon finding. Furthermore, calculi may and often do form in this infected, stagnant urine; also the bladder becomes infected immediately when it is opened and a tube inserted. Following a prostatectomy every patient has a cystitis. Its duration will depend upon the intensity of the chemotherapeutic measures directed at elimination of the infecting organisms. If the infection is not cleared up postoperatively, fibrosis of the bladder neck may occur. As a rule it requires 2-3 months of postoperative care to eradicate the infection. Treatment consists of bladder irrigations once a week and the appropriate chemotherapeutic or antibiotic agent according to the bacteria present.

Infections Occurring in Fractured Pelvis, Ruptured Bladder, and Cord Bladder

Each case is treated as an individual problem according to the principles outlined above.

Perinephric Abscess

Perinephric abscess is an abscess that forms around the kidney and is almost always due to rupture of a cortical abscess. The organism most commonly found in the pus is the staphy-

lococcus, the next most common being *B. coli*. It frequently follows staphylococcal abscesses on the skin or inside the nose.

Symptoms.—The symptoms most commonly found are pain, a sense of fullness in the lumbar region, chills, fever, and prostration.

Physical Signs.—As the abscess increases in size it will spread downward along the psoas muscle and also posteriorly in the loin, causing a bulging with recognizable deformity. The bulging area is tender to palpation and may be fluctuant.

X-ray Signs.—The x-ray signs of a perinephric abscess are usually diagnostic:

1. There is curvature of the lumbar spine with the convexity away from the abscess.
2. Obliteration of the psoas muscle shadow occurs on the affected side.
3. The ribs, or lumbar transverse processes, or both may be obscured.
4. There may be displacement of the colon or fixation of the diaphragm on the same side if the abscess has existed for some time.
5. The kidney may be shown by intravenous pyelograms to be fixed and to lack its normal mobility.
6. There may be anterior displacement of the kidney.
7. In some cases of perinephric abscess, there is lateral displacement of the kidney with medial displacement of the ureter.
8. Pyelograms may show rotation of one calyx, indicating the presence of an abscess.

Diagnosis.—This is made on the symptoms, physical signs, and x-ray findings.

Prognosis.—The abscess will continue to increase in size until such time as it is incised and drained. If it goes unrecognized, rupture into the pleural cavity or into the peritoneal cavity occurs, either of which may be fatal.

Treatment.—If the abscess is recognized early, it may disappear completely with intensive penicillin therapy. If, however, a recognizable mass is present in the loin, incision and drainage must be carried out. Two or three Penrose drains with wicks are inserted to maintain drainage. Penicillin therapy is given. When the acute condition subsides, the functional status of the kidney is determined and a decision made as to whether or not nephrectomy will be required.

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GENTOURINARY SYSTEM

Renal Carbuncle

A renal carbuncle may start by the fusion of several smaller cortical abscesses or by the progressive enlargement of one abscess. As it increases in size it becomes demarcated from the surrounding healthy kidney tissue, and its core becomes necrotic. The carbuncle may involve one whole pole of the kidney or even the entire kidney, together with perinephric fat.

The symptoms are those of an acute infection, with a high temperature and tenderness to palpation in the kidney region.

The carbuncle may rupture into the renal pelvis and empty itself spontaneously via the ureter, or it may rupture into the perinephric space, producing a perinephric abscess.

An acute fulminating infection is treated with intensive penicillin therapy. Nephrectomy

may be required during the acute stage to save the patient's life but, if possible, is deferred. When the acute condition has subsided, nephrectomy will be required if the suppurative process is found to be extensive.

Abacterial Pyuria

Abacterial pyuria is an infection of the urinary tract in which numerous pus cells are found but in which one fails to recover organisms either on direct smear or on culture. These infections respond very promptly to the intravenous administration of arsenicals (neoarsphenamine, Mapharsen). This has led to the belief that the infection is caused by an unidentified spirochete. The prompt response to arsenical therapy has led others to believe that the infection is caused by Leishman-Donovan bodies.

URINARY CALCULI

Theories of Origin

Theories concerning the etiology of urinary calculi are numerous and conflicting. There is no one constant series of events found in all cases presenting calculus formation.

The etiologic factors may be classified as follows:

Personal Factors.—

1. Deficiency of vitamin A
2. Dehydration
3. Prolonged decubitus
4. Bacterial infections
5. Congenital anomalies causing obstruction
6. Hyperparathyroidism

Until recently, deficiency in vitamin A was considered to be of great importance in the formation of urinary calculi. Subsequent studies of patients with calculi, however, revealed that only a very few were actually deficient in vitamin A. This theory has therefore been discarded.

Dehydration appears to be of some significance in the formation of urinary calculi. More cases of patients suffering from colic are seen in the hot summer months than during the winter. In World War II the incidence of calculi in the troops involved in

desert warfare was unusually high. This was thought to be due to dehydration.

It is generally accepted that prolonged decubitus tends to increase the incidence of urinary calculi. When a patient is immobilized for a prolonged period, there is increased mobilization of calcium which is derived from the whole skeleton but especially from the long bones.

There is an increased tendency to stone formation in patients so immobilized. This is considered due in part to increased mobilization of calcium and partly to the effects of stagnation caused by the prolonged supine position. It is interesting to note that, in this position, the renal pelvis is at the lowest level in the urinary tract in the horizontal plane. This means that urine has to pass upward to a higher level to reach the ureter and flow into the bladder. There is thus a natural tendency to stagnation in the renal pelvis when a patient is kept in the decubitus position for any length of time. This particular factor can be overcome by the use of an oscillating bed or by placing a board frame with a central fulcrum under the standard mattress, so as to permit the patient to alter his position.

Bacterial infection is the main causative factor in many urinary calculi. This is confirmed by the fact that infected urine is obtained by catheterization from the kidney involved and that certain types of calculi, e.g., calcium phosphate, form only in infected urine. The organism *B. proteus* is the greatest offender and the most difficult to eradicate. Staphylococci are the next commonest organisms which predispose to stone formation. Usually it is impossible to eradicate infection in a kidney in which a calculus is present until the calculus has been removed. Conversely, unless the infection is eradicated, calculi will almost certainly reform. The incidence of recurrence of renal calculi varies from 10-40% in different clinics.

Patients with *obstruction*, e.g., aberrant renal vessels at the ureteropelvic junction, stricture, etc., are particularly prone to develop calculi. This is due to the effect of prolonged impairment to drainage from the kidney, resultant stagnation of urine, and increased susceptibility to infection.

Approximately 5% of recurrent urinary calculi are due to *hyperparathyroidism*. Hyperparathyroid disease is discussed in Chapter 13.

Race and Geographic Distribution.—The white and yellow races are relatively susceptible to the formation of urinary calculi. The Negro race is relatively immune.

There appear to be definite stone-bearing areas in the world. Urinary calculi are much more common in India and China than in other countries. Just what effect soil and climate have on the formation of calculi is not known.

Theories of Physical Factors Associated With Stone Formation.—Considerable speculation regarding the origin of urinary calculi was based on the results of chemical analysis. In turn, a theory was developed in which the causes were attributed to various physical factors.

1. *The presence of stone-forming substances in the urine.* Every specimen of urine contains stone-forming elements, e.g., uric acid and calcium oxalate. The solubility of these substances depends upon the following.

a. The pH of the urine. At pH 5 pure uric acid is present to the point of saturation. At

pH 7, the urine is supersaturated with calcium oxalate.

b. The presence of other electrolytes in the urine. The solubility of a sparingly soluble electrolyte is increased by the presence of other more soluble electrolytes, e.g., the solubility of uric acid is increased by the presence of sodium phosphate.

c. The presence of certain nonelectrolytes. The solubility of a salt in a solution may be greatly increased by the presence of another salt in the same solution, e.g., the solubility of calcium oxalate in water is doubled by adding urea.

2. *The protective action of urinary colloids.* The urinary colloids consist of mucin, nucleic acid, chondroitin sulfuric acid, and a complex nitrogen-containing carbohydrate. They are suspended in solution, and their precipitation may lead to the formation of the nucleus of a future calculus. The nucleus of the developing calculus will continue to grow provided (a) it is retained within the urinary tract and (b) the urine continues to supply an excess of stone-forming salts.

This theory advocated by Swift Joly and others has considerable appeal. It tends to explain the physical factors concerned but is totally unable to explain many of the other known factors in calculus formation. It is perhaps much closer to the truth to say that the exact cause of calculus formation is not known in all cases, although many of the contributing factors have been recognized.

If a catheter is passed up each ureter to the renal pelvis and specimens of urine are obtained from patients with renal calculi, in whom hypercalcaemia is present, urine from the kidney containing the calculi is found to have an excess of calcium, whereas urine from the other kidney is found to contain a normal amount of calcium. This suggests that in the normal kidney there is some mechanism which holds back calcium and acts as a filter. If a calculus is present, this mechanism may become damaged, allowing an excess of calcium to escape in the urine. Since the normal exchange of calcium and phosphate ions is across the tubular membrane, it is possible that the disease may begin there.

Lymph Channel Origin of Calculi.—Carr has recently enunciated an entirely new theory of stone formation. He has shown that normal kidneys contain radiopaque bodies measuring 0.2 mm. in diameter. Several concretions may coalesce and lie in a line. He has shown that these concretions occur just outside the fornices of the calyces in line with but outside both the arteries and veins, i.e., in the lymphatic channels. As the concretion increases in size, it grows toward the pelvis, finally ulcerating through into it to give a true renal calculus. The so-called "plaques of Randall" he believes are in fact multiple concretions aggregated together in the lymph channels

ward, forward, and medially along the course of the ureter. It may also radiate into the testicle or the medial aspect of the thigh on the affected side.

There are three points of narrowing along the course of the ureter. A stone is likely to become lodged at these points of constriction: (1) the ureteropelvic junction, (2) the point at which the ureter crosses the iliac vessels, and (3) the entrance of the ureter into the bladder.

A sharp or irregular stone may become embedded in the mucous membrane of the ureter anywhere along its course.

TABLE 34
CLASSIFICATION OF CALCULI

	URINE		CHEMICAL COMPOSITION	MINERALOGIC NAME
	ACID	ALKALINE		
A Organic				
1 Uric acid calculi				
a Uric acid	+			
b Urates		+		
c Xanthine	+			
2 Cystine calculi	+			
B Inorganic				
1 Primary calcium calculi				
a Calcium oxalate monohydrate	+		$\text{CaC}_2\text{O}_4 \cdot \text{H}_2\text{O}$	Whewellite
Calcium oxalate dihydrate	+		$\text{CaC}_2\text{O}_4 \cdot 2\text{H}_2\text{O}$	Weddellite
b Calcium carbonatophosphate		Neutral to alkaline	$\text{Ca}_3(\text{PO}_4)_2 \cdot 2\text{CaCO}_3$	Carbonatapatite
c Calcium phosphate		Neutral to alkaline	$\text{CaHPO}_4 \cdot 2\text{H}_2\text{O}$	Brushite
2 Magnesium-containing calculi (May also contain carbonate and phosphate and magnesium ammonium phosphate)		+	$\text{MgNH}_4\text{PO}_4 \cdot 6\text{H}_2\text{O}$	Struvite

Pathology

Most calculi are due to obstruction, bacterial infection, or ulceration, either alone or in combination. Some form as plaques on the renal papillae without evidence of any of the above factors.

Symptoms of Renal and Ureteral Calculi

A calculus in the kidney may be large and silent. A stone weighing 432 Gm. (1 pound) was removed from a patient who had no symptoms referable to the kidney. Small calculi produce attacks of acute renal or ureteral colic as they pass down the ureter. The pain begins in the lumbar region posteriorly, is sudden in onset, colicky in nature, and passes down-

Diagnosis of Renal or Ureteral Calculus

1 History of acute pain in the loin, sudden in onset, colicky in nature, radiating downward, forward, and medially into the bladder, testicle, or medial aspect of the thigh on the affected side. The pain may be so severe as to cause collapse.

2 On physical examination, there is splinting of the muscles on the affected side and tenderness to palpation.

3. The urine usually shows scattered red blood cells. It should be remembered that a voided specimen of urine is desirable in these cases, as the passage of a catheter may in itself be sufficiently traumatic to produce scattered red blood cells in the urine.



Fig. 442—Plain film of kidney ureter bladder area showing fairly large calculus in left mid ureter opposite transverse process of 3rd lumbar vertebra

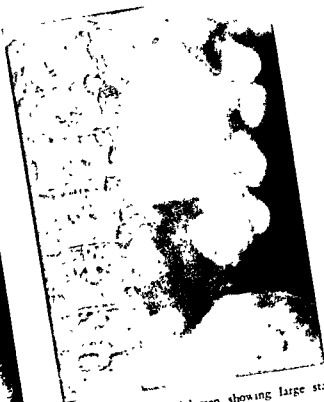


Fig. 443—X ray of abdomen showing large stag horn calculus in the left kidney.

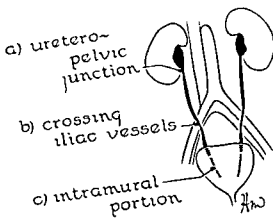


Fig. 444—The three points of narrowing of the ureter

4 A plain x-ray plate of the kidney, ureter, and bladder area will reveal the site, size, and number of calculi present if they are radiopaque. Approximately 5% are nonopaque.

5. Cystoscopic examination and ureteral catheterization are performed. X-rays taken in the anteroposterior and oblique diameters with the catheter in situ will show conclusively whether or not the calculus is in the ureter. If a calculus is present in the ureter, the opacity seen on x-ray will remain in contact with the catheter in both the anteroposterior and oblique projections. However, if the opacity is seen to lie one-half inch or so away from the catheter in either projection it can be said with certainty that the opacity is not due to a calculus in the ureter, regardless of the other symptoms present. In the latter case the shadow may be due to a calcified gland or phlebolith.

Treatment

- 1 Emergency relief of the colic by giving morphine 15 mg and atropine 0.4 mg
- 2 Prevention—try to correct the underlying condition if possible
- 3 Surgical—the particular type of surgical procedure to be employed depends upon the conditions present and may be any of the following

Operations on the Kidney.—

- 1 Remove the calculi—x-ray at the time of operation to make certain all the calculi are removed
 - a Pyelotomy—i.e., open the pelvis of the kidney and remove the calculus
 - b Nephrolithotomy—i.e., approach the stone through the substance of the kidney
- 2 Remove obstructions at the time of the pyelotomy or nephrolithotomy, e.g., if uretero-

pelvic obstruction is present do a plastic procedure at the site of obstruction

3. Nephrectomy—if the disease is advanced Operations on the Ureter.—

1. The ureteral orifice and ureter may be dilated with bougies
2. Or be cut on the affected side with cystoscopic scissors or a cautery wire.
- 3 If the calculus is low in the ureter and not too large, a stone basket may be passed and the calculus removed. Serious trauma to the ureter may be caused by rough handling of a stone basket. An edematous seminecrotic ureter may be perforated, the infection thereby being spread outside the ureter into the retroperitoneal plane with the production of a perinephric abscess
4. The ureter may be incised over the calculus and the calculus removed

Dietary.—

1. Vitamin A is said to be of some value, but this is doubtful.
- 2 Cystine stones can be prevented and perhaps even reabsorbed by (a) reducing proteins in the food to a minimum or (b) by giving alkalies to keep the urine constantly alkaline
- 3 Attempts have been made to dissolve calcium and phosphate calculi by using buffered citric acid solutions as irrigations (solutions G and M). In our experience these have not been successful
- 4 Some authors believe that the presence of citric acid in the urine increases the solubility of the calcium salts. Estrogenic hormone is given to increase the excretion of citric acid in the urine, thereby increasing the solubility of calcium. Aluminum hydroxide gel is given at the same time, to increase the elimination of phosphates by the bowel and diminish the amount available to the kidney

GENITOURINARY TUBERCULOSIS

Genitourinary tuberculosis may affect the kidney, prostate, seminal vesicles, epididymes, fallopian tubes, or bladder. It may be limited to any one of these organs, with the exception of the bladder. Its most characteristic feature is its marked tendency to spread and this plays the predominant role in deciding the treatment.

It must also be borne in mind that tuberculosis is a generalized infection and that urogenital tuberculosis is only a local manifestation. Such infection is usually secondary to a primary focus in the lungs, intestinal tract, tonsils, or bones. The primary focus can be found in approximately 80% of cases. In

searching for a primary focus, one should also bear in mind that an early pulmonary lesion may not be detectable by radiologic means until 6 months after its onset.

Age.—Genitourinary tuberculosis is rare under the age of 10 years and, if present at this age, is almost always of the acute fulminating type. Seventy per cent of cases occur between 20-40 years of age.

Incidence.—The incidence of genitourinary tuberculosis is approximately 1.2% in routine autopsies, 5% in autopsies on tuberculous patients dying of other diseases, and 10% in patients dying of tuberculosis.

The incidence of the bovine type in genitourinary tuberculosis is exceedingly low.

In cases of genitourinary tuberculosis, the prostate, seminal vesicles, or epididymis is involved in 65-75% of the cases. Primary tuberculosis of the prostate gland alone is rare.

Renal Tuberculosis

Renal tuberculosis is the result of the introduction, implantation, and germination of the tubercle bacillus in the renal bed. The extent of the lesion depends upon the virulence of the bacillus and the resistance of the host.

There are three main types of lesion in the kidney:

1. **Acute miliary**—fulminating bilateral renal lesions, which are part of a generalized miliary tuberculosis.

2. **Chronic form**, in which there is abscess formation with caseation, referred to as surgical renal tuberculosis.

3. **Toxic tuberculous nephritis**, which is a renal manifestation of grave systemic tuberculosis, without definite involvement of the urinary organs; exceedingly rare.

Etiology.—Renal tuberculosis is always hematogenous in origin, the primary focus being in the lungs, tonsils, intestinal tract, or bone. The infection in the primary area progresses until the tubercle bacilli finally invade the blood stream. If the invasion of the blood stream is massive, general miliary tuberculosis results. If the organisms enter the blood stream in showers, the phagocytic cells of the body may overcome them. If the patient's general and local resistance is sufficient, the secondary

lesion in the kidney may heal; otherwise it progresses with eventual destruction of the kidney. Renal and other secondary lesions in the prostate, seminal vesicles, and epididymis may all originate from the same primary focus in the lungs, tonsils, or intestinal tract. Conversely, the renal lesion may be derived from infection in the prostate, seminal vesicles, or epididymis, and carried to the kidney by the blood stream. It is the consensus that renal tuberculosis is blood borne and that tuberculous infection ascending the ureter or the lymphatics does not occur.

Pathogenesis.—It is now generally accepted that a bacillus-laden embolus is carried into the kidney by the blood stream and lodges in a capillary tuft. The glomerular focus may be walled off or a few bacilli may reach the capsular space and multiply while being washed slowly along the proximal convoluted tubule until they reach a favorable soil for growth in the medullary loop. The bacilli may, however, reach the medulla by way of the fine efferent glomerular capillaries. The final method of infection is by spread of cortical lesions to the medulla by the lymphatics.

Opinions differ as to whether the initial lesions in the kidney are predominantly cortical or medullary. Medlar, who did 100,000 sections on 44 separate tuberculous kidneys, found 367 definite tuberculous lesions of which 75% were in the cortex, 11% were in the medulla, and 14% were corticomedullary.

The initial glomerular lesion may heal, continue to grow slowly, or rarely develop into the main lesion. The medullary lesion usually grows relatively fast. Such a tubercle will spread and finally open into a renal tubule or papilla, discharging its contents into the renal pelvis. If the medullary lesion is near a calyx, it will give rise to early symptoms. If it is deep in the parenchyma there may be extensive damage before there are any clinical signs. Healing of this type of lesion is rare.

The Question of Initial Bilateral Involvement.—Of the many disputed points concerning renal tuberculosis none has been more discussed than the question as to whether, at the outset, one or both kidneys are involved. Many investigators believe the implantation is bilateral but that the disease develops in only one kidney.

The Excretion of Tubercle Bacilli in the Urine.—The presence of tubercle bacilli in the urine represents a break in the integrity of the renal mucous membrane and is regarded as the essential evidence of disease within the renal parenchyma.

Secondary infection occurs frequently. In urine obtained from a tuberculous patient organisms other than tubercle bacilli, e.g., *E. coli*, staphylococci, are found in 20% of cases. It is therefore important to exclude tuberculosis in all cases of persistent urinary infection.

Symptoms of Renal Tuberculosis.—

1. Frequency, day and night, is present in 75% of the cases.

2. Painful urination is often present.

3. Hematuria, microscopic or macroscopic occurs in 50% of the cases

a. Terminal bleeding common

b. Smoky urine

c. Microscopic red blood cells

d. Gross hematuria rare

4. Dribbling is usually a sign of pronounced vesical involvement.

Diagnosis.—

1. History.

2. The recovery of tubercle bacilli from the urine is the essential evidence of active disease within the renal parenchyma (a) by direct smear, (b) positive urine cultures, (c) guinea pig inoculation of urine from each kidney

3. Retrograde pyelograms demonstrating alterations in the renal architecture

Treatment of Renal Tuberculosis.—The incidence of bilateral renal tuberculosis will vary, depending upon the promptness with which observation and urologic investigation are undertaken. It is bilateral in 20-45% of cases. It is of the greatest value to know the patient's ability to fight his disease, the pathologic type of the disease, and the prognosis insofar as the pulmonary lesion is concerned.

The use of streptomycin with para-aminosalicylic acid has considerably altered the technique of treating renal tuberculosis. The recent addition of isoniazid has likewise improved the treatment. Isoniazid is 600 times more effective than para-aminosalicylic acid alone and is as bacteriostatic as streptomycin against the tubercle bacillus. However, like streptomycin, the tubercle bacillus develops a resistance to it in approximately 2-8 weeks.

Therefore the current use of these three drugs, each of which has a different mode of action on the tubercle bacillus, is as follows and for a period of 2 years:

a. Isoniazid—3 mg./kilo body weight in 3 divided doses daily

b. Streptomycin hydrochloride (preferred to dehydrochloride) 1 Gm. 3 times weekly

c. Para-aminosalicylic acid 5 Gm. daily

With this treatment there is clinical improvement in some and marked improvement in other cases. In bilateral renal tuberculosis it is usually advisable to remove the more severely infected kidney, giving the triad of



Fig. 445.—Pyelogram showing deformity caused by renal tuberculosis

chemotherapy concurrently. The chemotherapy is started one week preoperatively and continued for 2 years postoperatively. This frequently results in healing of the lesion in the less involved kidney.

Tuberculous epididymitis responds very slowly, if at all, to streptomycin therapy. This lack of response may be due to failure to obtain an adequate drug concentration in the tissue. Tuberculous scrotal sinuses respond promptly to streptomycin therapy.

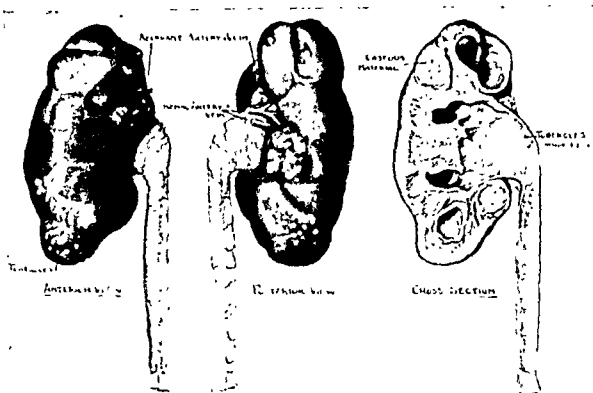


Fig 416 —Gross specimen of renal tuberculosis

Streptomycin is not used in patients with a small contracted tuberculous bladder as this will achieve only slight improvement of the clinical symptoms but will not alter the overall picture.

Institution of operative measures at the proper time for chronic renal tuberculosis rarely causes a flare-up of the associated pulmonary disease. Surgery should be delayed whenever possible until the pulmonary lesion has become quiescent.

In cases of renal tuberculosis, the normal-appearing ureter will show microscopic tuber-

culous lesions in 30% of cases. It is for this reason that many surgeons recommend nephroureterectomy for renal tuberculosis, even though the ureter appears normal at the time of operation.

Summary of Treatment (Table 35).—All cases of genitourinary tuberculosis, especially after surgery:

1. Complete bed rest
2. Sanatorium treatment, or the equivalent at home for a period of 6 months

The severity of the symptoms should be a strong factor in determining treatment. One

TABLE 35
TREATMENT OF RENAL TUBERCULOSIS

UNILATERAL	BILATERAL
Medical treatment—Isoniazid, streptomycin and para-aminosalicylic acid	Triad of chemotherapy for 2 years
Surgical treatment—Nephrectomy or nephroureterectomy if the kidney is completely destroyed, to be preceded and followed by isoniazid, streptomycin and para-aminosalicylic acid	<ol style="list-style-type: none"> a Isoniazid b Streptomycin c Para-aminosalicylic acid
	1 Remove the more diseased kidney, if healing has not occurred after 1 year of triple therapy, plus the triad of chemotherapy
	2 Bilateral cutaneous or intestinal ureteral transplant in advanced bilateral disease—to be preceded and followed for 1 year by triad of chemotherapy

does not like to do a bilateral cutaneous transplant because its care is difficult for the patient.

Tuberculosis of the Bladder

Tuberculosis of the bladder is secondary to tuberculosis of the kidneys or seminal tract in the male. In the female it may be secondary to tuberculosis of the pelvic organs. In the majority of cases the primary focus is in the kidney. The ureteral orifice on the affected side is usually involved.

Involvement of the bladder, secondary to a tuberculous focus in the prostate or urethra, occurs by direct mucosal extension. In such cases the trigone is practically always the point of first attack. In most cases, however, the bladder involvement is due to the planting of tubercle bacilli from infected urine directly on the vesical mucosa, the resistance of which has been lowered by the continuous irritation of infected urine. This leads to inflammation, edema, ulceration, and finally fibrosis of the bladder wall. The bladder becomes contracted and its capacity greatly reduced.

Symptoms.—

1. Intense frequency
2. Painful urination, the pain occurring at the end of micturition

3. Slight, intermittent hematuria

Diagnosis.—

1. Appearance of the bladder at cystoscopy, small tuberculous ulcers
2. The recovery of tubercle bacilli from the urine

Prognosis.—If the source of the vesical infection is removed (usually a tuberculous kidney), the bladder condition often heals rapidly. If, however, the original source of the disease is not eliminated, it is practically impossible to cure, or even materially relieve, a tuberculous cystitis. When treatment is completed, sanatorium care should be instituted.

Tuberculosis of the Prostate

Tuberculosis of the prostate alone is very rare. It is usually part of a progressive infection that is extending throughout the genital or urogenital system, being present in about 70% of cases of urogenital tuberculosis. In

most instances tuberculosis of the prostate occurs by direct extension from the seminal vesicles. Urogenital tuberculosis tends to spread from one portion of the urinary tract to another.

Symptoms.—

1. None at all
2. Frequency, dysuria, hematuria, pyuria

Diagnosis.—

1. Rectal examination—the prostate feels firm, irregular, and nodular
2. Examination of the prostatic fluid for tubercle bacilli
3. A plain x-ray of the pelvis to show, if possible, areas of calcification in the prostate

Treatment.—

1. Total perineal prostatectomy gives poor results. Chronic sinuses may result.
2. Sanatorium rest or the equivalent thereof often gives relief of symptoms, although the course of the disease is essentially unaltered.
3. Antibiotics are useful.

Tuberculosis of the Epididymis and Testicle

Autopsy records show that tuberculous lesions may be limited to the epididymes, seminal vesicles, or prostate but that such limitation is rare. Clinically the majority of cases exhibit multiple lesions. The infection in the epididymis usually begins in the globus minor and gradually extends throughout the epididymis and into the vas. The tuberculous process in the epididymis may assume such extensive proportions that the testicle becomes a small compressed organ.

The disease sometimes appears to be bilateral from the onset. When tuberculosis infects only one epididymis the opposite epididymis is likely to become infected sooner or later. For this reason it is advisable to do a vasectomy on the unaffected side.

Tuberculosis of the testicle is very rare. It is usually secondary to tuberculosis of the epididymis. The entire testicle may become a hard mass by gradual extension of the disease. The scrotum becomes adherent to the mass; the process finally ulcerates through the skin to form a chronic fistula discharging caseous material. The function of the affected organ

is destroyed and, should the process become bilateral, complete sterility is inevitable.

Sinuses on the posterior surface of the scrotum are from the epididymis. Those on the anterior surface are from the testicle. A persistent scrotal sinus of months' or even years' duration is frequently the reason the patient seeks medical advice.

Diagnosis.—

1 History of trauma is present in many cases, frequently it is this that draws the patient's attention to the fact that there is a swelling present in the scrotum

2 Loss of weight and general malaise are usually present

3 Bladder symptoms may be present

4 Fever is rare.

5 Pain and tenderness in the epididymis are mild and intermittent

6 There is history of exacerbations and remissions of the swelling

7. On palpation the epididymis is found to be swollen, nodular, and irregular in contour.

8 A persistent draining sinus may be present.

Differential Diagnosis.—

1. Tuberculous epididymitis

2. Inflammation due to other causes

Prognosis.—The high morbidity of genital tuberculosis, even when properly treated, is not generally appreciated. The disease is progressive and fairly rapid in its evolution. Prior to the use of antibiotics the ultimate mortality varied from 27-60%. The patient must be examined yearly for 10 years

Treatment.—

1. Epididymectomy—with transplantation of the vas to the skin of the groin

2. Six months' sanatorium treatment or the equivalent at home

3. Antibiotics

BLADDER

Anatomy.—The bladder is a reservoir for urine. It is a musculomembranous sac situated in the bony pelvis behind the pubis and in front of the rectum in the male. In the female it is separated from the rectum by the cervix uteri and the vagina.

When empty, the bladder is pyriform in shape, with the apex below. The ureter of each side is connected to each of the upper angles of the triangle, and the urethra is connected below. The urachus (obliterated allantoic duct) connects the bladder to the umbilicus. The superior or upper surface of the bladder is covered with peritoneum. The normal bladder capacity is approximately 500 ml of fluid.

The interior of the bladder is lined with folds and shows on the floor the vesical trigone, a pale-colored triangular space, marked at its upper and outer angles by the entry of the ureter on each side (ureteral orifice) and extending below to the opening of the urethra. The interureteric ridge runs transversely between the two ureteral orifices.

Blood Supply.—The arteries of the bladder are the superior vesical, middle vesical, and

inferior vesical in the male, with additional branches from the uterine and vaginal arteries in the female. These are all derived from the anterior trunk of the internal iliac artery.

The veins form a plexus around the neck, sides, and base of the bladder and terminate in the internal iliac veins.

The lymphatics terminate in the internal iliac nodes.

Developmental Embryology.—The rudimentary alimentary canal ends below in a blind pouch known as the cloaca, which has a prolongation known as the allantoic diverticulum which later becomes the urachus. The mesonephric duct which later becomes the vas deferens opens into the anterior part of the cloaca. The ureters develop as an outgrowth of this duct. In time this common duct is absorbed into the wall of the bladder and prostatic urethra, with the result that the vas and the ureter eventually develop separate openings. A urorectal septum then forms which divides the cloaca into an anterior, or urogenital, part and a posterior, or intestinal, part.

Extrophy of the bladder is a developmental anomaly in which the anterior surface of the

bladder fails to close. There is a corresponding defect in the abdominal wall, so that the floor of the bladder presents on the anterior abdominal wall. The ureteral orifices usually protrude.

Nerve Supply.—The nerve supply of the bladder, ureter, and urethra is of three sources sympathetic, parasympathetic, and somatic. The sympathetic fibers which innervate the bladder are derived from T11-12 and L1-2, and come together in a strand called the *presacral nerve*. Lateral roots connect this to the 1st and 2nd lumbar prevertebral sympathetic ganglia. A middle root from the preaortic nervous plexus

traction. The middle portion supplies the urethra and genitalia, the distal portion innervates the bowel.

The *somatic* fibers from S 3-4 pass in the *pudendal nerves* to innervate the striated muscle around the urethra and the external sphincter of the bladder. The pudendal nerve also contains sensory fibers from the posterior urethra.

Physiology of Micturition.—For many years it has been accepted that there is reciprocal innervation of the bladder, i.e., that the parasympathetic contracts the detrusor muscle and inhibits the internal sphincter; conversely,

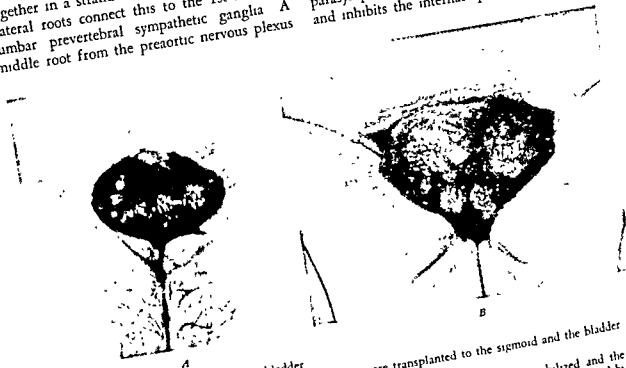


Fig 447 —Extrophy of the bladder
A, Case 1 At the age of 3 years the ureters were transplanted to the sigmoid and the bladder excised

B, Case 2 At the age of 2 years the lateral walls of the bladder were mobilized and the bladder closed over the balloon of a Foley catheter. The defect in the abdominal wall was closed by covering it with a flap of rectus fascia and skin. Reconstruction of the urethral sphincter has not been attempted yet in this patient. Hugh Young reports a successful outcome by use of this procedure.

also joins it. This network provides intercommunication between the celiac, semilunar, and aorticorenal ganglia. At the level of the promontory of the sacrum the presacral nerve contains both afferent and efferent fibers.

The *parasympathetic* fibers arise from the sacral segments 2, 3, 4 and run through the hypogastric plexus. The proximal portion of the hypogastric plexus forms a nerve group which is motor to the bladder and causes con-

traction that the sympathetic contracts the internal sphincter and relaxes the detrusor. This view is no longer held. Anatomically there is no true sphincter at the bladder neck. The detrusor muscle of the bladder, i.e., the longitudinal layer, is continuous with the bladder neck, so that what has been called the internal sphincter is really simply the edge of the detrusor muscle. The circular muscular layer of the bladder is responsible for the maintenance

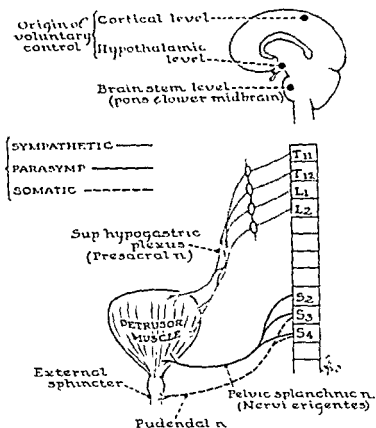


Fig. 418—Pathways responsible for normal micturition. Diagram shows sympathetic, para-sympathetic, and somatic nerve supplies of the bladder. Note that the parasympathetic fibers carry the impulses which are responsible for contraction of the detrusor muscle and that the pudendal nerve relaxes the external sphincter. The role of the sympathetic system in the act of micturition is somewhat indefinite.

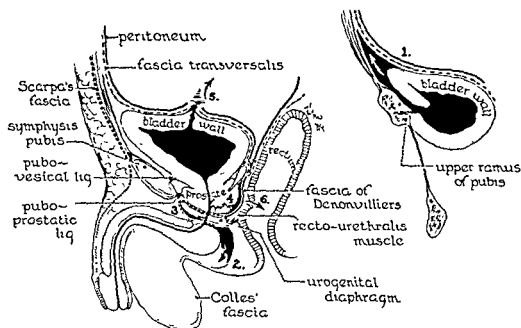


Fig. 449—Anatomic relations of the urinary bladder, urethra, and rectum, showing sites of injury.

of adequate intravesical pressure. The sympathetic nerves play no part in micturition. They carry pain and temperature sensations only.

The act of micturition is essentially a spinal reflex. Distention of the bladder causes a desire to void. This stretch reflex is carried by the afferent fibers in the parasympathetic nerves to the sacral spinal cord and up the cord to the pons, subcortical, and cortical areas of the brain where involuntary and voluntary control, respectively, are normally situated. With the removal of voluntary control, the act of micturition is initiated by a contraction of the vesicular muscle (i.e., detrusor or longitudinal muscle) via the *parasympathetics*. Once this contraction is fairly well established, the internal orifice of the bladder (bladder neck, "internal sphincter") opens, and the external sphincter (supplied by the pudendal nerve, i.e., somatic) relaxes (See also pp. 292 and 293).

The Neurogenic Bladder

Immediately following serious trauma to the spinal cord in which there is spinal transection, a condition known as "spinal shock" ensues. This is a state of depression of the spinal reflexes in the segments caudal to the lesion. It is generally transient in character.

The actual mechanics of this condition are unknown, but it causes paralysis with loss of reflexes below the level of the lesion. The bladder is immediately affected as shown by the following conditions:

1. Atonicity of the bladder wall
2. Absence of reflex contraction of the detrusor
3. Failure of sensory impulses to the brain and motor impulses from it

The bladder distends to a large size and overflow incontinence occurs in 36 hours.

The atonic bladder noted immediately after injury in the state known as *spinal shock* is the first of three merging but fairly well-defined stages of spontaneous recovery.

First Stage—This may last 1 day to 18 months. Overflow incontinence is the only type of urination to be expected. Cystometric studies during this stage show a large bladder capacity with low pressure and without evidence of reflex detrusor activity, i.e., atonic neurogenic bladder.

Second Stage—This is a variable period of time from days to months after the injury; a return of reflexes caudal to the injured segment usually takes place. It is manifested in the cystometrogram by higher intravesical pressure. Reflex contractions of the bladder wall occur, but they are of insufficient strength or duration to produce efficient emptying. The clinical picture is that of periodic overflow incontinence, i.e., autonomous neurogenic bladder.

Third Stage—Progression to a third and final stage of recovery depends upon the extent of the injury and upon whether (1) transection has been partial or complete, (2) level of injury, (3) general condition of the patient, (4) degree of infection in the bladder.

Treatment of Bladder in Spinal Cord Injuries

Immediate Care—

1. Repeated catheterization at intervals is condemned because severe infections ensue.
2. Indwelling urethral catheter, either with or without tidal drainage, is not to be recommended, except in cases where fairly prompt recovery is anticipated. A periurethral abscess is a frequent complication.

3. Suprapubic cystostomy is usually the treatment of choice. With the bladder draining well through a large suprapubic tube, infection can be controlled. The cystostomy should be as high as possible. Some objections to this type of immediate treatment have been raised, the claim being made that with prolonged drainage, fibrosis of the bladder occurs and the bladder capacity is permanently reduced. This, however, has never been proved.

4. Urinary antiseptics should be used, commencing at the time of the injury.

Late Care.—

1. Continue with mild urinary antiseptics throughout the period of treatment. The intention is to obtain a bacteriostatic rather than a bactericidal effect.

2. Introduce methods to prevent calculus formation, i.e., move the patient frequently from side to side; get patient up if possible, otherwise use a tilting board under the mattress.

3. Keep the bladder clean by repeated irrigations, using either a bulb syringe or tidal irrigator apparatus. Solutions of boracic acid, sulfonimides, or buffered citric acid solution, G, M, or I, may be used for irrigation.

4. Remove any calculi that form in either the bladder or kidneys.

5. When recovery appears to be taking place, as evidenced by a return of reflexes caudal to the injured segment and the showing of a higher intravesical pressure on the cystometrogram, the suprapubic tube can be removed and the bladder allowed to close. An indwelling urethral catheter may be of assistance at this time. Rarely is a secondary closure of a suprapubic wound required.

Final Stage.—Recovery to the final stage is often surprisingly satisfactory. If the lesion is one of the cauda equina, fairly good control of the bladder can be developed.

In many cases, fibrosis of the bladder neck occurs with resultant hypertrophy of the adjacent muscle. An hourglass type of bladder neck develops. This may be relieved by a transurethral resection in which successive pieces of tissue are removed from around the bladder neck. It is desirable to eliminate residual urine. Repeated resections may be necessary to accomplish this.

Many patients in spite of careful treatment continue to have a residual urine of 1-2 ounces. In these patients a catheter should be passed once a week and the bladder thoroughly irrigated, as stagnant residual urine is prone to become infected and form calculi.

(See Infections of the Bladder, pp. 862-870.)

(See Acute Urinary Retention, pp. 891-895.)

Rupture of the Urinary Bladder

An understanding of rupture of the bladder requires knowledge of the fascial attachments of this organ. It should be remembered that injury to the bladder is frequently associated with other injuries. The patient may have multiple bone injuries, including head injuries as well as a torn bladder, and be in a fairly advanced degree of shock. In this type of case, history taking, physical examination to determine the extent of injury, and treatment of the shock proceed concurrently. Radiologic investi-

gation usually follows as soon as it is feasible. In the author's opinion, an intravenous pyelogram series is useful, as it will show extravasation of dye from the bladder, if it is torn, and also outline the kidneys.

Ligaments of Bladder.—Each side of the bladder is connected to the tendinous arch of the pelvic fascia by a condensation of fibroareolar tissue which is often termed the lateral true ligament of the bladder.

Anteriorly, the same tissue forms two thickened bands on each side of the midline, termed the lateral and medial puboprostatic ligaments. The apex of the bladder is joined to the umbilicus by the remains of the urachus which forms the median umbilical ligament. From its superior surface the peritoneum is reflected in a series of folds which are sometimes termed the false ligaments of the bladder. The fascia in the anterior part of the perineum is divisible into a superficial fatty layer and a deeper membranous layer, the layer of Colles.

Etiology.—The types of trauma causing rupture:

1. Crushing injuries
2. Falls
3. Blows and kicks
4. Manual emptying of the bladder

Rupture of the urinary bladder is usually due to external violence when the bladder is distended. The bladder when contracted often escapes injury even in violent crushing accidents. Rupture of the bladder is more common in males.

Pathology.—It follows from the foregoing that four distinct types of injury to the bladder may occur.

1. *Avulsion of the bladder neck.* In this condition the pubic symphysis is suddenly and violently separated, tearing the bladder neck away from its ligamentous moorings to the pubis and pelvis. The pubic symphysis may snap back into place immediately after the blow, so that one may not suspect from the x-ray that it has been separated. Further, the bladder neck may escape injury even when there is wide separation of the symphysis.

2. *Perforation of the bladder by a spicule of bone during a crushing injury of the pelvis.*

This is almost always extraperitoneal, the perforation occurring below the peritoneal reflection. Urine and blood escape into the space of Retzius, spread upward toward the umbilicus and laterally to the ischial spines. If the rupture is on the anterior wall, extravasation about the vesical neck and between the bladder and rectum will occur. This causes chills, fever, tenderness, infection, sloughing, and gangrene.

3. *Intraperitoneal rupture of the bladder caused by a direct blow.* Forty per cent of bladder tears are intraperitoneal and 60% are extraperitoneal. An intraperitoneal rupture occurs when the posterosuperior wall of the bladder is torn (usually following a direct blow to a full bladder). Blood and urine extravasate into the peritoneal cavity, causing a peritonitis with abdominal distention, tenderness, rigidity, fever, and rapid pulse.



A.



B.

Fig. 450—A, X-ray showing separation of the pubic symphysis, fracture of the rami, and extravasation of dye into the retropubic space from the bladder which had been widely torn at the bladder neck. The patient had been run over by the double rear wheels of a six ton truck.

B, X-ray showing wide separation of the pubic symphysis and fracture of the rami without injury to the bladder. The patient, a ballet dancer, has been able to continue her dancing career.

4. *Rupture of a partially distended bladder across its base posteriorly following a blow.* This is also an extraperitoneal rupture.

Symptoms of Rupture of the Bladder.—Early symptoms are due to injuries. Late symptoms are due to extravasation of urine and infection.

Signs of rupture:

1. Shock
2. Helplessness, patient unable to walk or walks with difficulty
3. Partial or complete inability to void

6. The passage of a catheter with the injection of a measured amount of fluid re-measured on its return not a reliable test.

7. Evidence of extravasation of dye in a cystogram.

8. Cystoscopic examination, which may reveal the tear.

Treatment.—

1. Combat the shock and hemorrhage which is most important.

2. Explore the bladder by suprapubic cystotomy and provide drainage. This should be done even in doubtful cases.

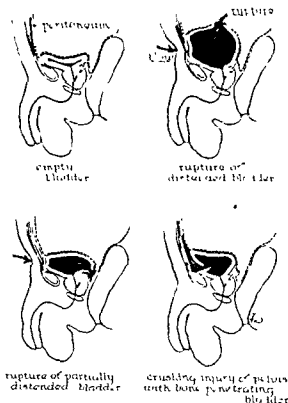


Fig. 451.—Diagram showing varying anatomic relations of bladder when empty, distended, and ruptured.

4. Blood at the urethral meatus.

5. Ecchymosis, contusions, and hematoma of lower abdomen, pubes, genitalia, and thighs.

Diagnosis.—Diagnosis is made on the following:

1. History
2. Shock
3. Local pains, tenderness, and swelling
4. Inability to void
5. Blood in urine

3. If the tear is intraperitoneal, open the peritoneal cavity, mop up any free fluid, and drain the peritoneum separately from the bladder.

4. If the tear is extraperitoneal, drain the bladder and all the extravasical areas of extravasation.

Prognosis.—The prognosis is good if the injury is recognized early and the bladder is drained.

GENITOURINARY SYSTEM

Tumors of the Bladder

Classification

I. Papillary

Papilloma

Papillary carcinoma

II Solid

Transitional cell carcinoma

Squamous cell carcinoma

Adenocarcinoma (mucus secreting)

Anaplastic carcinoma

III Mesoblastic tumors

1 Benign growths

Fibroma

Myoma

Myxoma

Angioma

2 Malignant

Sarcoma

Rhabdomyosarcoma

Theories of Origin.—The cause of cancer is unknown. There are several theories as to the cause of bladder tumors

1 Virus infections

2 Chemical irritation

3 Occupational, e.g., bladder tumors in aniline dye workers and the scrotal cancers of chimney sweeps

4. The influence of the constant flow of urine

Papilloma—Papillomatosis

Papillomas of the bladder may be single or multiple. When several small growths occur together or in rapid sequence, the condition is termed papillomatosis.

They may be found anywhere in the urinary bladder. The majority are situated on the trigone or at the junction of the trigone and bladder. They frequently occur at the ureteral orifices. They are less common on the lateral walls.

Pathology.—

Gross.—The papilloma appears as a pedunculated villous or papillary growth arising from the mucous membrane. It may be sessile, but this type is less frequent. Some papillomas have a wartlike surface, while others are almost smooth. The size of the growths varies considerably, some are small and may remain



Fig 452 A Cystogram showing marked filling defect of left side of bladder due to extensive bladder tumor
B Cystogram showing filling defect in right side of bladder due to pedunculated tumor protruding into bladder

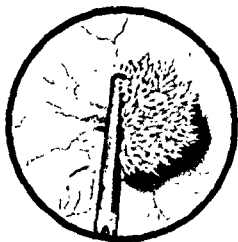


Plate 38.—Bladder Papilloma Close to Left Ureteral Orifice.

*Courtesy Marshall, Victor F.: The Diagnosis of Genito-Urinary Neoplasms, American Cancer Society, Inc.
From Pullen's Medical Diagnosis, W. B. Saunders Co*



Plate 39.—Carcinoma of Urinary Bladder.

Courtesy Marshall, Victor F.: The Diagnosis of Genito-Urinary Neoplasms, American Cancer Society, Inc

so over long periods. Both the pedunculated and the more malignant sessile tumors may attain considerable size.

Microscopic.—A typical papilloma shows multiple connective tissue stalks containing thin walled blood vessels, thus the tendency to spontaneous bleeding. Glandular or cystic formations may be present, or an inflammatory process may cause a leukocytic invasion of the central stalk, but these should not be mistaken for a malignancy.

Histologically there is no difference between the single and multiple forms. Ninety per cent are papillary in character. Many are benign and may remain so for years. The marked tendency to malignancy of the so-called pedunculated papilloma and the ill defined demarcation that exists between it and malignant growths have led many pathologists to regard all vesical papillomas as potentially malignant, even if initially they can be proved to be benign.

Papillary Carcinoma

Papillary carcinoma is a papilloma with malignant infiltration at the base and in the underlying bladder wall. It is the most common type of vesical carcinoma. The American Registry of Pathology classifies all papillomas as papillary carcinomas.

Squamous Cell Carcinoma (Flat Infiltrating Carcinoma)

Squamous cell carcinoma is less common than papillary carcinoma. It is a flat sessile growth with a cornifying epithelium which projects only slightly into the vesical cavity but invades deeply and spreads laterally. It is deeply and rapidly infiltrating at the outset. It is the most malignant of the vesical neoplasms and is radioresistant.

Pathology.—

Gross.—This carcinoma appears as a shallow red malignant ulcer surrounded by a hard everted border.

Microscopic.—Relatively thick connective tissue is seen between the cell nests, as distinguished from the papillary type of carcinoma. The cells progress along the surface of

the bladder and at the same time invade the deeper layers of the wall.

Leukoplakia is commonly regarded as a precursor of squamous cell carcinoma.

Adenocarcinoma and Adenoma

Adenocarcinoma, primary in the bladder, is a rare, highly malignant neoplasm closely resembling adenocarcinoma of the prostate gland. It arises chiefly from the paraprostatic glands that are sometimes present in the wall of the male trigone and in remnants of the urachus in the apex of the bladder. It may occur as a flat growth, showing extensive infiltration of the bladder wall, or it may have a thick pedicle and project into the bladder as a pedunculated tumor. It ulcerates early.

Interstitial Carcinoma

An interstitial carcinoma growing in the wall of the bladder may not produce any symptoms except a decreasing bladder capacity. The bladder is rigid and will not stretch under anesthesia when one tries to introduce fluid forcibly. The growth is usually located in the vicinity of the bladder neck or at the apex. The structure varies greatly. They are usually small in size and sessile, though some are pedunculated.

Mesoblastic Tumors

Benign—may be fibroma, myoma, myxoma, angioma, hemangioma, or neurofibroma.

Sarcoma—is rare and fast growing.

Metastases

Metastases of bladder tumors occur in 10-40% of all cases. Metastasis may occur as follows:

1 By direct local extension to the prostate, pelvic tissues, to the kidney via the ureter, to the penis or vagina.

2 By the lymphatic or blood stream to the long bones, liver, lungs, and regional lymph nodes; recurrence after removal fairly common.

Symptoms.—Painless hematuria is present in 80% of the patients. Frequency, dysuria, pyuria, and interruption of the urinary stream are other predominant symptoms.

Diagnosis.—Diagnosis is made on the following.

1. History
2. Physical examination
3. Examination of urine for malignant cells with special stains
4. Cystoscopy—the tumor viewed directly
5. Cystogram—filling defect may be seen
6. Biopsy

Treatment.—

1. Fulguration or coagulation of the tumor either transurethraly or suprapubically, depending upon the size
2. Local resection of the tumor-bearing area with 1 cm of healthy appearing tissue around it
3. Local resection plus the implantation of radon seeds
4. Radiation both externally and by opening the bladder and inserting an x-ray tube—produces a severe cystitis without appreciably affecting the tumor
5. Total cystectomy with transplantation of the ureters to either the bowel or skin
6. Permanent suprapubic cystostomy

The Use of Isolated Ileal Segments as Bladder Substitutes

The use of an isolated segment of ileum as a substitute bladder has proved of value in dealing with far-advanced cases of carcinoma involving the urinary bladder, rectum, and cervix, or in total pelvic exenteration and prior to cordotomy or alcohol injection of the spinal canal for the relief of intractable pelvic pain due to carcinoma.

Bricker's method of isolating 8-10 inches of ileum approximately 8" from the ileocecal valve, closing the proximal end and transplanting the ureters to the isolated loop by mucosa-to-mucosa anastomosis, and then bringing the proximal end of the loop out onto the skin surface has proved most useful. We have used this in far-advanced cases of carcinoma of the cervix where the pelvis is "frozen," in vesical-vaginal-rectal fistulas due to carcinoma of the cervix, in total pelvic exenteration, and in cases of recurrent carcinoma of the urinary bladder which could not be controlled by local excision, radiation therapy, or cobalt

therapy. In the latter cases the bladder and prostate are removed in toto at the time the substitute bladder is constructed. The results in all types of cases mentioned have been most gratifying

Metaplasia of the Bladder

The epithelial cells of the urinary tract are particularly prone to proliferation and metaplasia. After minimal injuries, such as an infection in the bladder or a calculus, epithelial changes may occur and result in projection of buds from the inferior layer of epithelium of the bladder into the submucosa. These are known as *Brunn's epithelial nests* which may become vacuolated, forming cysts. This is known as *cystitis cystica*. (Urethritis cystica or pyelitis cystica represents the same process going on in the ureter or the pelvis of the kidney.) Either of these conditions may be a precursor of leukoplakia and, in turn, of *squamous cell carcinoma*.

In other "Brunn nests," changes may occur that result in gland formation. The epithelial cells lining these glands are capable of secreting mucus. This process is known as *cystitis glandularis* and may undergo metamorphosis into a mucus-secreting *adenocarcinoma*.

Ulceration of the Bladder

May be due to the following:

1. Interstitial cystitis (submucous fibrosis)
2. Infections other than tuberculosis
3. Carcinoma
4. Radiation

Interstitial Cystitis.—(Synonyms: Hunner's ulcer, submucous fibrosis, elusive ulcer.) This is characterized by single or multiple minute lesions of the bladder wall. Ulcers are not always present. The etiology of this disease is entirely unknown. It is much more common in females. The lesions are frequently located on the dome of the bladder. Pathologically the mucosa is flattened and there is submucous fibrosis. The clinical picture is variable, but usually there is pronounced frequency and suprapubic pain. Microscopically the urine is normal except for a few scattered red blood cells in it.

The treatment of this condition is often extremely difficult and has led to a wide variety of methods:

1. Dilatation of the bladder under spinal anesthetic
2. Instillation of Novocain anesthetic into the bladder, followed by silver nitrate
3. Fulguration of the ulcers
4. Resection of the bladder, removing the ulcerated areas

Infectious Ulceration.—Ulceration associated with pyogenic infection of the bladder may occur but is uncommon. Ulcers may be single or multiple and disappear when the infection is eliminated. Ulceration in tuberculous cystitis is relatively common. (See section on Genitourinary Tuberculosis.)

Carcinoma.—The ulceration associated with carcinoma of the bladder is readily recognized. It is slow growing, clear cut, and appears punched out. Any ulcer of the bladder that becomes chronic should be suspected of malignancy.

Radiation Ulcer.—A radiation ulcer of the bladder may follow deep x-ray therapy to the pelvic region or follow the implantation of radium into the cervix. The actual ulceration may not commence until months up to 12 years after the exposure. The ulcer is indolent, slow growing, and may progressively increase in size. The underlying pathologic process is an endarteritis, with obliteration of the vessels. The treatment is symptomatic. Instillations of oil into the bladder give some relief of symptoms. Cerutin appears to have a beneficial effect on cases recognized early, before actual ulceration has occurred.

Diverticulum of the Bladder

A diverticulum or saccular ballooning out of the bladder wall is a relatively common urologic finding. It may vary in degree from multiple shallow sacculations, involving most of the bladder wall, to single or multiple sacs connected to the bladder by a narrow opening. They may reach a considerable size. A diverticulum with a narrow neck drains poorly and almost always contains stagnant infected urine. The causes of diverticulum may be

1. Congenital weakness of the bladder wall

2. Obstruction to the urinary outflow (prostatic obstruction, urethral valves, etc.)

It is probably more nearly correct to say that a diverticulum is always due to obstruction of the outflow. A diverticulum may be complicated by the presence in it of a calculus or a tumor.

Symptoms.—The diverticulum itself rarely produces symptoms. It is usually a coincidental finding at the time of investigating a patient for symptoms of urinary obstruction. Pyuria is a common finding.



Fig. 453.—X ray showing diverticulum of bladder

Treatment consists of the following:

1. Removing the diverticulum or the mucous membrane lining it
2. Removing the cause of the obstruction (prostate, vesical neck contracture, valves in the urethra, etc.)
3. For a calculus or a tumor in a diverticulum, removing the diverticulum

Vesical Calculus

Calculi in the urinary bladder may be single or multiple; they vary from the size of a garden pea to that of a full-term fetal head. They vary in shape from round to jackstone

and may be smooth or irregular. They are most commonly due to or associated with the following:

- 1 Infection
- 2 Obstruction of the bladder neck, usually associated with infection
- 3 Foreign bodies in the bladder
- 4 Neurogenic bladder

The general etiology of calculi has been discussed on page 870. In the obstructed bladder with its concomitant residual urine and associated infection, the *Bacillus proteus* is frequently the offending organism. A foreign body in the bladder, such as a surgical sponge left after an operation on the bladder, or a broken tip of a filiform dilator, is certain to produce a calculus. Many patients with a neurogenic or cord bladder following a spinal cord injury have some residual urine, which even though small in amount becomes heavily infected, thick, and foul, and frequently causes stone formation. This can usually be prevented by the passage of a catheter and bladder lavage once a week.

The chemical composition of a vesical calculus is generally mixed. Different compositions produce layers so that the stone presents a lamellated appearance in cross section.

Composition of Vesical Calculi.—

- 1 Uric acid, urates. The stone is flat or ovoid, yellow to dark red in color, friable, and may be either rough or smooth.
- 2 Phosphatic calcium phosphate, magnesium phosphate, ammonium phosphate. The stone is oval or round, white in color, and may be friable.
- 3 Calcium oxalate. The stone is round, brown to black in color, has a mulberry surface, and is extremely hard.

Complications of Vesical Calculi.—

- 1 Obstruction of bladder neck
- 2 Infection
- 3 Ulceration of bladder
- 4 Impaction of the stone in the bladder neck, diverticulum, or urethra

Symptoms.—

- 1 Pain radiating to the glans penis, present on voiding and on exercise
- 2 Frequency and urgency
- 3 Hematuria
- 4 Interruption of the urinary stream
- 5 Retention of urine

Diagnosis.—Diagnosis is made on the following.

1. History
2. Urinalysis, examining especially for red blood cells
- 3 X-ray evidence of calculus in the bladder—about 95% of bladder calculi contain calcium and are visible radiologically.
- 4 Cystoscopic examination with direct visualization of the calculus



Fig. 451.—Plain film of bladder area showing fairly large vesical calculus

Treatment.—

- 1 Crushing of the stone instrumentally through the urethra if it is not too large (litholapaxy)
- 2 Removing the stone by opening the bladder suprapubically (cystolithotomy)
- 3 Removal of the calculus by perineal section

Foreign Bodies in the Bladder

A wide variety of foreign bodies may be found in the urinary bladder. The commonest are surgical sponges, broken off tips of filiform dilators, and the wide variety of foreign bodies introduced by sexual perversers. Such objects may be toothpicks, lead pencils, chewing gum, hairpins, etc.

The presence of a foreign body in the bladder produces an intense acute inflammation with all the symptoms of an acute cystitis previously described. The foreign body can frequently be removed by instrumentation

through the urethra. If this is not possible, suprapubic cystotomy is done and the foreign body removed.

Extravasation of Urine

(See page 883)

PROSTATE GLAND

Anatomy.—The prostate gland is a musculo-glandular organ, which surrounds the neck of the bladder and the first portion of the urethra in the male. It is situated below the pubic symphysis and behind the inferior fascia of the triangular ligament. Its posterior surface rests on the rectum. The urethra traverses its length. The seminal vesicles which are situated on the posterior surface empty into the ejaculatory duct on each side in the prostatic urethra. The prostatic ducts also open into the prostatic urethra.

The prostate gland is made up of three lobes, two lateral and a middle, which give the gland a horse collar shape. It normally weighs about 20 Gm. Attached to the prostate are the puboprostatic ligament and the inferior fascia of the urogenital diaphragm (triangular ligament). It is surrounded by a firm fibrous capsule.

The blood supply of the prostate is derived from the internal pudendal, vesical, and middle hemorrhoidal arteries.

The prostatic capsule is surrounded by a rich plexus of veins which terminate in the internal iliac vein.

Embryology.—At about the 55 mm stage a series of buds arranged in five groups, arising from all sides of the urethra and from the upper pelvic portion of the definitive urogenital sinus, grows into the dense surrounding mesenchyme which later differentiates into the muscular and connective tissues of the gland. The posterior lobe rudiment develops a separate capsule.

Normal Physiology of the Prostate.—

Prostatic Secretion.—The daily secretion averages 0.5–2.0 ml. This is discharged through the ducts into the urine. Prostatic secretion contains, on chemical analysis, organic and in-

organic substances, sodium, potassium, calcium, protein, glucose, ascorbic acid, citric acid, acid phosphatase, alkaline phosphatase, and cholesterol.

Acid phosphatase is present in very small amounts before puberty but increases greatly during the sexual life. In carcinoma of the prostate, acid phosphatase appears in large amounts in the blood when the growth extends beyond the capsule of the gland.

The prostate does not have any internal secretion.

There is a large amount of lipid fluid in the prostatic secretion in the form of corpuscles, which may be mistaken for pus cells.

Prostatic fluid is alkaline. It apparently contains no secretion essential to fertilization. Young produced pregnancy in guinea pigs by insemination with sperm from isolated epididymes.

Effects of Endocrine Therapy on Prostate Gland.—There is no verified clinical or experimental evidence that endocrine therapy has any effect on either the normal or hypertrophied adult prostate gland of man.

There is no positive clinical or experimental evidence that administered androgen or estrogen causes carcinoma in the prostate.

When estrogen is administered to patients with carcinoma of the prostate, there appears to be retardation of the growth, which may decrease in size.

Conditions of the Prostate Gland

Anomalies

Absence of the prostate gland or congenital cyst rarely occurs. A congenital cyst may be removed surgically.

Prostatic Injuries

These may be due to the following:

1. External injury, e.g., falling astride a picket fence. Rupture of the urethra is a frequent complication and is the usual cause of a stricture located in the posterior urethra
2. Internal injury caused by the passage of instruments. A false passage may be made when passing a sound
3. Operative injury, which is very rare. This may occur during operations on the perineum

Prostatic Calculi

Calculi form in the prostate gland only when an infection has been present for a prolonged period. The calculi are usually multiple and vary in size from that of grape seeds to the size of marbles, completely replacing the glandular structure so that only the capsule is left. There may be hundreds of smaller calculi or only a few large ones.

They may not cause any symptoms or may cause typical symptoms of prostatic obstruction. They can frequently be palpated rectally and visualized on x-ray.

Treatment.—If the calculi are small, they are removed by doing a transurethral prostatic resection. If they are large and fill the entire prostatic capsule, they should be removed surgically, either by the suprapubic, retropubic, or perineal route.

Prostatism (Synonym: Benign Prostatic Hypertrophy)

Definition.—Prostatism is a term used to describe a condition in which urinary symptoms develop as the result of obstruction at the bladder neck due to either an increase or decrease in the size of the prostate gland. It is caused by hyperplasia of the gland (median bar), sclerosis of the gland (median bar), or by invasion of the gland by new growth (carcinoma).

Age Incidence.—Prostatic obstruction occurs with increasing frequency in each decade, from the age of 40 onward. The incidence is 40-49 (9%), 50-59 (20%); 60-69 (35%); 70-79 (43%).

Theories of Origin.—The so-called prostatic hypertrophy is neither prostatic nor hypertrophy. It is hyperplasia. It consists of tissue

which arises from the epithelium and the ducts surrounding the verumontanum on the floor of the urethra. The hyperplasia consists of nodules made up of fibromuscular and glandular tissue. The former arises from an analogue of the müllerian duct system and the latter from the prostatic duct epithelium.

Chronic infection or vascular changes in the prostate gland and hormonal imbalance have also been thought to be responsible for the gradual increase in the size of the gland. Castration, or the administration of estrogenic substances, does not result in reduction of the hyperplastic process.

Symptoms of Prostatic Obstruction.—The first symptom of prostatic obstruction is the gradual onset of difficulty in starting the urinary stream, followed by increased frequency, nocturia, hesitancy of the urinary stream, urgency, and, in many cases, hematuria. Incomplete retention develops (i.e., residual urine) and eventually a complete retention with inability to void. Some cases show incontinence due to stretching of the internal sphincter by the increased size of the prostate. Sexual debility is a common complaint. There may be pain either in the suprapubic region or the lower lumbar region posteriorly.

Diagnosis of Prostatic Obstruction.—The diagnosis of prostatic obstruction is made on the history of gradually increasing urinary difficulty and the symptoms listed above. On rectal examination an enlarged prostate is palpated. The patient is catheterized immediately after voiding to determine the amount of residual urine present. On cystoscopic examination the enlarged prostate is visualized at the bladder neck. A cystogram will also frequently show an intravesical protrusion of the enlarged prostate.

Treatment.—There are four accepted surgical methods of treating enlargement of the prostate gland.

1. **Transurethral resection.** In this operation successive pieces of tissue are removed from around the bladder neck and the prostate itself by means of a resectoscope. The gland is not as completely removed as in the other types of operation.

2. **Suprapubic prostatectomy.** This may be carried out in either one or two stages. A

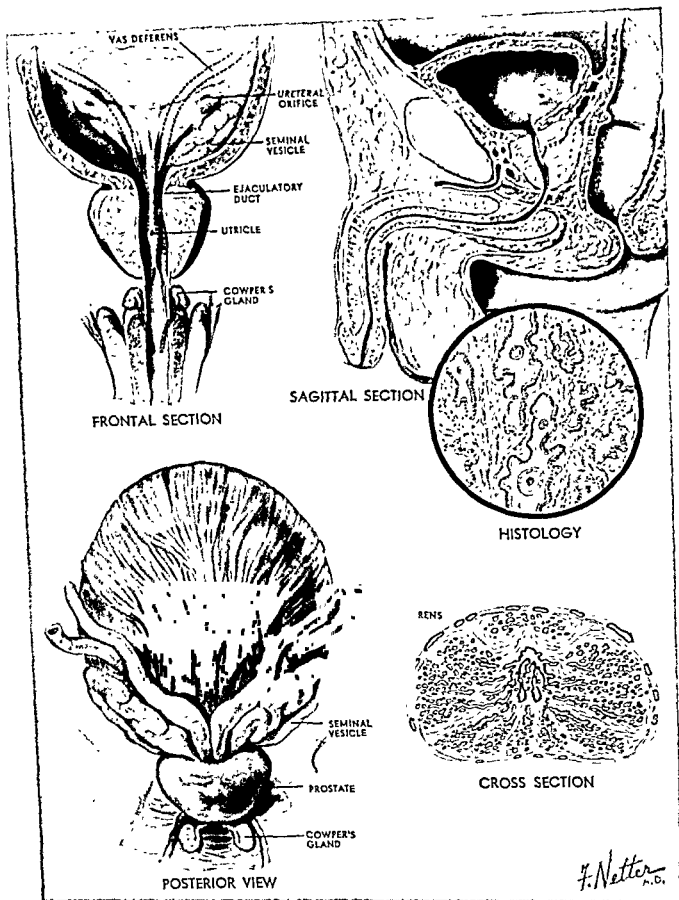


Plate 40.—Anatomy of the Prostate Gland.

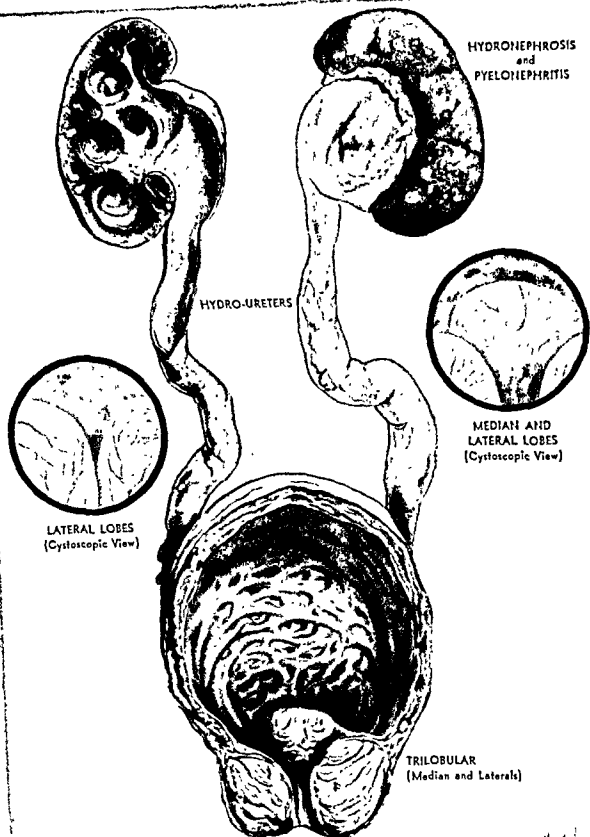


Plate 42.—Prostatic Obstruction Producing Dilatation of the Ureters and Renal Pelvis.

skin incision is made in the suprapubic region of the lower abdomen, the bladder opened, and a drainage tube inserted into the bladder. The prostate is removed at the time of opening the bladder or at a subsequent operation 2 or more weeks later. If the patient is debilitated or uremic, removal of the prostate is deferred until the patient improves.

3 *Retroperic prostatectomy.* The prostate gland is removed through a skin incision made in the lower abdomen. The entire procedure is done outside the bladder through the prostatic capsule, without actually opening the bladder.

4 *Perineal prostatectomy.* The prostate gland is removed through an incision in the perineum above the anus which exposes the gland from the outside, without entering the bladder.

Carcinoma of the Prostate

Incidence.—Carcinoma of the prostate gland affects 5% of all men who reach the age of 60 years. One of every five prostate glands removed for benign hyperplasia shows carcinoma histologically.

Carcinoma usually begins in the posterior lobe but may be found anywhere in the gland.

Diagnosis of Carcinoma of Prostate.
This is based on the following:

1. Rectal examination. The gland is usually irregular in shape and stony hard in consistency. The hardness may be limited to a small area of the gland or involve the entire gland, even extending out laterally into the ligaments and upward into the seminal vesicles. Such a gland is referred to as "fixed" or "frozen."

2. Examination of the prostatic fluid by special stains for carcinoma cells is unreliable even in proved cases of carcinoma.

3. Biopsy of the prostate gland establishes absolute proof of carcinoma but is occasionally misleading, since the biopsy may fail to show carcinoma that is present elsewhere in the gland.

4. X-ray evidence of metastases in the bones or chest.

5. Elevated acid serum phosphatase.

The Significance of the Acid Serum Phosphatase in Prostatic Carcinoma.—A phos-

phatase is an enzyme which splits organic phosphorus compounds to give free phosphate ions. Phosphatases differ in the pH at which they show their maximum activity and accordingly are known as *acid* or *alkaline* phosphatases.

Normal human prostatic tissue is the only tissue in the body that is rich in *acid phosphatase*. The concentration in adult human prostatic tissue is 500-2,500 units of activity per gram fresh tissue as compared with less than 5 units of activity at pH 4.9 for kidney, liver, duodenal mucosa, and bones. The enzyme normally is excreted in the prostatic component of the ejaculate. Its exact function is unknown. In prostatic carcinoma, when the capsule of the gland ceases to be intact and the growth infiltrates into the surrounding soft tissues or metastasizes to bone, the "acid" phosphatase which is normally below 3 units/100 ml of serum rises. Values above 6 units/100 ml are pathognomonic of metastasizing prostatic carcinoma.

When the secretory stimulus of the androgens is removed by castration, there is a rapid fall in the *acid* serum phosphatase by 45% within 48 hours, 75% within 2 weeks, followed by a transient gradual rise, and, finally, a prolonged decline until after 2-3 months, equilibrium is reached. In those patients who do particularly well clinically, the acid serum phosphatase remains at these new lower levels. A persistently high postoperative acid phosphatase after castration is due to stimulation by androgens from extragonadal sources (adrenals).

The Significance of the Alkaline Serum Phosphatase in Prostatic Carcinoma.—There is no correlation between the acid serum phosphatase and alkaline serum phosphatase in patients with metastasizing prostatic carcinoma. The amount of alkaline phosphatase in the blood is an indication of the activity of the bone defense. After castration, the early changes are not consistent, but after a latent period of 2-3 weeks there is a rise of the alkaline phosphatase attributable to the osteoblastic activity in the healing of skeletal metastases. As the extra bone formation finally decreases, the serum alkaline phosphatase level declines to normal.

Symptoms of Carcinoma of the Prostate.—

1. Prostatism—causing symptoms of obstruction

2 Pain—due either to direct extension in the pelvis and involvement of nerve sheaths or to metastases in the spine or pelvic bones

Treatment.—

1 Radical perineal prostatectomy in cases recognized early This is the only method of obtaining a cure The prostate, seminal vesicles, and bladder neck are removed en masse

2 Surgical castration

3 Medical castration—by giving stilbestrol 5 mg per day orally for 2 weeks, then 5 mg twice a week thereafter

Some surgeons prefer to bombard the prostate with large doses of estrogen, thereby causing the gland to shrink, and to follow this by a radical perineal prostatectomy. It is claimed that an inoperable carcinoma is thereby converted into an operable one

In patients suffering excruciating pain from metastases, spectacular and complete relief of pain is obtained, in most cases within 72 hours, by surgical castration

Chronic Prostatitis and Nonspecific Urethritis

Nonspecific urethritis and prostatitis are frequently erroneously regarded as being a chronic form of gonorrheal urethritis. Either or both may exist without a previous attack of gonorrhea. Chronic nonspecific urethritis alone is rare, whereas chronic prostatitis alone is common. Fifty per cent of cases with chronic prostatitis or urethritis give a previous history of gonorrhea, 36% have had what was diagnosed as a nonspecific urethritis, and 14% have never had any previous symptoms, the discovery of a large number of pus cells in the prostatic fluid being a coincidental finding. In this latter group the infection is comparable to an upper respiratory infection, except that the infection settles in the prostate gland which, because of its dependent position in the body with consequent poor drainage and complex histologic architecture, affords an ideal environment to harbor infection.

Symptoms.—The patients' symptoms are urethral discharge, frequency, burning, terminal hematuria, vague perineal discomfort, and backache. In the group of cases in which

there is a previous history of gonorrheal or nonspecific urethritis, urethral discharge is usually present and is the main symptom in over 70% of cases. In those cases in which there is no previous history of urethritis, a urethral discharge occurs in approximately 21%. Pyuria without symptoms is a common finding.

Origin of Condition.—The majority of these patients give a history of alcoholism and sexual excess immediately preceding the onset or recurrence of symptoms. This appears to be a factor in lowering the resistance of the urethra and prostate by the following:

1 Favoring subsequent invasion by organisms from the host which are normally nonpathogenic, via the lymphatics or possibly the blood stream

2 Exacerbation of a previous prostatitis in which there is still some residual infection present

3 Organisms from a carrier at the time of intercourse, which may not be pathogenic to the carrier

4 Virus

Bacteriology.—The urethral discharge when cultured shows staphylococci in 55% of cases, diptheroids 10%, mixed staphylococci, streptococci, and micrococci 32%, trichomonas vaginalis 2%, and gonococci 1%. Protozoa may also be found in the urethral discharge.

Diagnosis.—A diagnosis is made on the following:

- 1 The history and symptoms
- 2 The presence of urethral discharge
- 3 The three-glass urinalysis
- 4 Examination of the prostatic fluid

The symptoms of urethral discharge, frequency, burning, terminal hematuria, and vague perineal discomfort are almost pathognomonic of the condition. Palpation of the prostate is often misleading. In some cases the gland is firm and fibrosed; in others it is soft and normal to palpation. Examination of the prostatic fluid obtained by massage is essential to establish a diagnosis.

Criteria of Cure.—

- 1 The patient must be entirely free of symptoms, and there must be no urethral discharge
- 2 The three-glass urinalysis is normal.
- 3 The prostatic fluid on culture is negative for gonococci.

4 The number of pus cells in the prostatic fluid is reduced to below 5 per high power field

5 Re-examination in 3 months' time confirms these findings

Treatment.—

1. Prostatic massage—twice weekly—until apparently cured.

2 Dilatation of urethra—stricture of the urethra even though not severe and a small external urinary meatus are the two commonest causes of failure to eradicate the infection. A normal urethra should accommodate a Fr. No. 26 sound without difficulty.

3 Urethral irrigations are frequently helpful, particularly if there is much urethral discharge. Any one of several solutions may be used

a Protargol— $\frac{1}{4}$ of 1%

b Potassium permanganate—1:12,000 solution

c Silver nitrate—1:10,000 solution

d Chemotherapy

a Penicillin—300,000 units of penicillin G intramuscularly daily for 14 days

b Streptomycin, 1 Gm. daily for 14 days

c Aureomycin—according to weight

d Chloromycetin—according to weight

e Terramycin

f Atabrine, 90 mg t i d

g Arsenic

Prostatic massage is considered to be an essential part of the treatment. When this is deleted from the program of treatment, the percentage of cases "apparently cured" drops from 75% to 10%

Treatment may require weeks or even months to complete. Recurrence is unfortunately common and suggests that the disease was not entirely eradicated by treatment, despite clinical and laboratory tests to the contrary

Prostatic Abscess

Etiology.—Prostatic abscess may be single or multiple and follows—

1. Acute gonorrheal urethritis (uncommon now)

2. Any nonspecific prostatitis

3 Traumatic instrumentation

4 Urethral stricture

5 Metastatic infection, as a complication of pyemia, typhoid fever, influenza, carbuncles, or boils

Symptoms.—The symptoms are pain in the perineum, fever, chills, and increasing difficulty in voiding which may progress to acute retention.

Prognosis.—The prostatic abscess may rupture into the urethra, rectum, perineum, or bladder.

Treatment.—

1 Incision and drainage of the abscess through the perineum. A perineal urethrotomy may be done at the same time and an indwelling catheter inserted.

2 Chemotherapy. Usually one of the antibiotics can be used, the type depending upon the bacteriology of the infection.

Tuberculosis of the Prostate

(See page 878.)

PENIS AND URETHRA

CONDITIONS OF THE PENIS

1 Congenital anomalies (double phallus, hypospadias, epispadias)

2 Phimosis, paraphimosis, balanitis

3 Venereal warts (condylomata acuminata, condylomata lata)

4. Chancre, chancroid, lymphogranuloma venereum, granuloma inguinale

5 Plastic induration (Peyronie's disease)

6 Priapism

7 Tumors

Congenital Anomalies

Double phallus is a rare anomaly.

Hypospadias is a congenital defect of the anterior urethra. The urethra is incompletely formed so that it opens on the undersurface of the penis, in the scrotum, or perineum. This is a relatively common anomaly. Hypospadias is classified as first, second, or third degree, depending upon the site of the urethral opening. The first degree opening is just behind the normal site of the external urinary meatus

In hypospadias a hood or cap of foreskin overhangs the glans penis. This should be preserved for future use in plastic procedures on the penis. Associated with the hypospadias there is a marked chordee or downward curvature of the penis, caused by replacement of the corpus cavernosum urethrae by fibrous tissue. Correction of the hypospadias is achieved by a series of plastic operations. The first stage consists of removing the scar tissue causing the chordee and straightening the penis. At the second operation a new urethra is formed by using full or split-thickness skin grafts or by means of a tube pedicle graft. The urethra is buried, covered, and finally connected to the normal urethra. Before such operations are undertaken, the urinary stream is diverted by suprapubic cystostomy. The end results are usually satisfactory.

Epispadias is a rare anomaly. The urethra is situated above the corpora cavernosa and lies open on the dorsal surface of the penis. The roof of the urethra is lacking throughout part or all of its length. This condition is usually associated with exstrophy of the bladder which may be partial or complete. Surgical correction of the deformity with construction of a urethra and closure of the bladder can be carried out, although incontinence is likely to persist. An alternative treatment is to transplant both ureters into the bowel and excise the bladder.

Phimosis

Phimosis is a condition of the foreskin which prevents its retraction. The prepuce is long and redundant. The acquired form is seen in adults and is due to irritation which may be inflammatory, traumatic, or consequent to incontinence associated with prostatic obstruction or overflow from the bladder.

In the acute stage irrigations under the foreskin with potassium permanganate or boric acid solution hasten recovery. If the foreskin is sectioned along the dorsum of the glans penis, the healing process is further accelerated. When the acute inflammation subsides, circumcision is indicated to prevent recurrence.

Paraphimosis

Paraphimosis is strangulation of the foreskin by the circular constriction of the re-

tracted prepuce which can no longer be reduced. It frequently accompanies acute inflammatory conditions, such as gonorrhea, chancre, and chancroid. It requires immediate relief either by digitally reducing the glans through the ring or by surgical division of the constricting band (dorsal slit). Gangrene results if the strangulation is not relieved. Paraphimosis also occurs in the aged, when the elasticity of the foreskin is lost.

Balanitis

Balanitis is a superficial inflammation of the glans penis. In most cases the undersurface of the prepuce is also affected, producing a balanoposthitis. The glans penis becomes damp, red, and itchy. An exudate forms. Treatment consists of local cleanliness. Circumcision during quiescence is desirable so that debris and moisture can no longer collect beneath the foreskin.

Venereal Warts

Condylomata acuminata are single or multiple wartlike eruptions on the skin of the external genitalia. They are variable in shape and are most commonly situated around the corona (i.e., the sulcus proximal to the glans penis) and on the prepuce. They are not due to venereal disease but are most common due to excessive moisture. They are removed by treating them with aqueous podophyl 25%, silver nitrate stick, or by electrofulguration. Clinically the results are much more satisfactory if the redundant foreskin with attached warts is excised and the remaining warts fulgurated.

Condylomata lata, the warts of secondary syphilis, are moist broad warts that frequently fuse and cover an extensive area, even extending as far as the anus. Diagnosis is based on dark-field and serologic examination. Treatment consists of local cleanliness and antisyphilitic therapy.

Chancre, Chancroid, Lymphogranuloma Venereum

Syphilis (chancre) is the result of infection by the *Spirochaeta pallida*. In the primary



Plate 44.—Condylomata Acuminata (Venereal Warts).



Plate 45 —Carcinoma of the Penis.

Courtesy Marshall, Victor F.: The Diagnosis of Genito-Urinary Neoplasms, American Cancer Society, Inc.

stage, at the end of the incubation period, a chancre appears on the glans penis or shaft of the penis, at the corona, frenulum, or inside the prepuce. The lesion is painless. It is commonly single but may be multiple. The chancre has a hard, indurated, raised edge, like a button, with an intensely red ulcerated center. The inguinal nodes are enlarged, firm, and discrete (shotty). The treatment is antisyphilitic therapy.

Chancroid (soft chancre) is a venereal ulcer usually situated on the external genitalia and caused by the Ducey-Unna bacillus. Uncleanliness and dirty habits favor its development. The ulcers are irregular with a gray base and undermined edges. Suppurative inguinal adenitis may occur and is called a bubo.

Diagnosis is made on the appearance of the ulcer, the clinical course of the disease, the skin test (commercial antigen injected intradermally), and the presence of the Ducey bacillus. Syphilis must be excluded.

Treatment consists of local cleanliness and sulfonamide therapy. Cauterization of the ulcers with nitric acid has been recommended.

Lymphogranuloma venereum is a specific disease caused by a filtrable virus. A primary sore appears 2-7 days after exposure, usually on the corona in the male and on the posterior vaginal wall or posterior cervical wall in the female. The initial lesion is vesicular or nodular. Soon there is swelling in the inguinal nodes. The overlying skin breaks down and sinuses form. *Diagnosis* is made upon the clinical appearance and the Frei cutaneous test.

Antimony is specific for this disease. Aureomycin is also effective.

Granuloma inguinale is a lesion which begins as a papule in the groin, perineum, vulva, or prepuce. It soon ulcerates and invades the surrounding tissues. It does not show any tendency to heal. It is caused by a specific organism called the Donovan body (*Bacillus mucosus capsulatus*). Secondary infection and erosion cause marked deformity of the organs. A diagnosis is made by the presence of Donovan bodies. Specific treatment is the intravenous administration of antimony and potassium tartrate. Fuadin is also effective.

Plastic Induration of the Penis (Peyronie's Disease)

The induration is due to fibrous infiltration of the penis which begins in the septum between the corpora cavernosa. It may extend into any part of the penis and finally reaches Buck's fascia and the tunica albuginea, where it can be felt as hard indurated plaques. These plaques and fibrosis cause deformity of the penis during erection and marked pain. The etiology is unknown.

Various forms of treatment have been recommended, including radiation therapy, excision, plastic procedures, etc. The results are moderately satisfactory. Recently the weekly injection of 1 ml of Hydrocortone F directly into the plaque has been recommended. Although the number of patients so treated is still small, the results appear to be superior to any of the other forms of therapy.

Priapism

Priapism is prolonged involuntary erection of the penis without sexual desire. This is an uncommon condition and difficult to treat, as it does not respond to medication. It may occur at any age but is more common in young adults. There is usually some serious underlying disease, such as leukemia, syphilis, neurologic disorders, or malignancies. Treatment consists of treating the underlying cause if one can be discovered. Incision of one or both corpora, with expression of blood clots, which form after a prolonged period, may be required.

Prognosis.—Impotence usually follows.

Tumors of the Penis

Benign.—

1. Condylomata acuminata (venereal warts) (see previous description)
2. Condylomata lata (syphilis) (see previous description)
3. Angioma
4. Balanitis xerotica obliterans, a weeping dermatitis of the glans penis, regarded as premalignant

Malignant.—

1. Carcinoma (squamous cell carcinoma)
2. Sarcoma—very rare
3. Multiple hemorrhagic sarcomas—very rare

Carcinoma of the Penis

Carcinoma of the penis is a relatively common malignant tumor. It is thought that accumulated smegma and uncleanness are predisposing factors. It is practically never found in the Jewish race or Mohammedans, both of whom practice circumcision as a religious rite in the first week of life. Strangely enough, if circumcision is performed on a child of 5 years, it does not prevent him from developing carcinoma in adult life, suggesting that whatever the carcinogenic factor may be, it remains latent throughout most of the life's span. Equally interesting is the fact that carcinoma of the cervix is relatively uncommon in Jewish women. The exact etiology of carcinoma of the penis is unknown.

Site.—The majority of carcinomas of the penis arise on the glans or in the coronal sulcus. The lesion may be papillary, cauliflower, or infiltrating in type. Any of these may ulcerate. Metastases to the inguinal nodes occur early in the disease. Secondary infection of the nodes results in suppuration and necrosis.

Diagnosis.—This is made on the clinical appearance and biopsy. Early diagnosis is important.

Treatment.—

1. Partial amputation of penis
2. Complete amputation of penis with radical excision of the inguinal lymph nodes
3. Caustic excision of the tumor, with implantation of radon seeds
4. Irradiation

CONDITIONS OF THE URETHRA

1. Urethritis (gonorrhea, nonspecific; Reiter's disease)
2. Stricture
3. Perurethral phlegmon (perurethral abscess)
4. Tumors
5. Rupture

Urethritis

Etiology.—*Gonorrheal urethritis* is an acute specific inflammatory process involving the anterior urethra and caused by the Neisserian organism (gram-negative diplococci). The inflammatory process is characterized by an exudation of phagocytes in an endeavor to limit the infection. The production of pus results in a profuse urethral discharge. The glands of Littre in the anterior urethra are involved; this explains why an inflammatory stricture is found only in the anterior urethra.

Earlier teaching stressed the importance of avoiding spread of the infection to the posterior urethra and to the prostate by urethral irrigations or instrumentation. With modern chemotherapy in which neither irrigations nor instrumentation are used, spread of the inflammatory process to the posterior urethra and the prostate appears to occur spontaneously in some cases within the first 24 hours.

Symptoms.—Symptoms of an acute gonorrheal urethritis are a profuse urethral discharge, frequency, burning, and urgency.

Diagnosis.—This is made on the history of exposure and the finding of gram-negative diplococci, either intracellular or extracellular, or both, in smears of urethral discharge.

Complications of Gonorrheal Urethritis

1. Epididymitis (elevate the scrotum, apply heat)
2. Prostatitis—acute, may become chronic
3. Prostatic abscess
4. Urethral stricture
5. Arthritis
6. Gonorrheal endocarditis—very rare

Treatment.—(In order of preference)

1. Penicillin—300,000 units daily for 2 days
2. Streptomycin—1 Gm daily for 2 days
3. Sulfonamides—either trisulfas or sulfadiazine, 4 Gm daily for 3 days, gradually decreasing
4. Irrigations—Protargol 1/4 of 1%, potassium permanganate 1/2, 0.001 solution, silver nitrate 1/10,000 solution—no longer used in acute or newly acquired infections but of considerable value in the chronic or recurrent form

Nonspecific Urethritis (see page 894).

Reiter's disease is characterized by the triad (1) urethral discharge, (2) conjunctivitis, and (3) polyarthrititis.

Its cause is unknown. No specific organism has been recovered from the urethral discharge. *Conjunctivitis* may be severe and progresses to erosion. *Treatment* is not specific and must therefore be symptomatic and palliative. Recovery occurs after a period of months.

Stricture of the Urethra

Stricture of the anterior urethra is usually inflammatory but may be due to tearing of the urethra by a blow. Stricture of the posterior urethra is always traumatic in origin.

Diagnosis.—

- 1 History of a previous acute inflammation or trauma
- 2 History of a narrow urinary stream or forking of the stream
- 3 Signs of infection (frequency, urgency, burning, associated with a narrow stream)
- 4 Palpation of the urethra—an indurated area often palpable
- 5 Urethrogram—x-ray of the urethra taken at the time of injection of an opaque liquid or jelly, the narrowed area in the urethra visualized
- 6 Calibration of the urethra with a bougie-à-boule, determination of the site, length, and size of the stricture with this instrument

Treatment of Stricture —

- 1 Dilatation of the urethra. This may be carried out with filiforms and followers (gum elastic dilators attached to a fine guide), Philip's bougies, metal sounds (Laforte's), or by a Kollmann dilator
- 2 Internal urethrotomy. A specially designed urethrotomy knife is passed down the urethra to the site of the stricture and a longitudinal incision of the stricture is made.
- 3 Surgical excision of the stricture. This is performed with end-to-end anastomosis or tube graft.

Instrumentation of Urethra

- 1 For urinary retention
- 2 For stricture of urethra

Any instrumentation of the urethra should be preceded and followed by a thorough irriga-

tion of the urethra with a mild antiseptic. Urethral chills with high fever due to toxic absorption are practically never seen if this precaution is taken. Sterile technique should be maintained throughout. Adequate lubrication is essential. Petroleum jelly, liquid lubricant, and olive oil instilled into the urethra are all satisfactory.

Technique of Instrumentation

Catheterization of the urinary bladder may be required for acute retention due to prostatism or following surgical operations. There are several different types of catheters with which this procedure may be carried out:

1. Soft rubber catheter, either plain or Foley bag type
2. Soft rubber catheter with catheter stylet
3. Coudé catheter (one angle on distal end)
4. Bicoudé catheter (two angles on distal portion)



Fig 455 —Foley bag catheter in the bladder.

In patients with postoperative retention of urine or retention due to prostatism, an attempt is first made to pass a soft rubber catheter. This may be either a plain catheter or one with

a distensible balloon on the end (Foley bag catheter) if it is desired to leave it in place as a retention catheter. The urethra is well irrigated, and the catheter lubricated (either with sterile liquid paraffin, glycerine, or olive oil or lubricating jelly). The catheter is then passed *gently*. This is successful in the majority of cases. Additional lubrication of the urethra by the injection of 5-10 ml. of the sterile lubricant into the urethra prior to passing the catheter often converts a difficult into an easy catheterization.

In cases of prostatism it may be impossible to pass a soft rubber catheter. A curved catheter stylet may be used to increase the rigidity of the catheter. The catheter with the stylet is guided into the bladder over the obstructing prostatic lobe.

If the above two maneuvers are not successful a *condé* or *bicondé* catheter is passed. These are gum elastic silk woven catheters with one bend about 1 cm. from the tip (*coudé*) or two bends—1 cm. from the tip and the other 7.5 cm. from the tip (*bicoudé*). They are most helpful in cases of prostatic obstruction.

For many years it has been taught that decompression of the bladder should be carried out very slowly and gradually over a 24-hour period, in order to avoid sudden massive hemorrhage. Sudden massive hemorrhage following rapid decompression is so rare that most urologists in the course of a lifetime have not seen a case.

A *bougie-à-boule* is an instrument about 25 cm. long, with a narrow shaft and an acorn-shaped enlargement at one end and an olive-shaped enlargement at the other end. The enlargements at the end are calibrated in size from Fr. No. 14 to Fr. No. 24. The instrument may be either gum elastic silk woven or metal. It is used to explore the urethra to determine the presence of a stricture. Its advantage over a sound is that tissue-paper-thin strictures can be palpated, whereas with a sound they would be broken down without being recognized. The largest size (Fr. No. 24) is passed first. If an obstruction is encountered, the instrument is withdrawn and the next size passed, continuing thus down the scale until one is found that can be passed to

the bladder. On withdrawing the bougie, the enlarged end is felt to grate and pass irregularly in the scar tissue of the stricture. It is thus possible to chart the site, length, and caliber of the stricture. Such information is valuable in subsequent treatment.

Metal Sounds.—The usual metal sound is an instrument that should be used with great delicacy and care. Under no circumstances should force be used. The urethra is irrigated and the instrument (Fr. No. 12 or 14) lubricated. The point is passed through the meatus, the handle of the instrument being held over the patient's abdomen. As the deep urethra is approached, the handle is rotated through an arc of approximately 120 degrees downward, the curved proximal end of the sound thus sliding under the symphysis into the bladder. The sound should be closely opposed to the roof of the urethra throughout its entire passage.

Kollmann's dilator is a metal instrument with expanding blades that are controlled by a screw arrangement at the head. It is passed like a sound. A scale also attached to the head of the instrument indicates the caliber obtained as the blades are opened. This dilator is seldom used.

Periurethral Abscess (Periurethral Phlegmon)

Periurethral abscess may occur at the site of any inflammatory stricture or upon the use of an indwelling urethral catheter for a prolonged period, causing erosion of the urethra.

Extravasated urine spreads into the scrotum, causing marked swelling of the scrotum and penis, and into the abdominal wall. It is limited in its spread by the attachment of Colles' fascia inferiorly to the lower border of the urogenital diaphragm and laterally to the rami of the ischium and pubis; superiorly it spreads within the abdominal wall deep to Scarpa's fascia.

The treatment of periurethral abscess and extravasation of urine consists of the following:

1. Diversion of the urinary stream by suprapubic cystostomy.
2. Incision and drainage of the abscess.
3. Multiple incisions with through-and-through drainage in the area of extravasation.

New Growths of the Urethra

New growths of the urethra, benign or malignant, are very rare; 192 cases of primary carcinoma of the male urethra have been reported.

Symptoms and Signs.—

1. Difficulty in micturition is the commonest symptom of growths in the anterior urethra.

2. Hematuria is the commonest symptom of growths in the posterior urethra.

3. The growth may be palpable.

The dysuria when associated with a perineural mass and difficulty in dilating a previously easily dilated stricture are suggestive of new growth.

Diagnosis.—This is made on the following

1. Symptoms

2. Palpation of the urethra

3. Endoscopic examination

4. Biopsy

Treatment.—

1. Amputation of the penis, the treatment of choice

2. Extensive resection of the urethra

3. Direct application of radium for a relatively small growth in the fossa navicularis

4. Fulguration of small growth

Prognosis.—Carcinoma of anterior urethra—50% of patients are living after 2 years. Carcinoma of posterior urethra—50% of patients are dead one year after operation, indicating the difficulty of complete removal of the growth if situated posteriorly.

Urethral caruncle is a growth resembling a thrombosed vein which appears in the female at the external urinary orifice. It is relatively common, benign, and treated by excision or fulguration.

Rupture of the Urethra

Rupture of the urethra is caused by direct violence (such as falling astride a beam) or by indirect violence (such as crushing injuries of the pelvis). The site of the rupture is commonly in the membranous or prostatic urethra.

Signs of Rupture.—

1. Bleeding from the external urinary meatus

2. Hematoma at the site of injury

3. Inability to void

4. Variable degrees of shock

Treatment.—Depending upon the conditions present at the examination, one of several methods may be used to treat the ruptured urethra.

1. If a soft rubber urethral catheter can be passed, it should be left in place and taped in.

2. Immediate suprapubic cystostomy with repair of the urethra. The bladder is opened suprapubically, and a sound is then passed through the bladder and internal sphincter into the urethra to the site of rupture. Another sound is passed through the external meatus into the urethra until it meets the former (male and female sounds). The sound passed from the bladder side is used to direct the other sound into the bladder. When this has been achieved, a catheter is attached to the sound passed through the penis, and the sound and catheter are withdrawn in a retrograde manner. The catheter is left in situ as a splint for the urethra. A de Pezzer tube is inserted into the bladder and brought out through the suprapubic incision.

3. Immediate repair by exposing the site of rupture in the perineum, identification of the torn ends of the urethra, and end-to-end anastomosis of the ends over a rubber or Foley bag catheter.

4. Suprapubic cystostomy only, with repair of the urethra at a later date.

5. Perineal section and urethrotomy. A catheter is passed through the urethrotomy opening to the bladder to divert the urine. The urethra is repaired at a later date.

6. In the late cases, or in cases of old injury or those previously treated by suprapubic cystostomy, the scarred torn urethra is exposed in the perineum. The scar is excised, and a split-thickness skin graft is built up over a tracheal cuff placed on a Foley bag indwelling catheter. The cuff with its attached skin graft is then positioned on the catheter to fill the gap caused by the excision of the scar. Two inches or more of the urethra can be replaced by this method.

All cases of rupture of the urethra will require periodic dilatation throughout the remainder of life.

SCROTUM AND SCROTAL CONTENTS

Congenital Anomalies

Anorchidism—congenital absence of both testes

Monorchidism—development of only one testicle.

Synorchidism—the fusion of both testicles intra-abdominally; has only been discovered at operation or autopsy.

Polyorchidism—supernumerary testicles.

Cryptorchidism—congenital malposition of the testicle

The congenital anomalies with the exception of cryptorchidism are rare

Surgical Conditions of the Scrotum

Injuries.—The scrotum may be subjected to a direct blow, lacerating wound, or avulsion. A laceration is thoroughly cleaned with soap and water, débrided, and sutured. *Avulsion of the entire skin of the scrotum* may occur in industrial accidents, where wheels and belts are employed. The immediate treatment required is that for shock, followed by covering the scrotum with skin flaps.

Inflammation.—Inflammation of the scrotal wall usually occurs secondary to general diseases such as diabetes, cardio-renal failure, etc. It may also occur as the result of a peritesticular phlegmon, extravasation of urine, and chemical or physical trauma (e.g., frostbite).

Tumors.—Tumors of the scrotum are very rare. The treatment is excision.

Elephantiasis.—This condition, in which there may be enormous swelling of the scrotum, is due to infestation by filariae which fill the lymphatic channels, causing obstruction. The disease is rare in temperate, but common in tropical, climates. The enlarged scrotum may reach the knees. Treatment consists of excision of the burdensome excess tissue.

Surgical Conditions of the Scrotal Contents

Vestigial Structures in the Scrotum

Appendix testis (i.e., hydatid of Morgagni) is pedunculated and attached to the front of the head of the epididymis or lies in the groove between the testis and epididymis.

Appendix epididymis is a pedunculated hydatid. It is rare. It is attached to the front of the upper pole of the epididymis.

Paradidymis or *organ of Gicaldès* is a collection of small tubules found above the head of the epididymis and in the lower end of the spermatic cord in front of the vessels.

Vasa aberrantia or *ductuli aberrantes* are coiled canals with blind ends in the epididymis. The inferior (that of Haller) is the larger and is located in the tail of the epididymis.

Testicular Tunics

Hydrocele.—A hydrocele is a sac of fluid within the scrotum, surrounding the testicle, the sac being formed by the tunica vaginalis. This is the commonest type and is called *idiopathic vaginal hydrocele*. In *congenital hydrocele*, the processus vaginalis communicates with the abdominal cavity. Elevation of the scrotum causes the fluid to return to the peritoneal cavity. In *infantile hydrocele* the tunica vaginalis extends up to the internal inguinal ring. The sac, however, has no connection with the peritoneal cavity. *Encysted hydrocele of the cord* is associated with the spermatic cord. The processus vaginalis is closed above and below, so that the sac moves with the cord when traction is applied to the testicle. A *secondary hydrocele* may occur as a complication of epididymo-orchitis, either acute or chronic.

Symptoms.—The patient notices a gradually increasing painless swelling in the scrotum.

Diagnosis.—This is made on the presence of a scrotal mass and by transillumination. The spermatic cord and the external inguinal rings are normal to palpation and do not suggest an inflammatory process. In a *hematocele*, light is not transmitted. An *epididymis* is tender to palpation.

Treatment.—

1. **Aspiration** by means of a large-caliber needle. This procedure must be repeated at intervals. It is not curative; the hydrocele recurs and the sac gets thicker.

2. **Injection.** A needle is inserted into the sac and the fluid is withdrawn. Quinine and urea hydrochloride solution at intervals of one

week is injected into the sac on two or three occasions. Some claim permanent cure by this treatment.

3. **Excision of the hydrocele.** The sac is opened, everted, and the excess wall is excised. The edges are sutured behind the epididymis.

4. **Scrotal support.** This often provides comfort.

Hematocoele.—This is a collection of blood in the tunica vaginalis. It usually results from an injury. A common cause is the aspiration of a hydrocele. Repeated hemorrhages may occur, giving gradual enlargement. The testicle is flattened by pressure. Treatment consists of opening the sac, removing the clot, and excising the sac.

Chylocele.—Chylocele is the presence of chyle fluid (lymphatic fluid which contains cholesterol particles from the digestive tract) in the tunica vaginalis. It is rare but may occur in cases of chronic lymphatic obstruction, e.g., filariasis.

Hernia.—A hernia containing intestine may be present in the scrotum, as well as a hydrocele. It is one of the more important conditions to be differentiated from hydrocele. It does not transilluminate.

Epididymis

Injuries to the epididymis are rare but may follow a direct blow. Swelling and edema occur.

Spermatocele and Cysts.—Cysts of the epididymis, particularly the head, are much more common than spermatocele. They are usually multiple and filled with a clear fluid. A spermatocele is a unilocular retention cyst derived from some portion of the sperm-conducting system of the epididymis. It is therefore filled with cloudy fluid. Treatment is excision (of the cyst or spermatocele).

Inflammation.

GONORRHEA.—Acute gonorrheal epididymitis occurs as a complication of gonorrheal urethritis. The epididymis becomes enlarged, hard, and exquisitely tender. Pain is acute. Fever of 103°-104° F is often present. When epididymitis is acute, urethral discharge is scanty.

Diagnosis.—The diagnosis of epididymitis is determined by the following:

1. History of exposure
2. An enlarged tender mass found on physical examination
3. High fever
4. The finding of Neisserian organisms in the urethral discharge

Differential Diagnosis.—Epididymitis must be differentiated from torsion of the testicle. Prehn has described a useful sign in this connection, namely, that elevation of the scrotum gives relief of pain in epididymitis but increases the pain in torsion of the testicle (Prehn's sign).

Treatment—

1. Specific—penicillin or streptomycin
2. Local—elevate the scrotum (adhesive bag)
3. Application of hot compresses or ice packs—either works satisfactorily, although heat gives more relief from the pain.

Acute epididymitis is usually followed by resolution, provided it is not tuberculous in origin.

NONSPECIFIC EPIDIDYMITIS.—This lesion may occur without determinable cause, presumably due to lowered tissue resistance. The epididymis becomes enlarged, hard, and tender. There is no urethral discharge, and frequently no history of exposure.

Treatment—

1. Administration of penicillin or streptomycin—either will reduce the fever but will not alter the eventual course of the disease.
2. Elevation of the scrotum and applications of heat or cold.
3. Incision and drainage of the testicle (If the epididymis is incised, pus is nearly always found and varies in amount from a few drops to a half ounce. Incision and drainage usually give prompt relief of pain but do not otherwise influence the course of the disease. The acute infection may progress to abscess formation requiring drainage. Sterility of the affected testicle usually follows an epididymitis, probably due to obstruction.)

TUBERCULOUS EPIDIDYMITIS.—(See page 878.)

SYPHILITIC EPIDIDYMITIS.—Very rare—(syphilitic orchitis in the latent stage of the disease is not uncommon).

Vas

Injuries to the vas are uncommon. The vas may be severed or damaged in hernia operations, most commonly in children. Inflammations of the vas, either gonorrheal, nonspecific, or tuberculous, are more common and cause pain in the inguinal region. With the exception of tuberculosis, they tend to subside when the associated epididymitis present resolves under treatment. On rare occasions localized abscesses form and require drainage. Beading of the vas on palpation is a sign of previous inflammation.

Vessels

Injuries of the Vessels.—Injuries are uncommon, with the exception of injury to a varicocele, which may cause rupture of some of the vessels. Extravasation of blood into the scrotum occurs. This tends to be absorbed if the patient is placed at rest.

Varicocele.—A varicocele is that condition in which the veins of the pampiniform plexus have become varicose. The extent varies. It is usually associated with a lax pendulous scrotum. It is relatively common on the left side in young men, very rare on the right side, and occasionally bilateral. It is described as "a bag of worms" because of the appearance on the surface of the scrotum. In older men it may be due to venous obstruction caused by the pressure of a renal tumor.

There are several theories as to its cause, none of which is entirely satisfactory. The two most common reasons given are (1) incompetence of the valves in the left spermatic vein and (2) the greater intravenous pressure in the left spermatic vein. The stagnation of blood may result in partial atrophy of the testicle.

Signs and Symptoms—

1. Varicocele may be asymptomatic, or the patient may complain of pain in the testicle. In a great many cases there is a psychoneurotic element, as the patient is conscious of an inferiority rather than pain.

2. When the patient stands, the vessels of the pampiniform plexus become distended and readily palpable. On recumbency with elevation of the scrotum, the vessels empty

Treatment.—

1. Support the scrotum with a suspensory belt. Cold baths are sometimes helpful.
2. Operation in selected cases. The majority of the veins are isolated from the vas, making certain to leave the artery of the vas and a few veins behind. The main group is divided and a 4 cm. length excised. Each end is tied with a transfixing ligature and then approximated by tying the ligatures together.

Testicle

Contusions.—Blows to the testicle are fairly common. The majority of such injuries are fortunately slight. Severe trauma will cause ecchymosis of the scrotum, acute pain, swelling of the testicle, and exquisite tenderness. When the acute episode subsides, atrophy may follow.

Treatment includes bed rest, elevation of the scrotum, and ice bags to the scrotum.

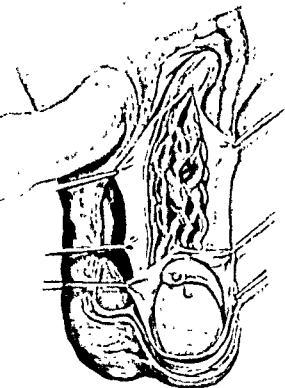
Wounds of the Testicle.—Stabbing wounds of the testicle are uncommon. Extensive lacerations of the scrotum, with partial or complete loss of glandular tissue, are more common in the accidents of industries and modern warfare. After treating shock, the area is thoroughly cleansed with green soap and water, debrided, and every effort made to preserve as much of the testicle as possible. Usually very little tissue can be saved as the tunica albuginea is extensively torn and the seminiferous tubules bulge through the rent.

Inflammations.—

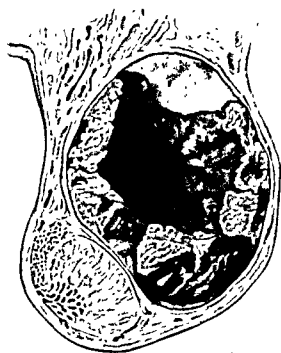
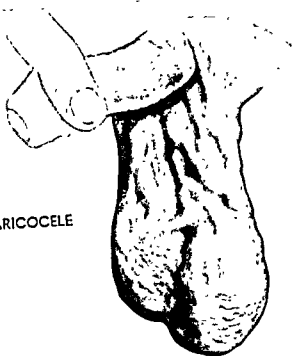
Acute orchitis.—Acute orchitis, nonspecific, may occur, as a rare complication of any generalized infection. It is characterized by a sharp rise of temperature, chills, headache, pronounced weakness, leukocytosis, pain, and swelling of the testicle. The epididymis and spermatic cord may be involved.

Treatment consists of elevation of the scrotum and the application of heat. Incision and drainage may be required later and occasionally orchectomy. Even if drainage is not required, testicular atrophy usually ensues.

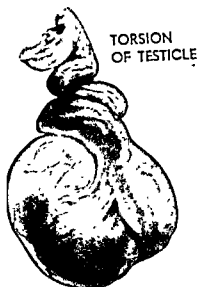
Orchitis of Mumps.—Acute orchitis occurs as a complication of mumps in 20% of cases. The incidence in infants is much less. Testicular atrophy follows in over half the cases. It has been claimed by some that the atro-



VARICOCELE



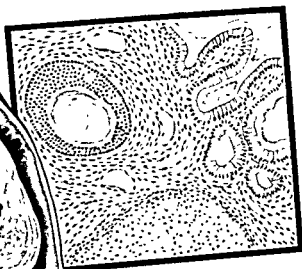
HEMATOCELE



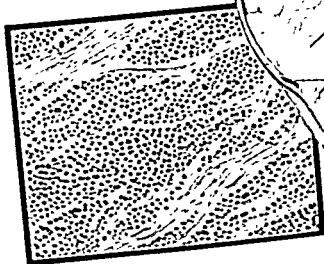
TORSION
OF TESTIS

F. Netter M.D.

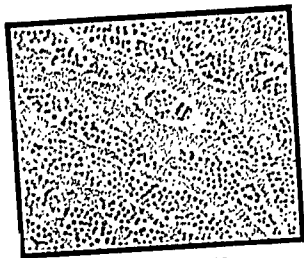
Plate 46 — Varicocele, Torsion of the Testis, and Hematocele.



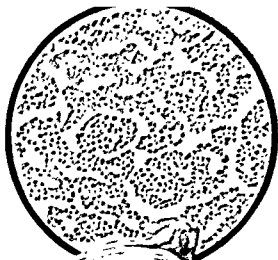
MULTICELLULAR TUMOR
(Adult Teratoma)



UNICELLULAR TUMOR
(Seminoma)



UNICELLULAR TUMOR
[with lymphoid stroma]



MULTICELLULAR TUMOR
(Embryonal carcinoma)



MULTICELLULAR TUMOR
(Choriocarcinoma)



F. Netter, M.D.

Plate 48.—Malignant Tumors of the Testicle.

is due to an acute inflammatory hydrocele, and that if this is drained during the acute stage, atrophy of the testicle will not ensue. These claims, however, do not appear to be true, as testicular biopsy clearly shows that an acute inflammatory reaction occurs within the substance of the testicle, which explains the subsequent atrophy.

Chronic Orchitis—(1) Tuberculous and (2) syphilitic

Clinically, syphilitic infections of the testicle are rare. Yet at autopsy, syphilitic fibrosis of the body of the testicle is so common that it can be said that syphilis attacks the testicle in preference to any other tissue, with the possible exception of the heart and aorta.

Latent syphilitic orchitis occurs frequently and often passes unnoticed.

Congenital syphilitic orchitis develops from the 3rd-10th month of life and results in diffuse fibrosis and atrophy (of the testicle).

Gumma of testis is a late manifestation of syphilis.

Neuralgia of Testicle—The complaint of pain in the testicle, usually intermittent, gnawing in character, and frequently severe, is most difficult to treat. It may be caused by involvement of the genitofemoral nerve in scar tissue, hernia, prostatitis, a localized healed lesion in the testicle, or low ureteral stone.

Torsion of the Testicle.—Torsion of the testicle is, in fact, rotation or torsion of the spermatic cord, which shuts off the blood supply to the testicle. Torsion can occur only if the epididymis is not attached to the posterior scrotal wall and if there is a complete and high investment of the testis and epididymis by the tunica vaginalis. If the tunica vaginalis is attached high to the cord, contraction of the cremasteric muscle with its spiral fibers may cause complete rotation of the testicle, shutting off the blood supply. The torsion is usually anticlockwise and nearly always intravaginal. The etiology is unknown.

Symptoms and Signs.—

1. Sudden acute onset of pain in the testicle, which may be agonizing.
2. Elevation of the testicle in the scrotum.
3. Prehn's sign—elevation of the scrotum accentuating the pain.

1. If of long duration, swelling of the scrotum with redness of the overlying skin; also some elevation of temperature.

Treatment.—

1. If soon after onset—try to reduce the torsion manually, by twisting the supported testis from within outward. If this increases the pain, twist it in the opposite direction.

2. Up to 24 hours after onset—expose the testicle through an inguinal incision, untwist it, return it to the scrotum, and suture it there.

3. If the torsion has existed longer than 24 hours, the testis will appear blue-black and lifeless, in which case it should be removed.

Cryptorchidism.—Cryptorchidism is congenital malposition of the testicle. It may be the following:

1. Abdominal (nondescent)
2. Inguinal (partial descent)
3. Retention of the testicle in the upper part of the scrotum
4. Perineal
5. Penile

Most cases encountered clinically are in the inguinal region and due to incomplete descent. They are underdeveloped and are frequently subjected to repeated trauma. For years it has been claimed that the incidence of malignancy in an undescended testicle is exceedingly high as compared to a normally descended testicle. Recent work would indicate that this is not true and that the incidence of malignancy is no higher than in a normally descended testis. Atrophy of the testicle with aspermatogenesis is a frequent finding.

TREATMENT FOR INCOMPLETE DESCENT OF THE TESTICLE (CRYPTORCHIDISM).—The ideal age for treatment is up to 9-11 years.

1. *Hormonal*: In some children the testes are either high in the scrotum or in the inguinal canal. In some of these, the injection of 500 units of anterior pituitary-like substance (A.P.L.) daily for 20 doses, followed by a rest period of 10 days, and then another series of 10 injections will cause the testes to descend. In many cases, however, the testicle is bound down to the surrounding tissue by adhesions. In these, A.P.L. will not cause the testicle to descend.

In practice, it is customary to give a course of A.P.L. prior to proceeding with surgery. If the testicle does not descend and surgery is

required, it is found that the tissues are more pliable after a course of A.P.L. and the testicle has an increased blood supply.

2. *Surgical*: If hormonal therapy fails to bring the testicle down into the scrotum, the inguinal canal should be opened, the cord and testicle found, and the congenital hernia repaired. The cord can then be lengthened sufficiently to allow positioning of the testicle in the new bed made for it in the scrotum.

Tumors of the Testicle.—The views held regarding tumors of the testicle have altered considerably during the past 30 years. In 1929 Zondek discovered prolan A in the urine of a patient suffering from teratoma testis. Soon it became common practice to regard the quantitative amount of prolan A found in the urine as an index to the type of tumor present and the prognosis. Opinion has now shifted so that it may be said that, whereas a positive prolan A in the urine indicates the presence of a testicular tumor, a negative prolan A does not exclude a tumor. In chorionepithelioma there is a very high excretion of prolan A.

Classification—

<i>Tumors of the Testicle</i>	<i>Incidence</i>
1 Seminoma (germinoma)	35%
2 Teratoma	42%
3 Embryonal carcinoma (including chorionepithelioma)	23%

Incidence—Tumors of the testicle are uncommon but occur with sufficient frequency to be important. They form approximately 20% of all tumors in the male, and are commonest from 20-40 years of age. Testicular neoplasms may be found at any age, even in the newborn, and most clinicians and pathologists agree that they are all malignant or potentially so.

Seminoma (Synonyms; *germinoma*; or *embryonal carcinoma with lymphoid stroma*)—One third of all testicular tumors are monocellular seminomas made up of rounded polyhedral elements with sharp cell borders. The cytoplasm is often clear. The cells are usually arranged in unorganized masses of cords divided by trabeculae of connective tissue. Variations in structure may occur. The lymphocytic infiltration of the connective tissue trabeculae in seminomas has led some clinics to use the term *embryonal carcinoma*, with

lymphoid stroma. This is considered to be a misleading term. Foci of necrosis are often encountered. Invasion of the adnexae and cord is exceptional, although in embryonal carcinomas it is the rule.

The resemblance between seminoma cells and some cells of the seminiferous tubules is taken as evidence that seminomas arise from spermatogonia cells of the seminiferous tubules.

Teratoma—There is a great range of variation in the structure of teratomas. Some of the more appalling facts about tumors of the testicle are that (1) 42% are either misdiagnosed or undiagnosed prior to admission (Schwartz and Mallis), (2) an average of 7.5 months elapses from the time when the patient first seeks medical attention to the time that he is referred for surgery (Schwartz and Mallis), and (3) 43% have metastases at the time of admission (Lewis). They are characterized by the presence of epithelial masses, glands, and cysts. Many of these structures are organized in combination with undifferentiated or specialized mesenchymal tissues such as cartilage. Transitions occur and the resulting picture is one of mixed figures in which some semblance of organization can often be discerned. Squamous cell nests and cysts, often keratinizing, are frequent; glandular appendages and adult hair are rare. Epidermoid cysts outnumber dermoid cysts 10:1.

Embryonal Carcinoma—Tumors of this type, which include embryonal adenocarcinoma and papillary adenocarcinoma, are often confused with seminomas, although they differ not only in fundamental cell type but in biologic behavior and prognosis as well. They show considerable variation in cell type, e.g., epithelial cells, cuboidal cells, columnar cells, and papillary structures. The cell type is of no known biologic significance and does not determine the prognosis.

Chorionepithelioma—These tumors may be suspected grossly because they are strikingly hemorrhagic. In addition they produce a very high excretion of prolan A in the urine (100,000 or more mouse units/liter).

Microscopically they are easily recognized because of their characteristic appearance. They have two cellular components arranged so as to duplicate the architecture of the placental

illi. The combination of compactly grouped cytotrophoblastic cells and giant multinucleated syncytial structures, arranged at the borders of the cellular masses, makes one of the most striking microscopic pictures in all pathology.

Signs and Symptoms of Tumor of the Testicle—Tumor of the testicle may occur at any age, but 80% occur from 20-40 years of age. There are two cardinal symptoms:

- 1 A painless swelling in the testicle.
2. Sense of weight in the scrotum. Attention is often drawn to the testicle by some insignificant trauma, or the patient may have noticed a gradual enlargement to which at first he paid little attention
- 3 The first symptom noted may be the result of metastases or of lymphatic involvement, e.g., loss of weight, abdominal pain, pain in the back or spine, or a slight cough
4. On palpation, the testicle is usually smooth and regular in early cases but nodular and cystic in later cases. The skin is red and shiny from stretching, the surface blood vessels are dilated, and at a very late stage, ulceration through the scrotum may occur

5 The presence of prolan A in the urine, i.e., a positive Aschheim-Zondek test, is indicative of tumor being present in the testicle, but a negative test does not exclude tumor

Differential Diagnosis.—

1. Tumor
2. Hydrocele (often present with tumor)
- 3 Hematocele
4. Tuberculosis
- 5 Syphilis

All testicular swellings must be regarded as malignant until proved otherwise

Metastases—Metastases occur via the lymphatics to the deep abdominal and aortic nodes and via the blood stream to give widespread distal metastases. The lungs are most commonly involved. Blood stream invasion may occur early.

Prognosis—The prognosis in testicular tumors is very poor. There is a 1-5-year cure in many cases of seminoma. So far, there has been no case of chorionepithelioma known to be cured. Taking all groups together, 40% are dead 2 years after surgery. The 5-year survival of all groups is 20%. The prognosis depends in part upon (1) the extent and rapidity with which the tumor grows and metastasizes and (2) the radiosensitivity of the tumor, e.g., abdominal metastases sometimes appear to melt away (temporarily) following radiation of the abdomen.

Treatment.—In the treatment of testicular tumor, one of several courses may be pursued as follows:

1. Simple orchiectomy
2. Orchiectomy and irradiation
- 3 Radical operation with removal of the regional and retroperitoneal lymph nodes on the affected side, followed by deep x-ray irradiation
4. Irradiation alone
5. Bilateral orchiectomy

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Film References

Title	Running Time	Sound or Silent	Procurable From
Kidney Function in Disease (1948) (By Arthur C. Corcoran, M.D., Don Carlos Hines, M.D., and Irvine H. Page, M.D., Indianapolis)	45 min	Sound Color	Eli Lilly & Co. Indianapolis 6 Indiana

GENITOURINARY SYSTEM

Film References—Cont'd

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Kidney Function in Health (1948) (By Arthur C. Corcoran, M.D., Don Carlos Hines, M.D., and Irvine H. Page, M.D., Indianapolis)	36 min	Sound Color	Eli Lilly & Co Indianapolis 6 Indiana
Transperitoneal Nephrectomy (Transverse Abdominal Incision) (1949) (By Charles Montgomery Stewart, M.D., Los Angeles)	10 min.	Silent Color	E. R. Squibb & Sons 745 Fifth Ave. New York 22, N. Y.
Use of the Artificial Kidney (Depicts the action of the artificial kidney) (1950) (By John P. Merrill, M.D., Boston)	14 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn.
Transplantation of Ureters Into Rectosigmoid and Cystectomy for Carcinoma of the Bladder (Illustrates technique of cystectomy, prostatectomy, and seminal vesiculectomy with dissection of lymph glands for carcinoma of the bladder) (1951) (By Charles C. Higgins, M.D., Cleveland)	27 min	Silent Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.
Urinary Infections Bacteriology, Pathology and Treatment (Traces the source of urinary infections and their underlying bacteriopathology and is suitable for undergraduate as well as postgraduate education) (1951) (By Grayson Carroll, M.D., Hollis Allen, St. Louis, and Victor Marshall, M.D., New York, collaborating)	40 min	Sound Color	Medical Film Guild 506 W. 57th St. New York 19, N. Y.

Chapter 32

Diagnosis of Acute Abdominal Conditions

James R. McCarrison, M.D.

An acute abdominal condition is one in which the patient is rendered acutely ill and prompt treatment is urgently required. An early and accurate diagnosis is therefore most important in order that appropriate therapy may be instituted before the situation becomes worse. In many instances immediate or early surgical intervention is necessary, but, in others, conservative forms of therapy are required and operative procedures definitely contraindicated.

Several fundamental principles should govern the method of diagnosis of an acute abdominal condition. There is the necessity of making a serious and thorough attempt at an early diagnosis, which is based on the following:

1. A careful and complete history of the present illness and of events in the past that might conceivably have some bearing on the present situation

2. A painstaking examination

3. The application of a thorough knowledge of anatomy and physiology

4. The possession of a broad concept of the various diagnostic possibilities; errors of diagnosis more often made through failure to consider possibilities than from lack of ability to distinguish among various clinical entities under consideration

5. The exclusion of nonsurgical conditions

HISTORY

The history of the present illness must include a detailed account by the patient, rela-

tives, or friends who know the facts of its onset and the development of symptoms. This should always be supplemented by the history of previous illnesses and the state of health of the patient.

SYMPTOMS EXPERIENCED BY THE PATIENT

Pain.—Abdominal pain is one of the most common symptoms which calls for speedy diagnosis and treatment (Cope). Pain is present in almost every case of acute abdominal disease, and often it is of such severity that the indication for surgical treatment is immediately apparent. However, when the symptoms are indefinite, the temptation to temporize must be resisted and a conscientious attempt made to arrive at the diagnosis when the patient is first seen. By making such an attempt, dangerous carelessness will be avoided, and a correct diagnosis will lead to correct treatment. Narcotic agents (e.g., morphine) should not be given until the diagnosis is clear, because they mask symptoms and signs of severe, progressive intra-abdominal disease, which may require immediate surgical treatment. Cope has laid down the rule that the majority of severe abdominal pains which occur in patients who have previously been fairly well, and which last as long as 6 hours, are caused by conditions of surgical import. The earlier the diagnosis and treatment, the higher is the recovery rate from acute abdominal disease.

Film References—Cont'd

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
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James R. McCorriston, M.D.

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Information as to the character of the pain is helpful. Gripping pain is characteristic of intestinal obstruction, whereas an intra-abdominal abscess, such as a pelvic abscess, produces acute throbbing pain. Alteration of pain by respiratory movements is significant. The pain of pleurisy is made worse during inspiration and is diminished or abolished by temporary

frequently after unusual physical activity. It is necessary to know the time of onset of symptoms in estimating the probable extent of pathologic changes that have taken place.

The location sequence of pain is illustrated by typical acute appendicitis in which vague upper abdominal distress is first noticed, then periumbilical pain, and, finally, right lower quadrant pain. Another example is renal pain beginning in the costovertebral angle and referred to the external genitalia.

The patient is usually able to indicate the point of maximum pain. Examples of situations in which this is of diagnostic importance include local pain of strangulated hernia and mid-epigastric pain of pancreatitis.

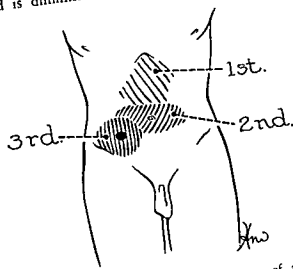


Fig. 456—Location sequence of the pain of acute appendicitis

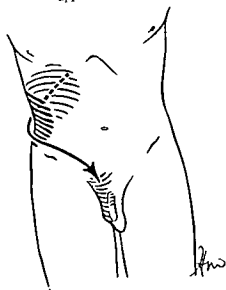


Fig. 457—Location sequence of renal pain

cessation of breathing. In most cases, pain of peritonitis is accentuated by deep inspiration, coughing, or movement.

The time sequence of symptoms is sometimes diagnostic, e.g., pain of peptic ulcer before and after meals, upper abdominal pain of gall bladder disease after a fatty meal and

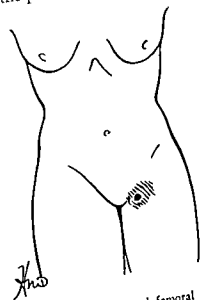


Fig. 458—Mass of incarcerated femoral hernia a point of maximum pain and tenderness

Vomiting.—In acute abdominal disease vomiting is a common symptom of importance. Vomiting begins shortly after the pain, when there is sudden severe stimulation of the peritoneum or mesentery (e.g., perforated ulcer, high small bowel obstruction), but it may begin much later than the pain in other instances (e.g., large bowel obstruction). Vomiting is usually more frequent when the condition is acute but infrequent or absent when subacute. Nausea or loss of appetite as an initial symptom is often just as significant as vomiting (e.g., in acute appendicitis). The character of the vomitus should always be noted. When vomiting commences, the vomitus consists of stomach contents, but later it con-

tains yellow bile. When the vomitus changes to a dark green or black color, or later to a feculent type, paralytic ileus or intestinal obstruction is likely to be present.

Bowel Function.—Information concerning the presence of *constipation*, *diarrhea*, or *change of bowel habit* may be very useful. The nature of the stool and whether or not it contains blood, pus, or mucus are often significant (e.g., carcinoma of colon, diverticulitis).

Menstrual History.—Knowledge of the menstrual history is essential when trying to diagnose an acute abdominal condition in a female. Amenorrhea or an unusual type of menstrual period may indicate the likelihood

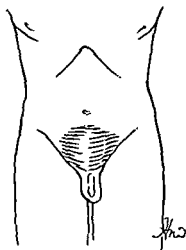


Fig. 459.—Local hypogastric distention owing to distended urinary bladder (dullness on percussion)

of pregnancy if the patient is of childbearing age

Urinary Function.—The history of urinary function is very important. Inability to pass urine or frequent voiding of small amounts is characteristic of urinary retention. Frequency of urination and dysuria are usually present when there is inflammation in or near some part of the urinary tract.

Age.—The age of the patient is of considerable diagnostic significance. Acute appendicitis may occur at any age, acute intussusception is commonly found in children under the age of 2 years, and acute obstruction of the colon due to carcinoma is uncommon in children and young adults

PHYSICAL EXAMINATION

A general physical examination of the patient should always be carried out. The examination should begin with *inspection*. The attitude in bed, facial expression, perspiration, respiratory movements, bodily movements, skin color, degree of tenseness, and other general characteristics are noted. Movement of the alae nasi and rapid breathing direct attention to the possibility of respiratory disease, extreme pallor associated with gasping respirations is suggestive of massive hemorrhage, and cold, clammy face and extremities indicate the imminence or presence of oligemic shock. Peritonitis causes the patient to remain immobile in order to avoid accentuation of pain, whereas the severe colics lead to restlessness.

The *pulse* is often unaltered early in acute abdominal conditions but will be weak and rapid if shock is present. The pulse rate is nearly always increased when hemorrhage or peritonitis is advanced. The pulse cannot be relied upon as a guide in the early stages of acute abdominal disease.

The *body temperature* may be subnormal, normal, or elevated. During a phase of shock it is likely to be quite low. In acute appendicitis or cholecystitis the temperature may be normal or elevated, but it often falls sharply if perforation of the viscus occurs. When the temperature is very high the chest or urinary tract should be suspected as a site of infection. Rigors or chills are frequent when fever is present in urinary tract infections

Inspection of the tongue is often helpful, as the tongue is usually coated or furred in acute abdominal conditions. In addition, the breath may be foul, particularly in intestinal obstruction.

The head and neck, eyes, ears, nose, throat, chest, and cardiovascular system should be examined, followed by the spinal column and nervous system.

Abdominal Examination.—Finally, a careful examination of the abdomen is performed. *Inspection* is very important and, with appropriate lighting, abnormal local or general distention, the presence of a large mass or external hernia, visible peristalsis, degree of mobility of the abdominal wall, and discoloration of the skin may be appreciated

Hyperesthesia is sought for by pin stroke or pinch. The point of the pin is drawn downward in vertical strokes over the anterior abdominal wall and loins. Hyperesthesia may appear in the distribution of peripheral nerves, whose branches are irritated by an inflammatory process, or it may appear in the segmental distribution of that part of the spinal cord which innervates the affected viscus. In acute appendicitis, hyperesthesia is frequently demonstrable over the abdominal distribution of the right 10th and 11th thoracic nerves.

The examiner, after making certain that his hand is warm, should begin *palpation* of the abdomen at a site distant from the point of maximum pain indicated by the patient. The hand should be applied with the palm flat on the skin and gentle pressure exerted with the pulps of the fingers. By this means the texture and temperature of the skin may be determined, muscular rigidity or contraction

appreciated, areas of tenderness located, masses felt, and movements of organs or abdominal masses with respiration detected. Contralateral tenderness on deep palpation of one side of the abdomen is often present in acute intra-abdominal disease but is absent in intrathoracic disease. If the patient flexes his hips, the abdominal wall will be more relaxed and the examination more satisfactory.

Muscular rigidity may be marked in conditions which cause severe irritation of the parietal peritoneum, as is the case in perforated peptic ulcer or traumatic rupture of a viscus. It is important to realize that rigidity may be minimal or absent in these acute conditions when the abdominal muscles are weak or flabby, when the patient is very ill due to toxemia, and when the patient is elderly or debilitated. It is a well-known fact that many aged patients exhibit much less pronounced symptoms and signs of acute abdominal disease than do younger adults and children. In oligemic shock with marked hypotension, rigidity may be reduced or completely obliterated despite the presence of severe peritoneal irritation. Simultaneous palpation of both rectus muscles is of value in assessing the character and degree of spasm. Unilateral rectus spasm may appear reflexly during renal colic but not in peritonitis, because there is nothing to limit the spread of irritating intraperitoneal fluid to one side. The loins are best palpated by the use of both hands, by which means masses and muscular rigidity are readily detected. Palpation of the abdomen after administration of a preoperative narcotic agent or after induction of anesthesia prior to laparotomy may make possible the detection of a

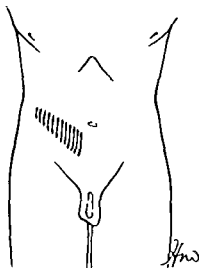


Fig. 150. Tenderness in acute appendicitis.

mass not previously felt because of intense muscle spasm

All hernial orifices must be scrupulously palpated, for hernias may be small and easily overlooked. Palpation of the femoral pulsations must be carried out, as one or both are absent in aortic or iliac arterial occlusion. When there is acute inflammation in relation to the psoas muscle, psoas rigidity is usually produced with flexion of the corresponding hip.

Percussion of the abdomen is of value in outlining the contour of the liver and spleen, in demonstrating free gas in the peritoneal cavity (especially if it obliterates liver dullness), and in outlining areas of resonance over gas-filled bowel or dullness over solid masses.

Rebound pain is elicited by pressing deeply on the side of the abdomen away from a suspected inflammatory lesion and then quickly releasing the pressure. As the abdominal wall springs out to its original position, sudden pain is felt at the site of pressure or at the site of the inflammatory lesion.

Accentuation or relief of pain on change of position is often of significance. An example of this phenomenon is increase of pain and nausea on change from the supine to the sitting position when volvulus of bowel is present. This condition is due to increased tension on the inflamed and stretched mesentery.

Careful and prolonged *auscultation* of the abdomen should never be omitted. Peristalsis may be increased, diminished, or absent in the presence of an acute abdominal condition. Peristalsis is absent if no sounds are heard over a period of several minutes, a condition that indicates the presence of paralytic ileus owing to diffuse peritoneal irritation. There are two common forms of increased peristalsis. Constant loud borborygmi of varying intensity but no definite pattern occur in acute gastroenteritis. This type of peristalsis has no definite rhythm, and its intensity varies without corresponding change in the associated abdominal discomfort. In acute mechanical bowel obstruction, abnormal sounds are produced by the rhythmic contractions of the intestines. Borborygmi of gradually increasing

intensity occur, which rise to a crescendo and then abate until only scattered tinkling sounds may be heard. The patient complains of cramps which come and go with the peristaltic activity. In chronic partial obstruction of the distal small bowel and during recovery from peritonitis, hollow gurgling and tinkling sounds are produced in the dilated, partially fluid-filled loops of bowel undergoing periodic contractions. These sounds are not rhythmic and may or may not be accompanied by abdominal cramps. Occasionally friction sounds are heard in peritonitis, and the transmission of cardiac and respiratory sounds through the abdomen occurs in the presence of intestinal distention and peritoneal exudate.

Rectal examination (with or without anoscopic and sigmoidoscopic examination) must never be omitted because it affords considerable information concerning the pelvic cavity. In males, anterior palpation will reveal abnormalities of the prostate or seminal vesicles, as well as fullness of the bladder. In females, anterior rectal palpation reveals abnormalities of the uterus and swellings in the pouch of Douglas. In both males and females high anterior palpation will reveal tenderness of the pelvic peritoneum.

High palpation along the rectal canal will reveal the presence of rectal lesions, impaction of feces, and foreign bodies. Lateral palpation may reveal a mass or elicit tenderness. Posteriorly, any mass or tenderness in the hollow of the sacrum or in the region of the sciatic foramina can be palpated.

After the gloved finger is withdrawn, it can be inspected for the presence of blood, pus, or mucus. Feces or other material present may be smeared on a piece of filter paper for the chemical detection of occult blood. Bimanual rectoabdominal examination is particularly valuable and in children permits exploration of the lower abdominal cavity as well as the pelvis.

In adult females, *vaginoabdominal* examination may elicit additional information. This is particularly valuable in examination of the female pelvic organs as it permits accurate evaluation of the size, shape, position, mobility, and tenderness of pelvic organs, tumors and masses (see Chapter 28, Female Genital Tract).

SPECIFIC TESTS AND SIGNS

The *shake test* is frequently of great value when the patient has difficulty identifying the point of maximum abdominal pain. To perform the test, the patient's pelvis is shaken transversely as he lies relaxed in the supine position. When the parietal peritoneum is irritated, this maneuver will cause pain and will enable the patient to indicate its exact location. A vigorous cough by the patient may yield the same information.

Fist percussion over the lower anterior thoracic wall causes acute pain in acute upper abdominal conditions (e.g., acute hepatitis, acute cholecystitis). It is performed by placing one hand flat on the skin and percussing it with the fist. Renal and spinal pain may also be revealed in this way.

The *psaos test* is performed by having the patient forcefully flex his thigh against the pressure of the examiner's hand. Pain is then experienced when there is an inflammatory lesion in contact with the psoas or iliacus muscle. Lesser degrees of psoas spasm may be demonstrated by the production of pain and resistance when the examiner hyperextends the patient's hip (e.g., in acute appendicitis).

The *obturator test* is useful in confirming the presence of an inflammatory lesion adjacent to the obturator internus muscle. When it is positive, medial rotation of the flexed thigh will produce hypogastric pain (e.g., in pelvic appendicitis).

Notable dullness is a characteristic sign of the presence of free fluid in the peritoneal cavity, and the sensation of a percussion wave transmitted through the fluid may be felt.

The *inspiratory sign of Murphy* can be demonstrated in acute cholecystitis. The patient is asked to take a deep breath while pressure is applied below the right costal margin. As the liver descends, the inflamed gall bladder is brought in contact with the depressed abdominal wall, and sharp pain is produced, causing immediate arrest of the inspiration.

In eliciting *Rovsing's sign*, even pressure is exerted over the descending colon, thus forcing gas around into the cecum, and pain is experienced in the right lower quadrant. This sign

is commonly present in appendicitis and indicates that inflammation of the cecum is present.

A *bluish discoloration* of the umbilicus and in the surrounding skin is occasionally found in cases of intra-abdominal hemorrhage. This is observed most frequently after rupture of an ectopic gestation and is called *Cullen's sign*.

Severe irritation of either the right or left half of the diaphragm produces *pain referred to the corresponding shoulder*. Examples are pain in the right shoulder with acute cholecystitis and pain in the left shoulder with ruptured spleen.

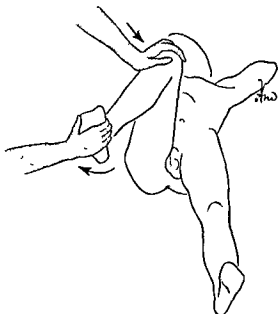


Fig. 462—The maneuver in the obturator test to stretch the obturator internus muscle.

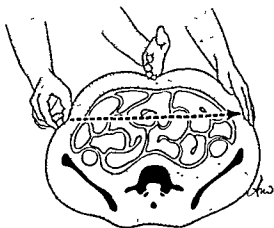


Fig. 463—Percussion wave through free fluid in peritoneal cavity.

LABORATORY AIDS

A complete *urinalysis* must always be carried out. Red blood cells are usually found in the urine in renal colic due to a stone and may be found in acute appendicitis. The presence of sugar in the urine is suggestive of diabetes mellitus, whereas pus cells and bacteria will lead the examiner to suspect the presence of urinary tract infection.

The *leukocyte count* is often of diagnostic importance but is occasionally unchanged in the early stages of an infective lesion, such as appendicitis or cholecystitis. Leukocytosis may be marked when an abscess or peritonitis is present. In suppurative lesions there is a relative increase in the polymorphonuclear leukocyte count. Leukopenia is characteristically found in typhoid fever and influenza.

The *hemoglobin level* should be determined in every case. It will be low after hemorrhage, provided that sufficient time has elapsed to permit hemodilution to occur. If dehydration is present, hemoconcentration is associated with elevation of the hemoglobin level and hematocrit reading.

When the presence of acute pancreatitis is suspected, an immediate *serum amylase* determination is of great value in confirming or excluding the diagnosis. It must be realized, however, that other conditions are occasionally accompanied by a significant elevation of the serum amylase level and that it may be normal or subnormal in the presence of extensive necrotizing pancreatitis.

ROENTGENOGRAPHIC EXAMINATION

Roentgenographic examination is often of great value in making or confirming the correct diagnosis of an acute abdominal condition. It is not always necessary, when the diagnosis is obvious or certain, but often provides much useful information (e.g., distention of coils of bowel and the presence of fluid levels in intestinal obstruction, separation of the gas bubble of the stomach from the diaphragm due to the presence of free blood after splenic rupture). X-ray films of the chest may reveal pulmonary disease which is not detectable by physical examination. Free gas in the perito-

neal cavity can almost always be detected by x-ray examination. When abdominal films are made, three projections should be used; i.e., in addition to a film with the patient supine, one should be made in an anteroposterior direction with the patient lying on his left side. If his condition permits, a film with the patient in an upright position should also be made. By such multiple exposures, shadows of free gas in the peritoneal cavity, gas-fluid levels in distended bowel or abscess cavity, radiopaque bodies, and soft tissue masses may be inspected and intelligently interpreted.

KNOWLEDGE OF ANATOMY AND PHYSIOLOGY

The examiner's knowledge of anatomy and physiology is often of aid in the interpretation of the physical signs and symptoms. From the anatomic point of view, the relationship of the abdominal organs to the parietal peritoneum and extraperitoneal structures should be carefully considered in every case of acute abdominal disease. Space-occupying or inflammatory lesions affect these structures and so produce symptoms and signs. Inflammation of the tissues adjacent to the psoas, quadratus lumborum, diaphragm, obturator internus, and anterior abdominal wall muscles causes irritation and reflex spasm which can be detected by examination. Examples include rigidity of the anterior abdominal muscles in acute peritonitis, a positive obturator test in pelvic appendicitis or acute salpingitis, and rigidity of the quadratus lumborum muscle in the presence of perinephric abscess. Knowledge of normal and abnormal physiology permits correct interpretation of many findings, such as the abdominal silence in paralytic ileus, the urinary frequency in pelvic peritonitis, and the pain in obstruction of a hollow viscus.

AWARENESS OF DIAGNOSTIC POSSIBILITIES

Nonsurgical abdominal diseases and extra-abdominal conditions must be excluded in the differential diagnosis of acute abdominal conditions. In order to do this the examiner must be aware of the *methods of detecting their presence*. Possession of a broad concept of the

various possibilities permits the examiner to come to a definite conclusion regarding the diagnosis, in most instances. Failure to consider possibilities is a common and serious error. Typical conditions are acute pancreatitis, acute tuberculous peritonitis, pleurisy, lead poisoning, and small bowel obstruction

The following classification of acute abdominal conditions and disorders which may simulate them is made partly on an anatomic basis. The conditions listed in italics are those for which immediate or early operative treatment is usually necessary

Acute Abdominal Conditions

- I STOMACH AND FIRST PART OF DUODENUM
 - 1 *Perforated ulcer*
 - 2 *Massive hemorrhage*
 - 3 Acute dilatation of stomach
 - 4 *Volvulus*
 - 5 *Acute phlegmonous gastritis*
- II SMALL INTESTINE
 1. Acute obstruction
 - a *Hernias*
 - b *Adhesions*
 - c *Inflammatory lesions*
 - d Paralytic ileus
 - e *Volvulus*
 - f *Intussusception*
 - g *Tumors*
 - h *Foreign bodies*
 - 2 Regional enteritis
 - 3 *Meckelian diverticulum*
 - 4 *Perforation*
- III LARGE INTESTINE
 - 1 Acute obstruction
 - a *Tumor*
 - b *Volvulus*
 - c *Adhesions*
 - d *Inflammatory stenosis*
 - e Paralytic ileus
 - f Fecal impaction
 - g *Hernias*
 - h *Intussusception*
 - i *Foreign bodies*
 - 2 Perforation
 - a *Traumatic*
 - b *Inflammatory*
 - c *Neoplastic*
 - 3 *Diverticulitis*
- IV APPENDIX
 - Acute appendicitis*
- V. GALL BLADDER
 1. *Acute cholecystitis*
 2. Biliary colic
- VI PANCREAS
 - Acute pancreatitis*
- VII. LIVER
 1. *Abscess.*
 2. *Rupture*
 - 3 *Acute hepatitis*
- VIII SPLEEN
 - 1 *Rupture*
 - 2 *Infarction*
- IX PERITONEUM
 - Peritonitis*
- X FEMALE GENERATIVE ORGANS
 - 1 *Ruptured graafian follicle*
 - 2 *Ruptured ectopic pregnancy*
 - 3 *Twisted ovarian cyst*
 - 4 Pelvic inflammatory disease
 - 5 *Ruptured ovarian cyst*
 - 6 Retroplacental hemorrhage
 - 7 *Rupture of pregnant uterus*
 - 8 *Abdominal pregnancy at term*
- XI GENITOURINARY SYSTEM
 - 1 Calculus
 - 2 *Perinephric abscess*
 - 3 *Rupture*
 - 4 *Torsion of ectopic testis*
 - 5 *Acute pyelitis*
 - 6 Urinary retention
- XII VASCULAR SYSTEM
 - 1 *Mesenteric vascular occlusion (arterial, venous)*
 - 2 Systemic arterial occlusion (aorta, iliac artery)
 - 3 Dissecting aortic aneurysm
 - 4 *Ruptured abdominal aneurysm*
 - 5 Pylephlebitis
- XIII LYMPHATIC SYSTEM
 - Acute mesenteric lymphadenitis*
- XIV MISCELLANEOUS CONDITIONS
 1. *Penetrating and perforating wounds*
 - 2 *Indirect and blunt trauma*
 - 3 *Wound dehiscence and evisceration*
 - 4 *Retroperitoneal hemorrhage*
 - 5 *Hematoma in rectus abdominis sheath*
 - 6 *Postoperative intra-abdominal hemorrhage*
 - 7 *Acute torsion of greater omentum or of appendix epiploica*
 - 8 *Ruptured diaphragm*

Conditions Which May Simulate Acute Abdominal Conditions

- 1 Pneumonia and pleurisy
- 2 Coronary occlusion
- 3 Diabetic acidosis
- 4 Periaarteritis nodosa
- 5 Tabes dorsalis
- 6 Lead poisoning
- 7 Herpes zoster
- 8 Influenza
- 9 Typhoid fever
- 10 Food poisoning and acute gastroenteritis
- 11 Blood disease (leukemia)
- 12 Tuberculous peritonitis

ACUTE ABDOMINAL CONDITIONS

STOMACH AND FIRST PART OF THE
DUODENUM

Perforated Ulcer

Perforation of a duodenal or gastric ulcer into the general peritoneal cavity is a sudden catastrophe. Delay in diagnosis and treatment may lead to the death of the patient. In some cases there is a history of symptoms of peptic ulcer or gastric carcinoma, but quite frequently there are few if any previous symptoms recalled by the patient. The signs and symptoms vary according to the period of time that has elapsed after occurrence of the perforation.

There are three generally recognized stages in a typical case. The *stage of prostration* is characterized by sudden agonizing epigastric pain which rapidly spreads over the entire abdomen, although often it is more marked on the right side. The extremities soon become cool or cold, there is marked perspiration, particularly about the face and neck, and the patient becomes livid or ashen in appearance. The expression becomes very anxious, and vomiting may occur. The vomitus contains undigested food and gastric juice, with or without blood-staining. The temperature is usually subnormal, the pulse small and thready, the respiratory movements entirely thoracic, and the blood pressure lowered. Characteristically, there is pain on the top of one (usually the right) or both shoulders. The knees are frequently drawn up in an effort to relieve the intense abdominal pain, and examination reveals extreme rigidity of the abdominal muscles and epigastric retraction. The patient lies perfectly still and avoids movement or change of position, because movement intensifies his terrible pain and may force him to cry out.

If sufficient gas has escaped into the peritoneal cavity through the perforation, there will be signs of pneumoperitoneum and consequent loss of liver dullness. The abdomen is tender throughout and often markedly so toward the right side. Examination of the rectum will reveal tenderness of the pelvic peritoneum. Peristaltic sounds diminish quite soon after the perforation occurs owing to

the inflammatory reaction which is set up by the presence of irritating gastric and duodenal contents in the peritoneal cavity.

The stage of prostration and shock passes off within a few minutes or an hour or two (depending upon the size of the perforation and the nature of the contaminating material) and merges into the so-called *stage of reaction*. The patient feels a little better, the symptoms of shock abate, and the temperature rises to normal. This is a dangerous latent period in which the unwary examiner may be lulled into a false sense of security. However, the pathologic process progresses relentlessly. During this stage the improvement is only relative

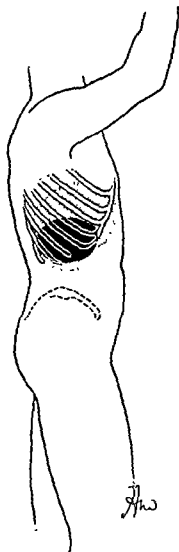


Fig 461—Area of resonance over lateral aspect of right lobe of liver as patient lies on left side, diagnostic of free gas in peritoneal cavity.

and abdominal pain continues. Examination reveals persistence of abdominal pain, rigidity, and tenderness, the respirations remain shallow and thoracic, the pelvic peritoneum becomes increasingly tender, and free gas and fluid in the peritoneal cavity may be detected. X-ray films of the abdomen will then confirm the presence of free gas, although, in approximately one fifth of the patients, free gas cannot be demonstrated. Movable dullness in the flanks may be demonstrable. An area of resonance over the lateral aspect of the right lobe of the liver, when the patient lies on his left side, is indicative of free gas over the liver. If previous peritonitis has resulted in adhesions in this region, this sign will be absent, and the presence of free gas will be revealed only by x-ray examination.

nal wall decreases, but the pelvic peritoneal tenderness persists.

There are four relatively common conditions which must be differentiated from perforated gastric or duodenal ulcer for which operative treatment is contraindicated. Biliary colic may involve severe abdominal pain and collapse but does not cause free gas in the peritoneal cavity, a tender pelvic peritoneum, or signs of severe generalized peritoneal irritation. A history of previous attacks of typical pain and jaundice is helpful. The pain of biliary colic radiates to the subscapular region. Concurrent jaundice indicates the true diagnosis.

Renal colic does not produce free gas, generalized peritoneal irritation, or tenderness

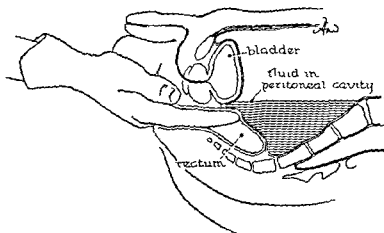


Fig. 465 —Palpation per rectum of pelvic peritoneum containing irritating fluid

The shoulder pain usually persists and, if bilateral, is suggestive of an anterior gastric perforation with irritation of both halves of the diaphragm. This pain is referred along the course of the supraclavicular nerves (C4), because sensory fibers of the phrenic nerve from the same spinal nerve roots are stimulated.

The final stage is that of *acute generalized peritonitis* characterized by distention, hic-cough, paralytic ileus, and frequent vomiting. The tenderness of the abdomen increases, the temperature may be normal, subnormal, or elevated, there may be chills, the pulse is very rapid and small, and the respirations are labored and rapid. The rigidity of the abdomi-

nal wall decreases, but the pelvic peritoneal tenderness persists. Previous episodes of hematuria or passage of a renal stone and radiation of the pain to the external genitalia are suggestive of the diagnosis. Hematuria is very significant, and radiologic visualization of the stone is diagnostic.

Acute bilateral or right-sided pleurisy may cause severe abdominal pain, but pulmonary signs will usually be detectable. Movement of the alae nasi and a rapid respiratory rate are suggestive of pulmonary disease. There will be no tenderness of the pelvic peritoneum, no free gas in the peritoneal cavity, and an x-ray film of the chest will reveal the pulmonary lesion.

In acute pancreatitis there may be agonizing abdominal pain, usually confined to the

epigastrium, with radiation to the back. Abdominal rigidity is not generalized and is rarely constant. A significantly elevated serum amylase level confirms the diagnosis. Free gas and tenderness of the pelvic peritoneum are absent, but vomiting and shock may be severe.

There are four other acute abdominal conditions which must be differentiated from perforated ulcer. Acute appendicitis with perforation can be differentiated by considering the history, the sequence of symptoms, and the local signs. The symptoms are rarely as severe as those of the initial stage of perforated ulcer but may be confused with those of the stage of reaction, especially if the perforation is small and the fluid trickles slowly down the

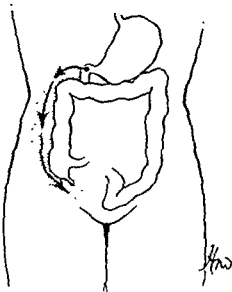


Fig 466.—Spread of irritating fluid down right paracolic gutter from small perforation of ulcer

right paracolic gutter into the right iliac fossa and pelvis. However, in appendicitis there is no loss of liver dullness, rarely any shoulder tip pain, no persistent tenderness over the duodenum or stomach, and no detectable free gas in the peritoneal cavity.

Intestinal obstruction presents no problem of differentiation except when the patient is first seen long after the onset of symptoms. Acute strangulation of bowel may lead to free fluid in the peritoneal cavity, collapse, severe pain, and repeated vomiting. In obstruction the

vomit becomes feculent. In the later stages both present the picture of peritonitis, but free gas in the abdomen will not be found in obstruction unless gangrenous bowel has ruptured.

Peritonitis due to other causes is differentiated by the history and the absence of free gas in the abdomen. It may be impossible to differentiate peritonitis due to perforation of other parts of the intestine.

In females ruptured ectopic gestation with massive hemorrhage into the abdominal cavity frequently leads to severe abdominal pain, vomiting, and collapse. A history of amenorrhea may be helpful but not diagnostic. Signs of massive hemorrhage with blanching of nails, lips, and mucous membranes are important findings. The pelvic peritoneum will be tender and free fluid demonstrable in the abdomen, but true loss of liver dullness does not occur, although shoulder tip pain from irritation of the diaphragm by blood may be a complaint.

It must be realized that *localized peritonitis* or *subphrenic abscess*, alone, may follow perforation of an ulcer. This may occur if the leak is very slow or is into the lesser peritoneal sac, permitting time for sealing-off from the general peritoneal cavity by fibrin deposits. In such an instance the signs and symptoms may be limited and less severe, being restricted to the upper portion of the abdomen, and the clinical picture will be that of *subphrenic abscess* (see Chapter 23, *Peritoneum, Omenta, and Mesenteries*). Very infrequently a small perforation will be sealed by deposition of fibrin and resolve after temporary signs and symptoms of upper abdominal irritation resembling those of acute cholecystitis or mild acute pancreatitis have disappeared.

Massive Hemorrhage

Massive hemorrhage from the stomach or first part of the duodenum is an acute abdominal condition easily diagnosed in most instances. The combination of hematemesis, hyperperistalsis, gross melena, and symptoms and signs of hemorrhagic shock is unmistakable. The history often provides evidence of previous hemorrhage, the peptic ulcer syndrome, or deterioration suggestive of a gastric

eploplasm. It must be differentiated from hemorrhage from esophageal varices which is a complication of portal hypertension. In the latter condition, symptoms and signs of portal hypertension, hepatic cirrhosis, or splenomegaly may be present. It is absolutely essential to determine the presence or absence of esophageal varices as early as possible by means of x-ray examination of the esophagus or, if need be, by esophagoscopy examination in the operating room after preparations for parotomies have been made and anesthesia induced.

Acute Dilatation of the Stomach

This is a condition in which there is sudden enormous distention of the stomach due to accumulation of fluid and gas. It is characterized by effortless regurgitant vomiting, absence of peristaltic sounds, rapid dehydration, hypochloremic alkalosis with or without edema, and severe collapse owing to oligemic shock. Epigastric pain may be present or absent. This condition is rapidly fatal if not relieved. The diagnosis is obvious when it occurs during an abdominal operation, owing to the swallowing of air or forcing of gas into the stomach under pressure. It usually follows abdominal operations and may be looked upon as a special variety of paralytic ileus, but it may occur in other illnesses (e.g., after trauma, during an acute febrile illness). The epigastrium and lower left chest are resonant, and the heart is displaced upward and to the left. Succussion splash may be demonstrated, and there is no abdominal rigidity. The vomitus consists of decomposing gastric, duodenal, and biliary secretions which gradually become rankish. Aspiration of gastric contents is the therapeutic and diagnostic test. The aforementioned characteristics of acute gastric dilatation differentiate it from high small bowel obstruction, peritonitis, uremia, drug poisoning, and intra-abdominal hemorrhage.

Volvulus of the Stomach

Volvulus of the stomach is an abnormal anterior or posterior rotation of a portion or the whole of the stomach about either the transverse or sagittal axis of its body (Maignot). It is extremely rare and cannot occur unless

the ligamentary supports of the stomach are abnormally long. The clinical features of acute volvulus are characteristic. Early vomiting is followed by intractable retching and inability to vomit. There is rapidly increasing distention limited to the epigastrium with simultaneous retraction of the lower abdominal wall. It is impossible to pass a tube into the stomach. These findings are due to initial pyloric obstruction followed by obstruction of the cardia and distention of the stomach by gas and fluids (closed-loop obstruction). The patient suddenly experiences agonizing epigastric pain radiating through to the back. If it is not relieved, signs of hypotensive shock soon appear. The only other condition requiring differentiation is *acute gastric dilatation*, which can be relieved by the passage of a gastric tube and aspiration of gastric contents. X-ray examination of the abdomen will reveal gastric distention with a gas-fluid level.

Acute Phlegmonous Gastritis

This extremely rare condition is a severe diffuse or localized inflammatory process involving the gastric submucosa which progresses to pus formation and perhaps necrosis of the gastric wall. It is difficult to diagnose. In the fulminating type of case the symptoms are those of acute toxemia, whereas the signs are those of peritonitis. Vomiting is intractable and the vomitus is blood-stained purulent material. There are hyperpyrexia, rapid pulse, marked leukocytosis, dry coated tongue, and epigastric rigidity and tenderness. Deterioration is rapid, and death ensues within a few days unless prompt treatment is given.

This condition must be differentiated from perforated peptic ulcer, acute pancreatitis, acute cholecystitis, pyogenic liver abscess, and hematogenous streptococcal or pneumococcal peritonitis. In phlegmonous gastritis a barium meal x-ray examination reveals an intact mucosa but a greatly thickened gastric wall. Perforated ulcer is differentiated by the presence of free gas in the peritoneal cavity and pancreatitis by the local signs and elevated serum amylase. Acute cholecystitis and liver abscess are differentiated by the local signs, history, and character of the vomitus. Primary peritonitis is differentiated by the history, local signs, and pure

culture of the causative organism from the peritoneal exudate aspirated by means of a needle.

SMALL INTESTINE

Acute Obstruction

Once complete obstruction of the intestine develops, death will inevitably result unless there is spontaneous correction of the condition, formation of an external fistula, or operative relief. Early diagnosis and treatment are essential because the mortality rate rises with each hour of delay. The symptoms vary in severity with the level at which the intestine is obstructed and are generally considered in relation to *high* or *low* small bowel obstruction. The onset of symptoms is slower the lower the level of obstruction. The obstruction tetrad of *crampy pain*, *vomiting*, *gradually increasing distention* and *obstipation* is usually present, although only one or two components may be apparent in the early stages. When there is strangulation of a segment of gut, the onset is very acute with severe symptoms: pain, shock, early vomiting, abdominal tenderness, and visible peristalsis. Ictulent vomitus, in the absence of advanced peritonitis, is characteristic of obstruction. Abdominal distention is not a prominent feature in early cases, especially if the point of obstruction is high, but always appears later. The outline of distended loops of small bowel may be visible or palpable, particularly in volvulus about an adhesive band. X-ray films of the abdomen are invaluable in revealing the extent of distention, the approximate site of obstruction, and the presence of fluid levels. Sigmoidoscopy and a barium enema x-ray examination will exclude obstruction of the large bowel. In advanced stages of small bowel obstruction there is evidence of rapidly increasing dehydration and toxemia, which progress to collapse, coma, and death if not relieved (see Chapter 24, Intestinal Obstruction). When there is strangulation of gut (hernia, volvulus), the abdominal tenderness is marked, peristalsis is inhibited or absent (paralytic ileus), vomiting is very severe, a tender mass may be palpable, and early fever and leukocytosis develop.

It may be impossible to differentiate the various forms of small bowel obstruction be-

fore exploratory laparotomy is performed. However, an incarcerated or strangulated external hernia can be diagnosed immediately by physical examination, and x-ray films may reveal a radiopaque foreign body such as a calcified gallstone in the small bowel (most frequently arrested in the ileum). Intussusception is usually found in the distal ileum and most commonly occurs in children under 2 years of age. The findings of a sausage-shaped mass in the abdomen, together with the passage of bloody stool in the presence of low small bowel obstruction, are diagnostic and a tender pelvic mass may be palpable on rectal examination. A barium enema x-ray examination will demonstrate the intussusception if it is of the ileocolic variety.

The differentiation of small bowel obstruction from *acute inflammatory abdominal conditions* may be difficult or impossible and they often coexist. Inflammatory conditions often cause secondary paralytic ileus even though the lumen of the gut is not obstructed (e.g., acute appendicitis, acute salpingitis).

Simple *constipation* can usually be diagnosed by the presence of impacted feces in the rectum. *Renal colic* and *biliary colic* are differentiated by the type of pain and local findings. *Acute pancreatitis* can be diagnosed by the history, local findings, and an elevated serum amylase level. *Acute coronary occlusion* is differentiated by the history, physical signs, and electrocardiographic changes, although it may be accompanied by a degree of paralytic ileus. *Acute pulmonary conditions* may cause unilateral upper abdominal pain but not the other signs of intestinal obstruction. *Enterocolitis* and *food poisoning* usually produce characteristic symptoms and signs, and the history combined with copious, often persistent, diarrhea differentiate them from bowel obstruction.

Regional Enteritis

An acute attack of this condition frequently produces right lower quadrant abdominal pain, fever, and leukocytosis. The pain is often *crampy* and may be associated with diarrhea, nutritional disturbances, and partial or complete intestinal obstruction. A tender mass may sometimes be palpated in the right lower quadrant. If perforation of the ileum occurs

there will be signs of acute localized peritonitis, since the process is usually limited by inflammatory adhesions

It may be impossible to differentiate regional enteritis from *appendicitis* or active *ulcerative colitis*. If confused with the former it will be diagnosed at operation, and if confused with the latter it may be recognized by barium x-ray studies of the intestines

Careful consideration of the history, the appearance of a complicating fistula, together with a high index of suspicion, will often lead to the correct diagnosis. Acute regional enteritis must also be distinguished from *tuberculous enteritis*, *actinomycosis* of the ileocecal region, *diverticulitis* of the ascending colon or cecum, and *ulcerated neoplasms* of the small intestine and cecum

Meckelian Diverticulitis

Acute inflammation, with or without perforation, of a Meckel's diverticulum can seldom be differentiated from *acute appendicitis*, and the differential diagnosis will be dealt with under that heading

Perforation of Small Intestine

This acute abdominal condition may be caused by a variety of disease processes and by trauma. The characteristic history of the causative lesion, together with signs and symptoms of acute peritonitis from rupture of a hollow viscus and the presence of pneumoperitoneum, lead to the correct diagnosis. However, evidence of a perforated viscus is an absolute indication for early laparotomy by means of which the diagnosis can be confirmed and the lesion identified

LARGE INTESTINE

Acute Obstruction

Acute obstruction of the large bowel may be caused by a variety of lesions. The commonest cause is *carcinoma*, which may produce sudden complete obstruction to the passage of both stool and gas, although there are usually symptoms of previous partial obstruction. In general, large bowel obstruction requires a longer time to produce serious signs and symptoms than does small bowel ob-

struction, although the clinical picture is quite similar in the later stages. It is characterized by enormous abdominal distention, particularly if the lesion is situated in a distal segment, owing to the distensibility of the voluminous cecum and colon and to distention of the small gut when the ileocecal valve is incompetent. Gas- and fluid-filled colon may be outlined by palpation and percussion in many instances. *Closed-loop obstruction* is a special type in which the ileocecal valve remains competent and the distention is limited to the cecum and colon proximal to the lesion. In this type, early recognition and treatment are necessary to avoid the complication of extreme cecal distention resulting in ischemic necrosis of its wall and perforation. In such a case there may be few physical or roentgenographic signs of small bowel distention. Vomiting of a reflex nature may occur early, but true obstructive vomiting appears late and may be minimal or even absent despite great abdominal distention and circulatory collapse owing to the pooling of fluid and electrolytes in the distended intestine. Visible peristalsis, loud booming peristaltic sounds, and a succus splash on shaking the patient are often found.

Volvulus of the cecum or pelvic colon produces acute, severe symptoms and signs, and surgical treatment is urgently required. In the former, there is closed-loop obstruction of the cecum and part of the ascending colon with simple obstruction of the ileum. The large tympanic mass of dilated cecum may be outlined visually by palpation and by percussion as it extends upward toward the left upper quadrant of the abdomen. Reflex vomiting may be frequent and copious from the outset, followed later by the vomiting of feculent material due to ileal obstruction. Change of position may increase the pain, owing to stretching of the twisted mesentery, and it may radiate to the back

In *volvulus of the pelvic colon* the distended, closed loop of colon rises out of the left side of the pelvis and projects toward the right upper quadrant. Here, too, the closed loop obstruction is associated with simple obstruction of the proximal colon

In *volvulus of the cecum or pelvic colon* early strangulation leading to gangrene and

perforation may occur. As strangulation develops, signs of an acute intraperitoneal inflammatory process appear, including those of paralytic ileus, peritonitis, leukocytosis, and rising pulse rate and temperature. In all cases of large bowel obstruction x-ray examination is diagnostic, particularly when a barium enema is introduced. The diagnosis of an obstructive lesion may be made by sigmoidoscopic examination when it is within reach of the instrument. Occasionally a rectal tube can be carefully inserted through a sigmoidoscope in volvulus of the pelvic colon, releasing the confined gas and fluid, leading to nonoperative reduction of the torsion.

Obstruction due to adhesions usually follows previous abdominal surgery or peritonitis, and its exact nature is determined at laparotomy.

Inflammatory stenosis of the large bowel may be caused by a variety of lesions, prominent among which is acute diverticulitis of the pelvic colon. Another example is postradiation stenosis of the pelvic colon or rectum following treatment of carcinoma of the uterine cervix.

Paralytic ileus causing obstruction of the colon is just part of the condition involving both large and small intestine when peritonitis is present (see Chapter 24, Intestinal Obstruction).

Fecal impaction is an unusual cause of acute large bowel obstruction but can be diagnosed by the history and rectal examination.

The large bowel (principally the cecum and pelvic colon) may become obstructed when a segment is caught in an internal or external hernia (incarcerated hernia).

Intussusception of the colon can be diagnosed by symptoms and signs of large bowel obstruction, palpation of an elongated mass in the course of the colon, the passage of bloody material per rectum, and barium enema x-ray examination.

Foreign bodies occasionally cause obstruction if they become impacted in a stenotic segment of colon, at the normally narrow pelvicorectal junction, or at the upper end of the anal canal. The diagnosis of the latter is confirmed by rectal examination and of the former by roentgenographic examination.

Perforation of Large Intestine

Perforation may be traumatic, inflammatory, or neoplastic. *Traumatic perforation* is caused by perforating or penetrating wounds, foreign bodies introduced per rectum, or blunt trauma. *Inflammatory perforation* may result from diverticulitis, obstruction with gangrene, or ulcerative colitis. *Neoplastic perforation* of the colon occasionally occurs. The history and physical signs of a perforated hollow viscus with rapidly developing, severe septic peritonitis and pneumoperitoneum are characteristic. Determination of the site of perforation may be made from the early signs, but, later, generalized peritonitis may make this impossible before abdominal exploration is performed. It must be differentiated from *perforated peptic ulcer* and *perforated appendicitis*.

Diverticulitis of the Colon

Diverticulitis may produce either obstructive or inflammatory symptoms. When obstruction is present, the clinical picture resembles that of obstructive carcinoma. When local inflammation or abscess develops, the symptoms resemble those of pelvic appendicitis, and certain diagnosis before operation may not be possible because a barium enema in these circumstances is inadvisable. A history of previous bowel symptoms, with attacks of diarrhea or constipation or blood and mucus in the stool, may be suggestive. The pain of *appendicitis* usually begins in the epigastrium, that of pelvic diverticulitis in the hypogastrium.

APPENDIX

Acute Appendicitis

It is generally recognized that every patient with acute appendicitis should be operated upon at the earliest possible moment, in order to reduce the mortality and morbidity of this progressive disease. The commonest reasons for delay are procrastination on the part of the patient or his medical adviser. Experience has shown that it is impossible to predict the severity of an attack at its onset. The pathologic changes present depend upon so many different local circumstances that there may be little correlation between them and the signs and symptoms produced.

The position of the appendix varies greatly from case to case, both in the direction it extends from its cecal attachment and in the position of the cecum itself (see Chapter 25, Appendix). Before considering the diagnosis of appendicitis in a patient with an abdominal scar, it is well to make certain that the organ is present. Conversely, a patient may mistakenly believe that the appendix has been removed during a previous operation (e.g., cholecystectomy). If the diagnosis seems clear it is better to operate than to depend on the patient's declaration that the organ has been removed.

The history of the onset of symptoms should be carefully considered as well as the remote history (frequently there have been previous attacks of pain in the appendiceal region). Preceding subacute attacks have often been looked upon by the patient as due to acute indigestion.

Common symptoms and signs include pain, vomiting, low-grade fever, local abdominal tenderness, and preceding constipation. Inconstant findings are rigidity of muscles, hyperesthesia, slight distention, and testicular symptoms in the male.

Early pain is usually experienced in the epigastrium or generally in the mid-abdomen, and only later does it become localized over the appendiceal area, which is usually in the right lower quadrant. This location sequence may vary considerably. The local pain may be due to appendiceal colic or to distention of the appendix by products of inflammation. The pain may be steady or there may be crampy exacerbations. Vomiting, as a rule, occurs early in this condition but may never occur, and in the latter case nausea or anorexia is just as significant. Local abdominal tenderness over the appendix is found early in the attack and is elicited best by deep palpation. This is found, classically, at McBurney's point which is situated at the junction of the lateral and middle thirds of a line joining the umbilicus and the right anterior superior iliac spine, and corresponding closely to the actual site of the appendix. It arises either from the appendix itself or from adjacent irritated peritoneum. In a child, or in an adult with the appendix in the pelvis, rectal palpation will produce pain in the region of the inflamed appendix.

The *shake test* is frequently positive, eliciting pain over the inflamed appendix. Hyperesthesia of the skin of the abdominal wall on the right side can often be found in the distribution of the cutaneous branches of the 10th and 11th thoracic nerves. It is most commonly found just to the right of and below the umbilicus (T10).

In acute appendicitis there is almost always local muscular rigidity, which varies greatly in intensity, although this sign may not be present early in the disease. If very severe, it is suggestive of early local peritonitis. Muscular

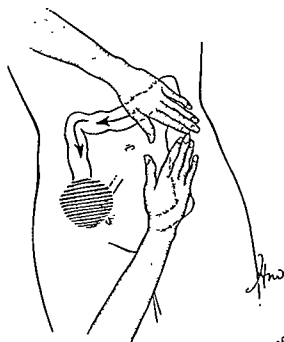


Fig. 467—Rovsing's sign. Compression of the descending colon forces gas into inflamed cecum, causing right lower quadrant pain.

rigidity may be absent, particularly if the appendix is pelvic or retrocecal in position. Psoas spasm is frequently found. In pelvic appendicitis the *obturator test* may be positive, and in high positions of the inflamed appendix, rigidity of the quadratus lumborum is common. *Rovsing's sign*, when present, is very suggestive.

Fever usually appears before the first day has passed and is low-grade. Occasionally fever may not appear, or it may be very high, particularly in young children. Chills are infrequent but may occur early in the disease. Pre-

APPENDIX

ceding constipation is the rule, but the bowel movements may have been regular or diarrhea may have occurred. The pulse rate is normal initially but accelerates with the development of peritoneal irritation or frank peritonitis. Cecal gurgling on palpation indicates a form of distention owing to atony, possibly a local localized paralytic ileus. Males occasionally have testicular pain accompanied by retraction of the right testicle, probably because both the testicle and the appendix are supplied by the 10th thoracic spinal segment.

The time sequence of symptoms and signs is of considerable importance in diagnosis. The usual sequence is as follows: pain, nausea or vomiting, local tenderness, fever, and leukocytosis. Leukocytosis is usually present but may be minimal or absent, particularly in elderly patients. It is frequently of great value to determine the position of the cecal gas shadow by means of x-ray films of the abdomen.

The preceding description applies to acute appendicitis, before perforation has occurred, but after perforation the picture is greatly altered. Since this is an advanced stage of the disease, the signs and symptoms are those described above plus those of either local or diffuse peritonitis. After perforation the pain increases and may spread widely throughout the abdomen, accompanied by increased, or recurrence of, vomiting. The signs of paralytic ileus soon appear if diffuse peritonitis ensues. When the peritonitis is localized, limited by omentum or bowel, a mass develops and is referred to as an *appendiceal abscess*. Such localized peritonitis varies in site according to the position of the cecum and appendix and may be iliac, retrocecal, paracecal, medial to the cecum, behind the terminal ileum, pelvic, or elsewhere in the abdomen. The physical findings will vary with its position and the contiguous structures irritated (e.g., retrocecal appendix causing psoas rigidity, medially situated appendix causing ureteral irritation and ated appendix causing ureteral irritation and dysuria, paracecal appendix causing loin tenderness and quadratus lumborum rigidity). Characteristically, when there is a pelvic appendiceal abscess there are bladder and rectal irritations, and the tender bulging mass is palpable per rectum. Later on, in the presence of an appendiceal abscess, especially if it is lo-

cated in the pelvis, the small or large intestine may become secondarily obstructed.

Acute appendicitis is so very common that familiarity breeds contempt and the diagnosis is often made after a very perfunctory examination. This is bound to lead to errors in diagnosis and is, therefore, inexcusable.

Appendicitis must be distinguished from several other conditions. Extra-abdominal conditions include diaphragmatic pleurisy, spinal disease, and influenza. Early *basal pneumonia* with pleurisy may produce pain, tenderness, and rigidity in the right lower quadrant. The rigidity may give way on deep palpation, and Rovsing's sign is never present. Elevation of the respiratory rate and pulse rate may be marked. Careful chest examination will make the diagnosis clear, and x-ray examination may confirm it. Abdominal pain may be prominent in influenza, but there will be signs of an upper respiratory infection and generalized muscular aches and pains. Vomiting, if it occurs, often precedes the abdominal pain. *Spinal disease*, in the form of acute osteomyelitis, may simulate appendicitis but is recognized by careful examination of the spinal column and the absence of deep abdominal tenderness.

Typhoid fever, although uncommon nowadays, may produce symptoms similar to those of appendicitis. The general malaise, splenomegaly, roseola, leukopenia, bradycardia, and history of onset serve to differentiate the two conditions.

Early appendicitis may be mistaken for *indigestion*, *intestinal colic*, or *acute gastritis*, but careful consideration of the history of onset and findings will permit differentiation.

Acute appendicitis, because its signs and symptoms are variable, may be simulated by almost any acute abdominal condition, but those that enter into the differential diagnosis vary with the position of the appendix and with the presence or absence of appendiceal perforation.

Since the duodenum, right kidney, gall bladder, and liver are near an inflamed high-lying appendix, acute conditions involving these organs must be distinguished from it. Inflammation about a *penetrating duodenal ulcer* is differentiated by the characteristic preceding history of pain, coming on 2-3 hours

after a meal, which is promptly relieved by ingestion of food or alkali.

Right-sided renal or ureteral colic, due to stone, may resemble acute appendicitis. The history and nature of the symptoms, severe colic with intervening remissions, radiation of pain into the external genitalia, hematuria, and x-ray evidence of a stone serve to identify this condition. If doubt remains, cystoscopy with retrograde pyelography will be diagnostic. *Acute right-sided hydronephrosis* produces a swelling, palpable in the side of the abdomen and loin, which is tense, tender, and rounded and may be mistaken for an appendiceal abscess. However, such a mass is movable and will be associated with urinary symptoms. Abdominal rigidity will not be persistent. *Acute pyelitis* on the right side is frequently confused with appendicitis but can be identified by frequent rigors early in the disease, high fluctuating fever, dysuria, urinary frequency, pyuria, and bacilluria. The abdominal muscles are not rigid.

Acute cholecystitis may be indistinguishable from appendicitis, especially when the elongated and inflamed gall bladder descends into the right iliac fossa. If muscular rigidity is not too great and the abdominal wall thin, the diagnosis may be confirmed by palpation of the tender gall bladder which moves with the liver during respiration. Pain may be referred to the right subscapular region (and shoulder), and there is never resonance over an enlarged gall bladder because it lies anterior to the colon. If gall bladder colic is experienced simultaneously and a past history of gall bladder symptoms is obtained, cholecystitis may be identified with certainty. Noticeable jaundice and bile in the urine are additional diagnostic features.

Strangulation of the omentum or of an appendix epiploica by acute torsion may simulate appendicitis by the production of pain and tenderness to the right of the umbilicus and differential diagnosis may not be possible.

Perinephric abscess of metastatic origin may resemble an abscess due to appendiceal perforation. The history of onset without the typical features of the development of appendicitis serves to differentiate it.

When the appendix is in its common iliac position, several other conditions must be seriously considered in the differential diagnosis.

Carcinoma of the cecum or of the right half of the colon, which has become ulcerated and has perforated the wall, producing an abscess, may simulate appendiceal abscess. The differentiation may be impossible but the history may suggest the true diagnosis. *Ileocecal tuberculosis* and *acute regional ileitis* may also be indistinguishable prior to operation. Another condition which causes similar symptoms is *acute mesenteric lymphadenitis*, but in this case differentiation is only academic, as appendectomy is the treatment of choice for both conditions.

Perforated duodenal ulcer with slow leak of bowel contents down along the ascending colon into the iliac fossa and pelvis may resemble appendicitis. However, the onset is sudden, the initial shock often severe, and pneumoperitoneum definitely distinguishes it.

Rupture of the right rectus muscle or development of an abdominal wall abscess can be differentiated by the history and local findings.

When the appendix is situated in the pelvis, the differential diagnosis of appendicitis in the female differs from that in the male. In both sexes *diverticulitis* of the colon, *bowel obstruction*, and *ureteral stone* often present problems of differential diagnosis. In *obstruction of the small bowel* characteristic distention and peristaltic sounds are present, as well as the other findings, so that it may be clearly differentiated from appendicitis. *Large bowel obstruction* in the pelvis may be confused with pelvic appendicitis but can be differentiated by the history, findings, and the presence of early distention which is uncommon in appendicitis. *Diverticulitis of the pelvic colon* may cause obstructive or inflammatory symptoms. When an abscess develops, the findings closely resemble those of pelvic appendicitis, but the signs and symptoms are predominantly left-sided. Differentiation may be impossible prior to operation unless there have been proved previous attacks of diverticulitis.

In the female, acute conditions involving the pelvic generative organs must be differentiated from acute pelvic appendicitis (see Chapter 28, Female Genital Tract). *Threatened abortion* can be identified by the history of amenorrhea, uterine bleeding, nature of the pain, and absence of local signs of appendicitis. In *ectopic gestation* (with hemorrhage) anemia, a

displaced uterus and the absence of the symp-

it The former rarely causes epigastric or generalized abdominal pain but usually causes bilateral hypogastric pain from the beginning. The presence of vaginal discharge is helpful, as is the pelvic examination. The true diagnosis may not be reached prior to operation, but laparotomy is less dangerous than delay in treatment of acute appendicitis.

Other gynecologic conditions easily confused with appendicitis are *ovarian cyst*, *hydrosalpinx*, and *leiomyoma with a twisted pedicle*. In such an instance pain and vomiting occur almost simultaneously, and nausea is very severe and persistent. A movable rounded pelvic mass may be demonstrable by vaginoabdominal palpation.

Once diffuse or general peritonitis, caused by appendicitis with perforation, has become established, specific diagnosis may be impossible. The diagnosis of peritonitis will be relatively simple, but the possible causes will include *primary peritonitis* (pneumococcal, streptococcal), *secondary peritonitis* of other origin, *mesenteric vascular occlusion*, *advanced acute intestinal obstruction*, *severe pyelophlebitis*, and *acute pancreatitis*. Primary peritonitis can be identified by pure culture of the offending organism from the intraperitoneal fluid and pancreatitis by the elevation of the serum amylase. Identification of other causes may be possible on the basis of the history of the illness.

GALL BLADDER

Acute Cholecystitis

This is an acute inflammation of the gall bladder which may occur in the presence or absence of gallstones. It varies greatly in intensity from mild to very severe with gangrene of the wall. The chief symptoms are *pain*, *vomiting*, and *fever*. The *pain* varies considerably, especially when a stone causes simultaneous gall bladder colic. It is aching in nature and accompanied by a sensation of swelling in the right subcostal region. When there is associated pericholecystitis, the pain may be diffuse in the right upper quadrant and referred to the subscapular region or to the

right shoulder. When the liver or gall bladder is greatly enlarged, the pain may extend into the right lower quadrant. *Vomiting* is variable, but in the presence of gallstones and peritonitis, nausea and vomiting may occur repeatedly. Characteristically, the *fever* is not high, rarely exceeding 103° F., but it may be irregular with accompanying chills. The *pulse* rate is often normal in cholecystitis, but an increase indicates absorption of toxic materials from the biliary tract or advancing peritonitis. *Tenderness* over the site of the gall bladder is always present, and the organ can be felt if it is enlarged and if muscular rigidity is not pronounced. An inflammatory mass occasionally develops through involvement of contiguous organs. Pericholecystitis, or localized peritonitis, produces muscular rigidity over the inflamed area. *Constipation* is frequently present, probably because of the inflammation affecting adjacent coils of bowel. *Jaundice* may accompany the attack and is caused by a gallstone in the common bile duct or by inflammatory compression of the common hepatic or bile duct producing obstruction.

Acute cholecystitis must be distinguished from *appendicitis*, *penetrating duodenal ulcer*, *biliary colic*, *pleurisy*, *pancreatitis*, and *hepatitis*. The differentiation from *appendicitis* has been discussed in the preceding section.

A *penetrating duodenal ulcer* with localized peritonitis may produce physical signs similar to those of cholecystitis, but a past history of pain coming on 2-3 hours after a meal, relieved by ingestion of food or alkali, will make the diagnosis clear. Other helpful evidence includes the occurrence of *melena*, symptoms suggestive of *pyloric spasm*, and *heartburn*. An immediate barium meal may demonstrate the presence of the ulcer.

Biliary colic may be distinguished from cholecystitis by its acute onset and severe colicky pain. There is no abdominal wall rigidity and the patient frequently rolls about the bed in agony, whereas he tends to lie still to avoid increasing the pain of cholecystitis. The temperature is normal or subnormal, and collapse may be severe. Administration of atropine often relieves the pain promptly.

Diaphragmatic pleurisy accompanying pneumonia or pulmonary infarction produces high fever and severe chills. The abdominal tender-

ness, if present, is superficial and signs of infiltration of the base of the lung may be found. Expectoration of sputum or hemoptysis will also be suggestive of such a condition.

Acute pancreatitis may resemble cholecystitis closely or coincide with it. However, the pain of pancreatitis is in the epigastrium or to the left of the midline and usually penetrates to the back. Tenderness is found in the epigastrium and left hypochondrium and paralytic ileus frequently develops early in the disease. Collapse is often profound. The serum amylase will be significantly elevated.

Acute hepatitis is characterized by tenderness over the entire liver, including its lateral aspect. Jaundice may be evident and the urine may contain bile. The liver is often moderately enlarged and the gall bladder not palpable. Prodromal symptoms of anorexia, nausea, and vomiting for several days are typical.

Biliary Colic

This has been discussed in the differential diagnosis of acute cholecystitis. It is easily differentiated from *right renal colic* by the history and local findings, by jaundice if it is present and by examination of the urine and x-ray films.

PANCREAS

Acute Pancreatitis

This was formerly regarded as being relatively rare but it has been recognized more frequently in recent years. It is an acute abdominal condition which is often misdiagnosed because the possibility is not considered. It is particularly important to diagnose acute pancreatitis accurately since experience has shown that conservative treatment alone is indicated and operative measures should be restricted to the treatment of certain complications, such as an abscess. Acute pancreatitis is uncommon before the age of 40, and the victim is frequently obese. It often begins after the ingestion of a heavy meal, and it may or may not be associated with gall bladder disease.

The symptoms of pancreatitis vary widely in severity and variety. Pain is a constant symptom and the onset is usually very sudden. At times it is severe enough to cause collapse and

syncope. The pain is agonizing, often causing the patient to cry out. It is situated primarily in the epigastrium but may reach one or both loins. It usually radiates to the back and subscapular area on the left side. Sometimes there is pain in the left side of neck or left shoulder, referred from the irritated diaphragm. The initial intensity gradually diminishes, but the pain may be felt over the entire abdomen. Signs and symptoms of *hypotensive shock* appear unless the attack is mild. Nausea and vomiting are often constant and persistent features. The vomiting does not become feculent unless paralytic ileus supervenes. Dehydration is early and severe. *Epigastric tenderness* is always present and often severe, but the pelvic peritoneum is not tender. *Epigastric rigidity* is usually present, but the abdominal wall may be relaxed. The edematous pancreas is rarely palpable. In about one half of the cases *slight jaundice* is present, due to obstruction of the bile duct by the edematous pancreas or a gallstone. There may be *bluish discoloration* in the flanks and umbilical region, owing to extravasated blood and pancreatic enzymes, constituting a grave prognostic sign which occurs late in the acute hemorrhagic form. *Glycosuria* and *hyperglycemia* may be present because of interference with function of the islets of Langerhans. The serum amylase level is markedly raised and may remain elevated for a few days or fall sharply after 24-48 hours to a subnormal level. The serum lipase is usually elevated early in the disease also and remains so for several days in most instances. Depression of the serum calcium level, owing to a major degree of pancreatic destruction and fat necrosis, indicates a serious prognosis. *Leukocytosis* is usually moderate (15,000-20,000/c mm). X-ray examination of the abdomen frequently shows isolated, distended loops of small bowel (sentinel loop) containing fluid levels in the upper midzone of the abdomen. Widespread *paralytic ileus* often develops later.

Acute pancreatitis must be differentiated from *cholecystitis*, *perforated ulcer*, *intestinal obstruction*, *appendicitis*, *mesenteric vascular occlusion*, *acute gastritis*, and *coronary occlusion*. The details of the differentiation are described elsewhere in this chapter. A careful consideration of the history and findings, sup-

ported by a significantly elevated serum amylase, makes the diagnosis of acute pancreatitis certain.

LIVER

Liver Abscess

Acute liver abscess may be *amebic* or *pyogenic*. *Acute amebic abscess* is a pyrexial illness with enlargement and tenderness of the liver, and there is a history of dysentery and amebae in the stools in approximately one half of the cases. The manifestations include intermittent fever, night sweats, weakness, loss of weight, nausea, anorexia, malaise, and vomiting. Chills and mild jaundice may occur. There are right upper quadrant pain and tenderness, and limited chest movements on the right side. Pain is often referred to the right shoulder, scapular region, and iliac fossa. Local edema of the thoracic and abdominal walls is occasionally apparent. Pain is caused by sudden movements so that the patient holds the right arm close to his side. There may be congestion of the lower part of the right lung, and the right side of the diaphragm may be immobile. There is leukocytosis of moderate degree but no alteration of the differential count. X-ray films may show a soft tissue shadow of the abscess and a raised, fixed hemidiaphragm. Examination of the stool often reveals evidence of amebiasis. The diagnosis is confirmed in the operating room when pus is aspirated from the abscess. The symptoms and signs of *pyogenic abscess* are similar to those of amebic abscess, but exploratory aspiration should not be attempted because of the danger of disseminating infection in the pleural or peritoneal cavity. If a liver abscess ruptures into the peritoneal cavity acute generalized peritonitis results.

Liver abscess must be differentiated from *subphrenic abscess*, *cholecystitis*, and *perinephric abscess*. *Right perinephric* and *subphrenic abscess* may originate from a perforated retrocecal appendix or may be of metastatic origin. There is pain on pressure forward in the costovertebral angle. There are general symptoms of an acute suppurative process, but the liver will not be tender anteriorly, nor will there be nausea and vomiting.

The history of appendicitis or the presence of a focus of suppuration elsewhere may be helpful in making the diagnosis.

Rupture of the Liver

This serious condition will usually be suspected on consideration of the history of the injury. Examination may reveal abrasions or bruising over the right side of the abdomen or thorax. There are tenderness and rigidity of the overlying abdominal muscles and signs of shock due to hemorrhage. If internal hemorrhage is massive, the patient will be pale, and movable dullness will be found in the flanks. Early recognition and prompt treatment constitute the only hope of saving the life of the patient.

Rupture of the liver may be associated with damage to other viscera, so that differential diagnosis is of no importance once it is decided that serious visceral damage has been sustained and immediate laparotomy is necessary.

Acute Hepatitis

This may produce symptoms and signs of an acute abdominal condition, especially in young individuals. It is characterized by *tenderness* over the whole liver, including its lateral aspect. There is usually a history of *anorexia*, with or without nausea and vomiting, over a period of days before acute symptoms develop. The *temperature* may be normal or slightly elevated, and leukocytosis is *moderate*. Often there is no abdominal wall rigidity, but there may be muscle guarding on palpation over the tender, enlarged liver. Often there is *jaundice* and *bile in the urine*. Acute hepatitis must be distinguished from *cholecystitis*, which does not produce tenderness over the whole liver.

SPLEEN

Rupture of the Spleen

Spontaneous rupture of a diseased spleen occasionally occurs, but usually there is a history of trauma. The injury may have been *minor*, or there may have been a serious injury sustained in the region of the left upper abdomen or chest. The symptoms are those of rapid and profound collapse due to internal hemorrhage. If treatment is not immediately

available death will ensue; in fact death from hemorrhage may occur within a few minutes, before anything can be done. The chief diagnostic features are *local pain* in the left upper quadrant, *free blood in the abdomen* (pain, ileus, distention, rigidity, movable dullness) and *hemorrhagic shock*. Other characteristic signs are *left shoulder pain*, *tenderness in the left flank*, *muscular guarding* in the left flank, a *mass* in the splenic region, *low-grade fever*, *pallor*, and *slight jaundice*. The pain is continuous. If hemorrhage is massive, movable dullness may confirm the diagnosis. Other viscera may be seriously damaged by an injury causing splenic rupture. The differential diagnosis of rupture of the spleen is of little importance. If it is suspected, immediate laparotomy is urgently indicated.

Splenic Infarct

Embolism of the splenic artery or one of its branches produces a splenic infarct. This usually occurs in an enlarged abnormal spleen. It causes sudden severe pain in the splenic region and sudden increase in the size of the spleen. A friction rub may be heard over the spleen, owing to the formation of an inflammatory exudate on its serosal surface. Evidence of embolism elsewhere and of auricular fibrillation is helpful in the diagnosis. It can be differentiated from splenic rupture by noting the absence of a history of trauma and hemorrhagic shock. *Delayed splenic rupture* (intra-splenic hematoma) may be difficult to differentiate if a source of emboli cannot be found.

PERITONEUM

Acute Peritonitis

Acute peritonitis is a very common accompaniment of many acute abdominal conditions. Streptococcal and pneumococcal peritonitis are hematogenous and are designated *primary peritonitis*. Bacteria may reach the peritoneal cavity by rupture of a viscus, by passing through the wall of a diseased viscus, or through a penetrating abdominal wound. Abscesses may burst and infect the whole peritoneal cavity. The common causes of widespread peritonitis are discussed elsewhere in this chapter and include perforation of (a) appendix, (b) peptic

ulcer, (c) small intestine, (d) colon and (e) biliary tract, and infection from (a) gangrenous bowel, (b) salpingitis, (c) upper urinary tract infection, and (d) rupture of an hepatic abscess.

The symptoms vary according to the region and extent of peritoneum involved, the nature of the infective agent, and the rapidity of the onset. As described in connection with peritonitis complicating other acute abdominal conditions, there are the following symptoms and signs: abdominal pain, nausea, vomiting, abdominal rigidity, distention, paralytic ileus, toxemia, hyperesthesia, fever, tachycardia, and collapse when there is extensive involvement.

Acute peritonitis must be differentiated from the *colic*, *intestinal obstruction*, *internal hemorrhage*, and *pleurisy*. The differentiation is discussed elsewhere in this chapter.

FEMALE GENERATIVE ORGANS

Ruptured Graafian Follicle

If hemorrhage from a ruptured graafian follicle is severe, the findings may resemble those of ruptured ectopic pregnancy or pelvic appendicitis. It typically occurs midway between the menstrual periods.

Ruptured Ectopic Pregnancy

Ectopic gestation commonly ruptures, leading to slow or rapid, massive intraperitoneal hemorrhage. The history will reveal the presence of *amenorrhea* for 1-2 months, perhaps *morning sickness* and *uterine bleeding*, and, rarely, the *passage of a membrane per vaginam*. In addition to amenorrhea, other signs of early pregnancy, such as *engorgement of the breasts*, are present. There is *abdominal pain*, chiefly in the hypogastrium and lower quadrants of the abdomen, together with *tenderness*. Before rupture occurs, bimanual pelvic examination may reveal the presence of a *tender, movable mass* beside the uterus, as well as some *uterine enlargement* and *softening of the cervix*. The diagnosis should be suspected in every case of lower abdominal pain in a woman of child-bearing age. Before rupture occurs the condition must be distinguished from *appendicitis*, *threatened uterine abortion*, *salpingitis*, and *ovarian or broad ligament cyst*.

When rupture of an ectopic pregnancy takes place, there is a sudden lower abdominal pain, immediate vomiting, signs of severe hemorrhagic shock, a tender swollen abdomen, and free fluid in the peritoneal cavity. The pouch of Douglas is tender on palpation, the cervix may feel soft, and the uterus is enlarged.

This condition must be differentiated from perforated peptic ulcer or gall bladder, intestinal obstruction, pancreatitis, perforated appendix, torsion of an ovarian cyst, and severe hemorrhage from a graafian follicle. The history and signs indicative of massive hemorrhage, together with other features mentioned above, make the diagnosis of ruptured ectopic pregnancy certain.

The hemorrhage is occasionally small and repeated, leading to the formation of a pelvic hematocoele, with no period of hemorrhagic shock. The pelvic hematocoele is evidenced by fullness of the hypogastrium, it is palpable by manual pelvic examination, and pressure on it causes pain. It may cause urinary retention or frequency.

Twisted Ovarian Cyst

Torsion of the pedicle of an ovarian cyst produces acute abdominal symptoms. The principal features are lower abdominal pain, vomiting, and a tender swelling in the lower abdomen. The onset of the vomiting coincides with that of the pain. There is a rounded, tender mass palpable in the pelvis. Signs of peritonitis appear as time passes and rigidity may then prevent palpation of the mass through the abdominal wall.

This condition must be distinguished from pelvic appendicitis and salpingitis, especially in its early stages, and from pelvic peritonitis of any origin in its later stages. The onset, however, is very abrupt and characteristic. The true nature of the condition may not be determined before exploration of the abdomen is carried out.

Pelvic Inflammatory Disease

This term embraces all forms of infective lesions of the female pelvic organs, but acute salpingitis is by far the most important and common one. There is a history of recent gonorrheal infection, abortion, curettage, or delivery. The signs and symptoms of acute

pelvic peritonitis are present, including lower abdominal pain, nausea, vomiting, and moderately high fever. Tenderness is present over the lower abdomen, and there are varying degrees of rigidity and distention. Purulent vaginal discharge is usually present and vaginal examination reveals bilateral tenderness in the pelvis. Smears of the discharge will contain gonococci if the cause is gonorrhea.

Appendicitis may be confused with acute salpingitis but may be differentiated by the characteristic time sequence of symptoms of appendicitis combined with restriction of pain to the right side. The patient with peritonitis due to appendicitis characteristically appears more seriously ill than the patient with peritonitis due to acute salpingitis of gonorrheal origin. At times salpingitis cannot be distinguished from appendicitis before operation.

Ruptured Ovarian Cyst

The symptoms and differential diagnosis are similar to those of ruptured graafian follicle, but the former may be more severe. Disappearance of a previously palpable ovarian swelling is helpful in the diagnosis.

Retroplacental Hemorrhage

This may occur during the third trimester of pregnancy and cause considerable abdominal distress. On examination, the uterus is greatly distended and tense, pain and tenderness are acute, and shock occurs. An associated toxemia of pregnancy or a history of local trauma may be present. The fetal heart sounds are absent. The history and findings make the diagnosis certain.

Rupture of Pregnant Uterus

This usually occurs near term or during labor. Spontaneous rupture through a cesarean section scar is common. Obstruction of labor may lead to rupture of the lower uterine segment if not promptly treated. Rupture may be caused by external violence or by obstetric maneuvers. If the patient is in labor, uterine contractions cease and abdominal pain and tenderness rapidly develop. Hemorrhagic shock appears, the fetal parts are palpable within the abdomen but outside the uterine wall, and the fetal heart sounds usually stop.

Abdominal Pregnancy at Term

An extrauterine pregnancy occasionally survives well into the third trimester or until term, and abdominal pain and discomfort become very distressing. The diagnosis may be made by palpation of the uterus separate from the bulk of the fetus and its placenta and membranes and by inability to perform successful version to improve the position and lie of the fetus. The diagnosis becomes certain when cesarean section is performed.

GENITOURINARY SYSTEM

Calculus

Small calculi produce attacks of acute renal or ureteral colic as they pass down toward the bladder. The pain begins in the lumbar region on the affected side, is sudden in onset, is colicky in nature, and passes down along the course of the ureter. It may radiate into the external genitalia or medial side of the corresponding thigh. The pain may be severe enough to cause collapse. There is rigidity and tenderness of the abdominal muscles over the course of the affected ureter. The urine may contain only scattered red blood cells or may be grossly smoky or bloody in appearance. An x-ray film usually reveals a shadow of the stone or stones since 95% of calculi are radiopaque. Intravenous pyelography or retrograde pyelography will confirm the diagnosis in doubtful cases. The typical history and findings serve to differentiate renal colic from *biliary colic*, *cholecystitis*, and *acute appendicitis*.

Perinephric Abscess

This is nearly always caused by rupture of a cortical abscess of the kidney which, in turn, is often metastatic from a distant abscess, e.g., furuncle. The symptoms include *pain* over the renal region, *fullness* in the lumbar region, *fever*, *chills*, and often *prostration*. A large abscess is palpable as a tender mass in the loin and downward along the psoas muscle. When well developed the mass is fluctuant. *Roentgenographic examination is diagnostic*: the lumbar spine is curved away from the affected side, the psoas muscle shadow is obliterated, a soft tissue shadow may be present, and the

diaphragm may be immobile. In addition, an intravenous pyelogram may show fixation of the kidney, or displacement of the kidney anteriorly, with distortion of a calyx.

The diagnosis presents no problem when the possibility is entertained, but perinephric abscess may require differentiation from *hepatic abscess* or *subphrenic abscess*. The history, physical findings, and x-ray signs clarify the diagnosis.

Rupture of Kidney or Bladder

Rupture of the kidney is caused by external violence, crushing injury, or penetrating wounds. If death from massive hemorrhage does not ensue promptly, the diagnosis is made from the history of injury, a degree of hemorrhagic shock, hematuria (gross or microscopic), local tenderness and rigidity, and, at times, a palpable mass. Renal rupture may be associated with injuries of other viscera, such as the liver or spleen, but such hematuria is pathognomonic of renal injury irrespective of other lesions. An intravenous pyelogram is diagnostic.

Rupture of the bladder is usually due to external violence when the bladder is distended and is often caused by spicules of bone in cases of fractured pelvis. A history of a crushing injury, a fall, or a blow in the region of the bladder should suggest the possibility of ruptured bladder to the examiner. The test may be intraperitoneal or extraperitoneal, but both produce an acute abdominal condition. Blood and urine escape into the peritoneal cavity or retroperitoneal tissues, causing acute generalized peritonitis in the former and extraperitoneal cellulitis in the latter. Lower abdominal pain, chills, fever, and local tenderness appear promptly, but signs of peritonitis, in intraperitoneal rupture, may be masked for a time by severe shock produced by the injury, e.g., crushed pelvis.

The diagnosis can be made by a consideration of the history, state of shock, and local symptoms and signs. The patient is unable to void properly, there is escape of some bloody urine from the urethra, and an intravenous pyelogram or a cystogram will show extravasation of the dye. Associated visceral injuries must not be overlooked, e.g., rupture of rectum.

Torsion of Ectopic Testis

Torsion of an incompletely descended testis causes extreme sickening pain, usually in the inguinal region. Vomiting and shock may be severe. Absence of the testis from the scrotum on the affected side and a tender mass in the inguinal region confirm the diagnosis.

Acute Pyelitis

This is characterized by pain and severe tenderness over the kidney, high, spiking fever, severe chills, marked leukocytosis, dysuria, frequency, and pyuria. There is usually general malaise and, occasionally, nausea and vomiting. Abdominal pain and associated paralytic ileus, with or without a diffuse mass in the corresponding upper quadrant of the abdomen, may be misleading features, but a careful consideration of the history, physical findings, and urinalysis will clarify the diagnosis.

Urinary Retention

Retention of urine in the urinary bladder causes acute lower abdominal pain, and the distended bladder becomes evident as a rounded, tender suprapubic mass. The history, the mass, and the relief afforded by catheterization confirm the diagnosis.

VASCULAR SYSTEM

Mesenteric Vascular Occlusion

Mesenteric arterial embolism or *venous thrombosis* is an acute abdominal condition with a grave prognosis, but early diagnosis and treatment may permit a successful embolectomy in the former or lifesaving bowel resection in the latter. The symptoms and signs may be indistinguishable from those of strangulation of bowel with paralytic ileus. In both instances serious changes in the bowel wall quickly develop. However, in mesenteric vascular occlusion, *distention* appears very early. The affected gut occasionally forms a *palpable mass*. A *history* of endocarditis or the presence of auricular fibrillation may suggest the likelihood of embolism. Previous thrombotic episodes favor *mesenteric thrombosis*. An enema may demonstrate *bloody bowel contents* which have been *carried out*

of the lower end of the affected segment of intestine. *Peritonitis* gradually develops and is generalized.

It may be impossible to differentiate this condition in its later stages from *advanced peritonitis* of other origin or from mechanical bowel obstruction in its later stages.

Arterial Occlusion

Occlusion of the aorta or common iliac artery by embolism or thrombosis may cause acute abdominal symptoms, but the diagnosis is rarely in doubt. There is a sudden onset of pain in the lower limb, or limbs, whose arterial supply is suddenly diminished, and loss of the femoral pulse. Acute lower extremity pain, with numbness and paresis, develops rapidly, the severity depending upon the degree of ischemia present, along with blanching and then mottled bluish discoloration of the skin.

Dissecting Aortic Aneurysm

This serious condition produces excruciating abdominal pain, profound shock, abdominal distention, and some abdominal rigidity. The collapse persists, as does the severe abdominal pain, and the femoral pulse may be absent on one or both sides. Back pain is agonizing and persistent.

At its onset, dissecting aortic aneurysm may be confused with *perforated ulcer* or *acute hemorrhagic pancreatitis*. The history, persistent collapse, loss of femoral pulse, and absence of pneumoperitoneum differentiate it from perforated ulcer, and the normal or only slightly elevated serum amylase will aid in the differentiation from acute pancreatitis.

Dissecting aortic aneurysm frequently involves the renal arteries, causing infarction of both kidneys, hematuria, and symptoms of renal colic. Anuria is produced when the renal circulation is completely obstructed.

Ruptured Abdominal Aneurysm

Rupture of an aneurysm of the abdominal aorta, the splenic artery, or some other abdominal artery may rarely occur. Such a rupture is usually quickly fatal, but if the hemorrhage is not too rapid there may be sufficient

time for diagnosis and treatment. The signs and symptoms are those of massive intra-abdominal hemorrhage and free fluid in the peritoneal cavity or an enlarging retroperitoneal mass (hematoma). A history of the presence of an aneurysm and x-ray films demonstrating the outline of the sac or displacement of organs are helpful in the diagnosis.

Pylephlebitis

This may follow a suppurative process anywhere within the abdomen and is characterized by septicemia with high, spiking fever and severe chills. As hepatic involvement develops, right upper quadrant pain, an enlarged tender liver, and jaundice make their appearance. If the splenic vein is involved, splenomegaly also appears. Examination reveals signs of lower right pulmonary irritation and a raised immobile right hemidiaphragm, which can be confirmed by x-ray examination.

Pylephlebitis must be distinguished from *acute nonsuppurative hepatitis* and *liver abscess*. This may be difficult, but the history is usually helpful and the symptoms of pylephlebitis are much more severe.

LYMPHATIC SYSTEM

Acute Mesenteric Lymphadenitis

This condition is common in children and young adults and is manifested by pain, tenderness, and rigidity in the right lower quadrant of the abdomen, usually associated with nausea and vomiting. Fever and leukocytosis are variable. Occasionally a mass, due to enlarged mesenteric lymph nodes, is palpable in the right lower abdomen over the root of the mesentery.

Acute mesenteric lymphadenitis cannot be differentiated from *acute appendicitis* in most cases. The differentiation is only academic because the treatment of the two conditions is the same.

MISCELLANEOUS CONDITIONS

Penetrating and Perforating Wounds

Such wounds of the abdomen may be diagnosed by the history and presence of the wound. Wounds of entry distant from the

abdomen may confuse the examiner if he does not take into account the possibility of penetration of the abdomen. Signs of hemorrhage, peritonitis, and pneumoperitoneum are all of importance, indicating the necessity of immediate treatment.

Indirect and Blunt Trauma

Blunt injury of the abdomen can be diagnosed from the history and by examination which reveals evidence of visceral damage. When doubt exists, early laparotomy is indicated.

Wound Dehiscence and Evisceration

Postoperative abdominal wound disruption with partial evisceration is easily diagnosed by inspection of the wound. One or more coils of intestine usually protrude from the wound.

Wound dehiscence occurs most commonly in patients with malnutrition and malignant neoplasms. Frequently there has been postoperative peritonitis and paralytic ileus with abdominal distention. Violent muscular efforts associated with vomiting and coughing are often precipitating factors.

Wound dehiscence without separation of the skin wound usually occurs 7-10 days after operation. It should be suspected when ileus is persistent or develops without other signs of bacterial peritonitis. An almost pathognomonic sign is the escape of serosanguineous fluid from the wound, along a drainage tract or between skin sutures. If the dehiscence is of sufficient size, extruded loops of gut will be palpable under the skin and will produce a visible swelling. Wound abscess should be differentiated from dehiscence, but they may coexist.

Retroperitoneal Hemorrhage

This may be caused by trauma, breakdown of a retroperitoneal sarcoma, or a dissecting or ruptured aortic aneurysm. Blood behind the posterior peritoneum and infiltrating between the leaves of the mesentery causes abdominal pain, severe back pain, nausea, vomiting, and paralytic ileus. If blood loss is great, hemorrhagic shock ensues. Deep tenderness on abdominal examination is a usual finding.

Hematoma in Rectus Abdominis Sheath

A tear of the rectus abdominis muscle almost always occurs in the lower portion, producing a very tender, tense mass at this site. Most of the cases are seen in women during the third trimester of pregnancy, and there is usually a history of a blow to the abdomen. The mass is vertically elongated if the blood is confined by the rectus sheath. Aspiration of dark blood from the rectus sheath aids in the diagnosis.

Postoperative Intra-abdominal Hemorrhage

If massive hemorrhage occurs, shock and abdominal distention are produced. Pain may be severe, especially at the site of the wound. Early arrest of hemorrhage is essential. This complication must always be seriously considered when progressive shock appears early or late in the postoperative period. The history of events during the operation or the oozing of blood from the wound may help to confirm the diagnosis.

Acute Torsion of the Greater Omentum or an Appendix Epiploica

These have been mentioned in the differential diagnosis of acute appendicitis.

Ruptured Diaphragm

This is caused by violent direct or indirect trauma, often of a crushing type. The signs are those of the presence of abdominal viscera in the thorax, displacing the lung and heart. The diagnosis is confirmed by x-ray examination. Collapse may be profound, owing to severe trauma, associated injuries, and hemorrhage.

CONDITIONS WHICH MAY SIMULATE ACUTE ABDOMINAL CONDITIONS

Pneumonia With Pleurisy

This may simulate an acute abdominal condition by causing *severe epigastric pain* and *abdominal rigidity*. Pulmonary and pleural signs, with a raised temperature, pulse rate,

and respiratory rate, permit a correct diagnosis. True abdominal tenderness does not occur.

Coronary Occlusion

Severe coronary occlusion, accompanied by *shock, collapse, and epigastric pain*, may simulate an acute abdominal condition. At times *paralytic ileus* may develop, but it is rarely persistent. The history, absence of signs of peritonitis, heart signs, constricting chest pain, left arm pain, and pulse irregularities serve to differentiate acute coronary occlusion from abdominal conditions. Typical electrocardiographic changes are confirmatory.

Diabetic Acidosis

Abdominal pain and vomiting may occur in severe diabetic acidosis. *Some tenderness and rigidity* of the abdominal wall may occur. Glycosuria, acetone odor of the breath, deep sighing respirations, diminished serum bicarbonate concentration and elevated blood sugar level, and a history of diabetes mellitus permit differentiation from abdominal conditions. It must be realized, however, that acidosis may be precipitated by an infective intra-abdominal lesion, so that the latter must be carefully excluded.

Periarteritis Nodosa

This generalized disease produces signs of infection with fever, prostration, sweating, loss of weight, and eosinophilia. Abdominal symptoms may closely resemble those of acute ulcerative enterocolitis. Pronounced *abdominal pain, severe intestinal colic, nausea, vomiting, and melena* often occur. Other signs include those of acute cardiac or renal failure and peripheral neuropathy. Acutely painful areas are found in the skin and subcutaneous tissues. The diagnosis is often very difficult but can be made by microscopic examination of a subcutaneous nodule or the appendix.

Tabes

Gastric crises of tabes dorsalis are becoming relatively rare. *Extreme abdominal pain with copious vomiting* occurs in a crisis, but the abdominal rigidity disappears between the pains. Neurologic examination will reveal ab-

normal pupillary reactions and tendon reflexes. A gastric crisis must be differentiated from perforated ulcer

rigidity, and the vomitus does not become feculent.

Lead Poisoning

Lead *colic* of the small intestine is accompanied by constipation. The pain may be severe enough to produce *shock*. Rigidity of the abdominal wall appears only during the spasms of pain. The history of exposure to lead and a lead line on the gums are usually present. These features and the hematologic finding of stippling of the erythrocytes permit differentiation from other forms of colic.

Herpes Zoster

Herpes zoster involving the lower thoracic nerves gives rise to *severe abdominal pain* and *reflex muscle contraction*, the origin of which may be obscure before the skin rash appears. However, the pain is burning in character, and there is associated hyperesthesia. No peritoneal irritation is present, and the pain of unilateral herpes does not cross the midline.

Influenza

This may give rise to *abdominal pain* of moderate severity but can be differentiated from abdominal conditions by the history and by the presence of an upper respiratory infection. There are generalized muscular aches and pains but little or no abdominal rigidity. Fever is present from the beginning, and malaise is pronounced.

Typhoid Fever

This is discussed in the differential diagnosis of acute appendicitis.

Food Poisoning and Acute Gastroenteritis

The acute symptoms of staphylococcal food poisoning may simulate those of acute abdominal disease. *Abdominal pain*, *severe malaise*, *vomiting*, and *collapse*, with or without *cramps* and *diarrhea*, are frequently noted. The history may reveal the nature of the offending food, and other individuals may be similarly affected. There is no abdominal

Acute Leukemia

Acute leukemia may cause rapid enlargement of the spleen with *tenderness* and *rigidity in the left hypochondrium*. The white blood cell count and differential count provide the key to the diagnosis as well as generalized lymph node enlargement.

Tuberculous Peritonitis

Tuberculous peritonitis is not often an acute condition, but it may cause rather *acute abdominal distention*, *pain*, and *ascites*. There is little or no abdominal tenderness or rigidity, and tuberculosis elsewhere in the body will suggest the diagnosis. The abdomen has, typically, a doughlike consistency, and a mass of rolled-up omentum may be palpable.

ACUTE ABDOMINAL CONDITIONS OF INFANCY

(See also Chapter 30.)

There are several acute abdominal conditions peculiar to the *neonatal period* which have not been mentioned previously in this chapter. All of these acute conditions require immediate or early surgical relief. They are of congenital origin and may be listed as follows:

1 Lesions causing acute intestinal obstruction

- a Atresia of intestine or colon
- b Meconium ileus
- c Annular pancreas
- d Duplication of alimentary tract
- e Congenital obstructive band
- f Imperforate anus
- g. Volvulus of intestine

2 Omphalocele

An *obstructive lesion of the intestine or colon* is diagnosed by consideration of the history, physical examination, and x-ray findings. *Imperforate anus* can be diagnosed by inspection and digital rectal examination. The other obstructive lesions listed are not always identified preoperatively, but their sites can usually be determined with the aid of x-ray studies.

Vomiting of feedings and abdominal distention always occur in intestinal obstruction. If the infant vomits greenish material and exhibits visible peristalsis in the upper abdomen, together with dilatation of the stomach, it is likely that there is atresia of the duodenum or upper jejunum, meconium ileus, or annular pancreas obstructing the duodenum. If absence of vernix cells in the rectal contents can be demonstrated by means of the Farber test, the presence of complete obstruction is confirmed. X-ray films of the abdomen, with the infant held in various positions, will demonstrate dilated, gas-filled bowel and the point beyond which the gas cannot pass.

An *omphalocele* may be small or large and can be diagnosed by inspection. A defect in the abdominal wall is evident at the umbilical region, and coils of bowel can be seen through the semitransparent, thinned-out membrane. Immediate operation is the only hope of saving the life of the infant because drying and rupture of the membrane will lead to evisceration, contamination, and fatal peritonitis.

Congenital hypertrophic pyloric stenosis is a subacute abdominal condition which may develop in a young infant, and its description may be found in Chapter 30.

FINAL COMMENTS

An acute abdominal condition presents one of the most fascinating and complex problems in surgery. Decisions concerning treatment must frequently be made promptly on the basis of less evidence than might be obtained if more time for observation and investigation were available. The surgeon must decide whether an operation should be carried out immediately or deferred for a period of time, and, if so, for how long. Certain diagnostic steps should be taken immediately; these have been described in detail near the beginning of this chapter.

Diagnosis of an acute abdominal condition involves consideration of so many possibilities that it is wise first to consider the more common conditions. Less than 20 acute conditions are of common occurrence, although more than 70 have been discussed in this chapter. The vast majority of cases can be included in the following abbreviated list.

COMMON CONDITIONS REQUIRING IMMEDIATE OR EARLY TREATMENT

Operative Treatment

1. Acute appendicitis
2. Acute cholecystitis
3. Intestinal obstruction
4. Perforated viscus
5. Massive hemorrhage
6. Strangulated hernia
7. Ruptured ectopic pregnancy
8. Twisted ovarian cyst

Nonoperative Treatment

1. Paralytic ileus
2. Acute pancreatitis
3. Renal colic
4. Urinary retention
5. Acute hepatitis
6. Biliary colic
7. Acute gastroenteritis
8. Pelvic inflammatory disease

It should be realized that it is better to recognize an acute surgical condition of the abdomen and to act promptly than to wait for further information of a more confirmatory nature in the later stages of the disease and operate either too late to do good or under circumstances less favorable for recovery (Lichtenstein). However, the surgeon who opens the abdomen should be capable of dealing with any condition he may find there.

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
The Diagnosis of Surgical Lesions of the Alimentary Tract Part I The Primary Examination (Shows importance of history-taking, physical examination and laboratory procedures in making correct diagnosis) (1950) (Directed by Arthur W Allen, M D, Boston)	14 min	Sound	Director, Armed Forces Institute of Pathology Washington 25, D C.
The Diagnosis of Surgical Lesions of the Alimentary Tract Part II Special Techniques and Apparatus (Illustrates special tests used to assist in making diagnosis of various surgical lesions of the alimentary tract) (1950) (Directed by Arthur W Allen, M D, Boston)	20 min	Sound	Director, Armed Forces Institute of Pathology Washington 25, D C.

Peripheral Vascular Diseases

Josephus C. Luke, MD

CONGENITAL VASCULAR ANOMALIES

1. Hemangioma
2. Congenital arteriovenous fistula
3. Glomus tumor

Hemangioma

This group of tumors consists of an actual new formation of blood vessels, probably arising from isolated elements of the vascular mesenchyma which have not been included in the formation of the normal vascular tree. The evidence for this theory is the relative frequency of circumscribed angiomas, with one to three entering arteries and an equally small number of efferent channels. The extensiveness of the hemangioma is probably an indication of its date of commencement, the small ones commencing toward the end of fetal life, while the extensive ones involving superficial and deep tissues arise early in the development of the embryo. The tumor usually continues to enlarge until growth ceases, but in those where arteriovenous connections are present, such as the cirrroid type, the increase in size is continuous. Pregnancy and the onset of menstruation may result in an increase in the rate of growth.

Most of the hemangiomas are present at birth and most of the remainder appear in early childhood. It is the commonest tumor of infancy and childhood and, for some unknown reason, is twice as common in females as in males.

Capillary Hemangioma

In this group are several varieties which have acquired different names, but all are similar in respect to their congenital origin and the fact that they occur in various surfaces, chiefly the skin and mucous membranes. The blood vessels are capillary in type, lined by a single layer of endothelium, and sparsely filled with blood.

Port-Wine Stain.—This deforming manifestation of a capillary hemangioma appears at birth and grows with the development of the child. The condition involves the derma and exhibits diffuse telangiectasia but no tumorous enlargement. With years, the color tends to change from a pink to a bluish purple hue. The face and oral cavity are most frequently involved, but the trunk and limbs are also commonly affected.

Treatment is difficult, and no one method is likely to give a good result. The abrasive method of Jonsson is probably the best available. In this, the involved area is rubbed with sandpaper to destroy the cutis and the capillary dilatations. Care must be taken to leave part of the derma, as scarring will otherwise result. Novocain with Adrenalin is used as the anesthetic to produce insensitivity and reduce hemorrhage. A moderately large area can be treated in one sitting. Tattooing of the abnormal area is also used, employing a mixture of pigments to produce as normal a skin color as possible. Only small areas can be dealt with at one time and, consequently, treatment may

to explain the rather rare condition of congenital arteriovenous fistula (usually multiple) in which direct communications exist between otherwise normal arteries and veins. Various varieties occur, the localized ones resembling the cavernous hemangiomas and the generalized variety revealing their presence by generalized changes in the extremity.

Localizing Types.—The terms *cirroid* and *racemose aneurysms* are used in this variety, the latter applying to those in the neck (see above). The points of differentiation from

a cavernous hemangioma are the greater extent of the lesion, more rapid progression, the pulsatile character, and the presence of a bruit over the lesion. Frequently in association are other vascular anomalies and abnormalities such as capillary hemangiomas and marked engorgement of superficial veins.

Generalized Types.—As mentioned previously, the extremities are most commonly involved in this variety, and the diagnosis is made by the recognition of the effects of these vascular communications. Varicose veins, due

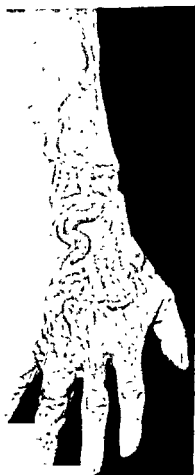
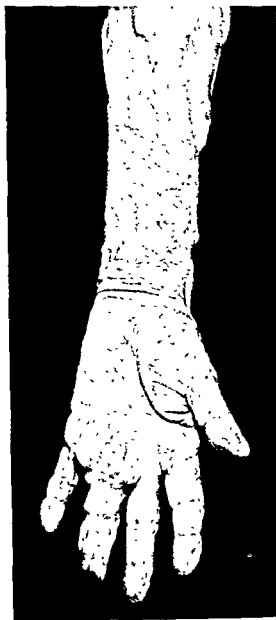


Fig. 171.—Generalized type of congenital arteriovenous fistula involving the palm and 5th finger. Note the marked enlargement of the forearm veins.



Fig. 472—Arteriogram of Fig. 471 showing the dilated and tortuous radial and ulnar arteries and the multiple arteriovenous communications in the palm and 4th finger



Fig. 473—Congenital arteriovenous fistula involving the sole of the foot, associated with changes in the leg due to marked venous stasis



Fig. 474—Arteriogram of Fig. 473

to increased venous pressure, are almost invariably present, coming on early in life and being gradually progressive. Consequently in a young patient with severe varicose veins, the condition should be especially suspected. The complications of these veins, such as ulceration and eczema, may first bring the patient to medical attention. There is an associated enlargement of the limb because of the venous engorgement, increased length of the leg because of epiphyseal vascular stimulation, and increased warmth. Increased sweating and growth of hair are also commonly seen. Various types of surface hemangioma may be associated. A bruit or thrill is rarely present due to the small size of the fistulas.

Laboratory aids in arriving at a diagnosis are the finding of increased oxygen values in the venous blood and arteriography which indicate the multiple intercommunications. Treatment is unsatisfactory due to the numerous small fistulas making impossible their interruption. Excision or stripping of the dilated superficial venous channels is indicated, especially where complications as the result of these veins are present. Compression bandages or a strong elastic stocking, to give an increased tissue tension inhibiting the arteriovenous shunt, should be worn continuously. A scrupulous search with a stethoscope should be made over the area to determine points of localized bruits. Such a bruit indicates a collection of large arteriovenous shunts which, being localized, are capable of surgical excision.

Glomus Tumor

Glomus tumors are not definitely congenital in origin but are included in this group for convenience in classification. The glomus body is a specialized arteriovenous communication which develops shortly after birth and which has the function of local and general heat regulation and also some connection in the regulation of blood pressure. These glomus bodies are microscopic in size and are most frequently found in the extremities but are also present in other parts of the body. As many as 70 such bodies may be present in one toe. They consist of thickened arteriovenous vascular channels associated with large epithelioid cells which are peculiar to these bodies and are

known as glomus cells. Numerous nonmyelinated nerve fibers are in close association.

Glomus tumors are overgrowths of the glomus body enlarging to macroscopic size. A common situation is beneath the nail, but they are also present in other locations in the extremities. The enlargement is slow and metastases do not take place. Recurrence has occurred following excision, but such a recurrence may be due to the development of a new tumor in a contiguous area.

The diagnosis is not difficult if the condition is kept in mind. They manifest themselves as small pinhead or slightly larger bluish nodules in the skin or beneath a nail. Pain is the predominant symptom. It is neuralgic in type, present in the region of the tumor, and is aggravated by cold or heat. Local pressure on the spot produces severe, excruciating, and lancinating pain to such a degree in some cases that the patient shrinks from the slightest contact. When beneath the nail, erosion of the underlying phalanx may take place by direct pressure of the tumor. Treatment consists of surgical excision.

TRAUMATIC ARTERIAL LESIONS

Arterial Spasm

The smooth muscle of the arterial wall has the property of local contraction of a small segment or, as the result of irritation of a localized area, of producing an arterial spasm of the entire limb. Whether the mechanism of this vessel contraction is the result of local overstretching of the smooth muscle fibers and consequent spasm or whether the injury causes severe sympathetic stimulation is not certain. The fact remains that subsequent to trauma of many kinds, arterial spasm can occur severe enough at times to cause complete ischemia of the limb and subsequent gangrene.

This condition is seen most often in wartime due to the passage of high velocity missiles close to major arteries with momentary lateral stretch of the vessel. It has followed other soft tissue wounds, the reduction of fractures, and various orthopedic operations and has been reported as following the injection of sclerosing fluids in the treatment of varicose veins. All degrees of spasm occur from the minor unimportant transient degrees

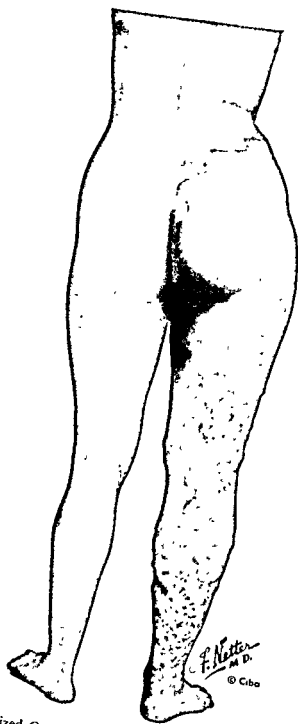


Plate 50.—Generalized Congenital Arteriovenous Fistula Involving the Right Leg.
Note the enlargement of the leg and increased length, the superficial vein dilatation, the venous stasis effects in the leg, and the associated port-wine stain.

Courtesy Luke, Josephus C. CIBA
CLINICAL SYMPOSIA 3 99, 1953



FIG 1
ARTERIOSCLEROTIC OCCLUSION
OF ABDOMINAL AORTA AND
BIFURCATION. (VIEWED THROUGH
SURGICAL EXPOSURE)

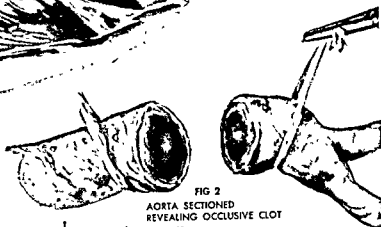


FIG 2
AORTA SECTIONED
REVEALING OCCLUSIVE CLOT

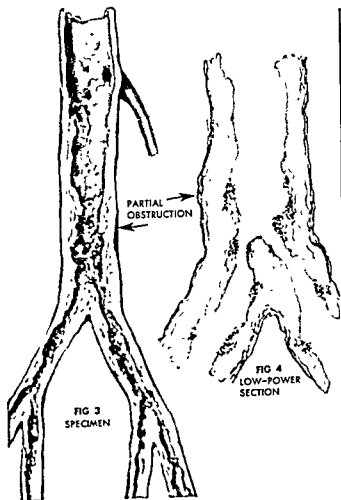


FIG 3
SPECIMEN

PARTIAL
OBSTRUCTION

FIG 4
LOW-POWER
SECTION

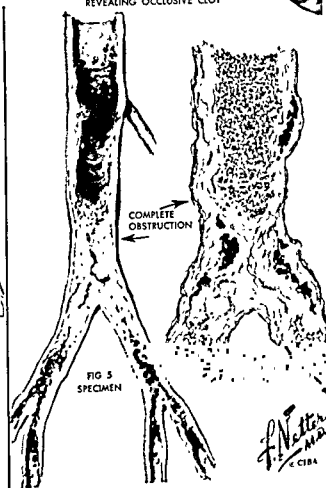


FIG 5
SPECIMEN

COMPLETE
OBSTRUCTION

J. Netter M.D.
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Plate 51.—Aortic Occlusion.

Courtesy DeBakey, M. E., Conley, D. A. and Creech,
O. A. CIBA CLINICAL SYMPOSIA R 45, 1956

to the severe type with resultant gangrene. The condition known as Volkmann's ischemic contracture of the forearm and hand following elbow injuries and fractures is probably due to this mechanism.

The prognosis of the limb in the severe states depends on early recognition of the condition in the acute phase so that gangrene can be forestalled. The color, temperature, and sensation of the hand and foot along with the peripheral pulses should be palpated routinely after every limb operation or injury because the majority of extremities lost from this cause are the result of late recognition. Dressings should be loosened, splints removed, and abnormal stretching of joints released. Antispasmodic drugs such as Priscoline 50 mg. doses intravenously are of some value; release of sympathetic tone by procaine block of the appropriate sympathetic chain should be done. Increased body warmth and the correction of anemia and blood volume are also indicated. If conservative treatment fails, then surgical exposure of the affected artery should be done without delay. The spastic vessel will frequently relax on contact with warm saline solution applied to the adventitia or injected under pressure intra-arterially. Procaine solution can also be used similarly. Resection of the spastic segment may be necessary as a last resort with replacement by a vascular graft.

Arterial Contusion

Damage to a segment of arterial wall can be produced by direct violence of the original traumatic agent, by operative trauma, or by damage to the artery by a fractured segment of bone. The result of this trauma is hemorrhage into the arterial wall leading to possible thrombosis because of the intimal damage or secondary hemorrhage from later rupture of the damaged artery. As indicated above, possible widespread arterial spasm may also be associated.

Treatment is only indicated when total ischemia is present or threatened, and it is only when the artery is exposed surgically that the lesion can be differentiated from arterial spasm. Arterectomy of the damaged segment is indicated with replacement by a vascular graft when an artery of major caliber is involved.

Partial Division With False Aneurysm

When a major artery is partially severed, especially if done by a small sharp object such as an ice pick, fragment of a grenade shell, or occasionally a bone spicule, bleeding occurs from the arterial hole into the surrounding tissues but is prevented from escaping to the exterior by the smallness of the missile puncture wound. A deep hematoma develops, with thrombosis and fibrosis of its outer layers, containing fluid blood in its interior which flows to and from the arterial tear. The lesion is also aptly called a pulsating hematoma and is a false aneurysm inasmuch as the cavity has initially no endothelial lining.



Fig. 475—Section of brachial artery removed from case of severe fracture of the humerus showing arterial contusion.

The diagnosis of such a lesion depends on a careful search in all puncture wounds and fractures for the following signs:

1. A pulsating swelling in the region of injury.
2. A systolic bruit and thrill over the involved area, the bruit being propagated distally.
3. Later evidence of nerve compression by the hematoma which may increase gradually in size.

It is only in the most major degrees of arterial puncture and partial division that the distal circulation is impaired.

Initial treatment consists of immobilization of the limb and pressure over the injured part. If the pulsating tumor is not increasing in

size and evidence of progressive nerve pressure is absent, then conservative therapy is indicated for a time because the smaller varieties tend to heal spontaneously. These aneurysms may persist undetected for years but are prone to thrombosis which extends to the major limb artery with consequent gangrene. If persisting beyond 3 months, surgical correction is indicated with removal of the false sac (if possible) and closure of the arterial tear.

is small or if produced by a closed type of injury, then a massive hematoma rapidly collects.

In either instance the distal parts are threatened with gangrene due to partial or complete ischemia, the degree of ischemia paralleling the amount of remaining functioning collateral vessels.

Rapid surgical measures are indicated consisting of replacement of the blood loss and

Incidence of Gangrene in Main Vessel Interruption

	Ligation Only	World War I wounding + ligation	World War II wounding + ligation
Subclavian	0	8.8%	24.0%
Axillary	14%	2.7%	25.0%
Brachial	0	4.0%	?
Femoral	17.2%	20.2%	32.0%
Popliteal	26.6%	34.7%	86.0%

Fig 476

Late Results Following Arterial Ligation

Muscle

1. Intermittent claudication
2. nocturnal cramps (rest pain)
3. paralyzed extensor hallucis longus muscle
4. paralysis of small muscles of hands and feet

Sensory

1. cold dead feeling of limb
2. anaesthesia of glove or stocking type
3. hyperalgesia
4. delayed pain response to noxious stimuli

Fig 477

When the arterial wall defect is extensive and located in a major artery, then resection of the segment is indicated with replacement by a vascular graft. Resection with end-to-end anastomosis is possible in the smaller sizes, when involving a lesser artery, excision only is indicated.

Complete Division

This type of arterial trauma is the most serious of all, both from its high mortality and high incidence of gangrene of the limb. If the wound is open, death from exsanguination is probable unless rapid first-aid measures are carried out. If the external wound

exposure of the severed artery. Ideally, débridement of the severed arterial ends should be done with end-to-end suture. However, in the majority of cases this is not feasible because of the extent of the arterial damage. The majority of such cases will require ligation of the damaged artery with subsequent all-out efforts to keep the functioning collateral vessels as dilated as possible. This is accomplished by repeated lumbar sympathetic procaine blocks or sympathectomy if the patient's condition will allow. With the rapid improvement in the techniques of vascular surgery, arterial ligation should be replaced by the insertion of a segment of vein or homologous arterial graft to

bridge the defect. With these methods a high incidence of limb salvage will be achieved. The greatest number of these cases are seen during warfare, and it is likely that during World War III, banks of homologous or synthetic arterial grafts will be available to the forward surgeon for this type of case. Reports from the recent Korean conflict indicate that about an 80% salvage rate can be achieved in devitalized limbs with the use of this technique.



Fig. 478.—Femoral arteriogram showing traumatic arteriovenous communication between profunda branch artery and vein

Arteriovenous Fistula

A small sharp object such as a fragment of shell may puncture both the main artery and vein. In such an instance the usual false aneurysm does not occur because the escaping arterial blood flows directly into the vein. Such fistulas rarely close spontaneously and soon become lined by vascular endothelium. Due to

the pouring of arterial blood directly into the venous system through a relatively large opening, marked abnormal physiologic changes result which affect the entire cardiovascular system.

1. An immediate fall in blood pressure which later returns to normal levels, but a permanently lowered diastolic pressure which remains, giving an increased pulse pressure

2. Increase in pulse rate; abruptly slowed by 8-16 beats/minute (depending on the size of the fistula) when the artery is compressed proximal to the fistula—phenomenon known as Branham's sign

3. Increased venous pressure in the veins both distal and proximal to the fistula due to the direct dumping of arterial blood into the vein

4. Increased cardiac output due to increased inflow

5. Gradual increase in total blood volume

6. Gradual dilatation and hypertrophy of the heart which may go on to cardiac failure when the fistula has been present for long periods

7. Rapid development of the arterial collateral circulation in the region of the fistula

Clinical manifestations include a thrill over the fistulous tract with a bruit which is loudest in systole but is also heard in diastole (so-called machinery murmur). Decreased peripheral arterial flow will be evident, depending on the size of the fistula. Diminished distal arterial pulses will be present and the ischemia may be severe enough in the large fistulas that distal gangrene may develop. The size of the extremity is increased, and also elevated surface temperature is present. In later cases, evidence of increased venous pressure will be present in the form of varicose veins, some dependent cyanosis, edema, and possibly the complications of venous stasis (ulceration and eczema). An arteriogram will give the location and size of the fistula.

Treatment to close the fistulous tract is indicated because of the late cardiovascular changes. This should be delayed for more than 2 months to allow the collateral vessels time for maximal dilatation. A preliminary sympathectomy of the involved limb is also useful in

this respect to minimize the dangers of acute arterial ischemia to the distal limb immediately after operation. The surgical operation of choice is repair of the arterial opening so that the continuity of the main artery is preserved. However, this is frequently not feasible and the operation most commonly employed is

ARTERIAL DISEASES

Thromboangiitis Obliterans /

Thromboangiitis obliterans was first described as a definite entity by Leo Buerger in 1908 and so is commonly known as Buerger's disease. It had been previously described but

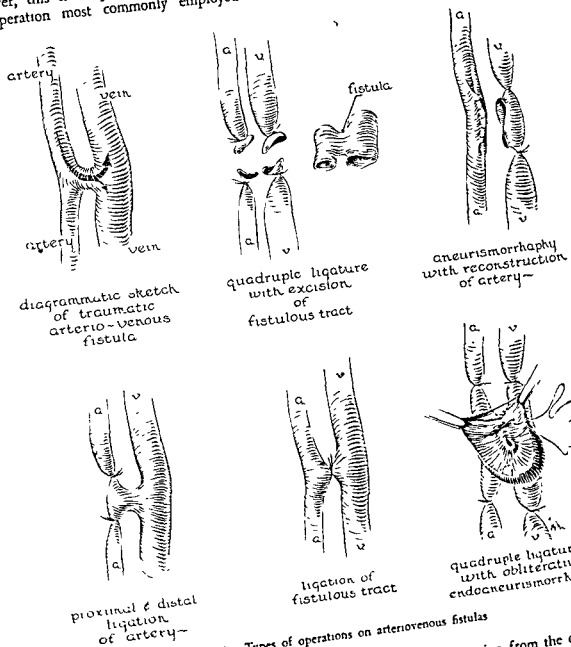


Fig. 479—Types of operations on arteriovenous fistulas

quadruple ligation, tying the artery and vein both proximal and distal to the fistula with, preferably, excision of the fistulous area. If any doubt exists in respect to adequate distal arterial supply, then grafting of the affected arterial segment is indicated

with little differentiation from the other types of occlusive arterial diseases. Buerger's pathologic studies made him conclude that the condition was a subacute inflammation of the vessels leading to thrombosis with subsequent organization and partial recanalization of the

thrombus. The disease can be defined as a segmental, inflammatory, obliterative disease of the arteries and veins which occurs almost exclusively in young men, involving the extremities and occasionally the viscera. The Jewish race appears more susceptible than the Gentile for reasons which are unknown.

Etiology.—The exact cause of this disease is uncertain. Various theories have appeared but each is incomplete. These include focal infection, an allergic response to unknown stimuli and hence related to periarteritis nodosa and the collagen diseases, and, finally, tobacco. This latter factor is the only one concerning which definite knowledge exists, and it appears to be true that the disease is adversely affected by smoking. The causative factor in tobacco smoke is as yet unknown as it has been shown that dencotized tobacco is just as harmful as the true product.

The high preponderance of this disease in males suggests also some protective influence in the female. This is possibly the vasodilating effect of the estrogens or some other side effect because experimental evidence has shown that female rats are less likely to get gangrene of the tail and that male rats are protected from the effect of ergotamine injections when given theelin. The unprotected male rat is invariably affected following ergotamine.

Pathology.—The disease varies in intensity in different individuals and may be a slowly progressive one with long periods of quiescence or may be a rapidly fulminating type with rapid progression and early loss of one or more limbs due to gangrene. The disease can be divided into four types according to severity.

- 1 Nonprogressive type in which a good collateral circulation develops and which becomes quiescent after one or two episodes.
- 2 The type presenting sudden major arterial or venous occlusion.
- 3 Slowly progressive type which may eventually lead to gangrene or leave a badly impaired arterial tree.
- 4 Fulminating rapidly progressive type requiring multiple amputations.

Microscopically, one sees a diffusely cellular thrombus blocking the arterial lumen, affecting chiefly the smaller arteries such as the dorsals

pedis, posterior tibial, ulnar, etc. The arterioles and the large arterial trunks are seldom involved. A well-preserved media is present and there is some degree of endothelial proliferation. Fibrosis and lymphocytic infiltration of all coats are present, especially in the adventitia and about the vasa vasorum. The accompanying nerve is frequently involved in this periarterial fibrosis, and the vein can show the same involvement of the wall as is seen in the artery. It appears as if some factor primarily involves the endothelium causing a proliferative and irritative effect which results in exudative changes and thrombosis. It is possible that the fibrosis is a reparative aftereffect. Calcification in the vessel wall is very rare.

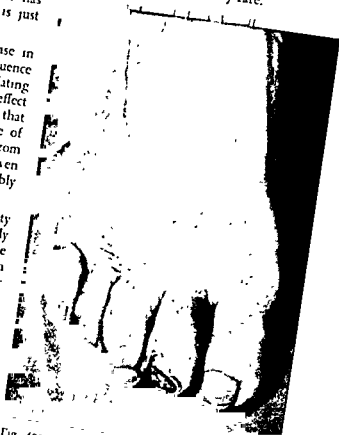


Fig. 480—Typical toe gangrene in Buerger's disease

Diagnosis.—It must be emphasized that this disease occurs between the age limits of 15-45 years, and it is very doubtful whether such a diagnosis should be made in a person whose symptoms commenced after the age of 45 years. Frequently a diagnosis of Buerger's

comes narrowed or blocked by atheromas or thrombus formation that symptoms occur from ischemia. The same applies to other more important arteries such as the coronary or cerebral vessels.

Pathology.—The changes in arteriosclerosis are the result of the wear and tear phenomena taking place throughout life. However, in certain individuals and in certain disease states, arteriosclerosis appears to develop earlier than

families to the early development of this condition. An elevated blood cholesterol appears to be an influencing factor, and an increase in certain lipid fractions may also play a part.

Two main pathologic types of arteriosclerosis are seen, the more common being the subintimal degeneration and intimal thickening which goes on to produce atheromatous ulcers projecting into the vessel lumen or which become calcified to form the typical plaque



Fig. 184.—Diabetic arteriosclerotic gangrene involving the right great toe. The trophic ischemic changes in the toes and foot are well shown.

in the majority. Calcified spots in major arteries are not uncommon in the late 30's, and arteriosclerosis appears earlier in individuals with diabetes or other lipoid metabolic upsets such as *xanthoma tuberosum* and *myxedema*. There is an increased incidence in certain

The variety is known as *atherosclerosis*. Fibrosis of the media and adventitia is also associated and the elastic laminae show degeneration. The above changes account for the rigidity and tortuosity which an arteriosclerotic vessel demonstrates. Calcification may

be present not only in the plaques but may also be diffusely laid down in the area between the media and intima. With such changes therefore in all the coats of the vessel, especially the intima, it is not surprising that thrombus formation takes place in the lumen. A second variety of arteriosclerotic change is that frequently seen particularly in the arteries of the extremities. This consists of necrosis and calcification of the media, but without gross intimal change, and again the vessels become transformed into rigid tubes (*Mönckeberg type*).

The important phase in the production of arterial obliteration is the development of a thrombus in the vessel lumen or the occlusion of the vessel by masses of atheromatous material. Frequently the two processes are combined. This is most likely in vessels where atheromatous ulceration is present. Such ulcers are commonly situated in large arteries such as the lower end of the aorta where the volume and force of the blood flow prevent for a long time a complete occlusion. Blockage may occur in smaller vessels where the changes are minimal and no evidence of calcification is seen on x-ray.

Symptoms and Signs in the Extremities.—

It is a fact that the arteries of the upper extremity are rarely involved from the clinical standpoint by arteriosclerosis obliterans, and if one artery does become blocked, the collateral circulation is sufficiently good to prevent distal ischemic changes. Therefore, it is the lower extremities that almost invariably suffer from this disease. The patient seeks medical attention usually because of the sequelae of inadequate arterial inflow to the limb, and it must be emphasized that these symptoms are not typical of arteriosclerosis obliterans alone but are present in arterial ischemia due to any cause. The most important symptom is pain which is of two main types. The first is *rest pain* where malnourished tissue produces a scalding, burning pain, probably the result of ischemic changes in the somatic nerves. This pain is always worse at night when the legs are horizontal, aggravated by elevation of the limb, and relieved by hanging the legs over the side of the bed. Pain is also present in association with gangrenous and ulcerative lesions. The second main va-

riety of pain is the type known as *intermittent claudication*. This is a most exact and diagnostic symptom and, as before mentioned, can occur in any variety of arterial obliterative disease of the limb. Intermittent claudication is a cramplike, squeezing pain occurring most commonly in the calf muscles on exercise. The location of the pain depends on the level of the arterial blockage. It may be felt in the foot when the occlusion is in the region of the

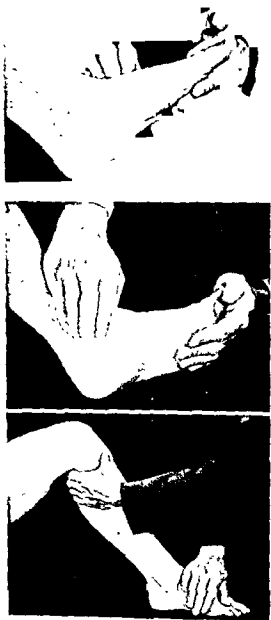


Fig. 485—Technique of palpation of the dorsalis pedis, posterior tibial, and popliteal arteries.

knee, in the calf when the block is in the thigh, or in the lateral thigh when the iliac or common femoral artery is involved. This pain is due to muscle ischemia and therefore is felt only on exercise. Rapid walking, walking uphill, or climbing stairs will initiate the pain sooner than a slow pace on level ground. After a rest of 1-2 minutes, the pain disappears, only to appear again after a similar distance has been covered. The distance a patient can walk before the onset of intermittent claudication is a good index to the severity of the arterial occlusion and may vary from 50 feet in the severe cases to a quarter mile in the milder ones.

Other symptoms of arterial ischemia are coldness of the feet, nonhealing infections or ulcers, and various sensory changes, including a hyperesthesia or a numbness and sensations of pins and needles. Examination shows evidence of varying degrees of trophic ischemic changes in the soft tissues of the foot, such as thinning with shininess and loss of elasticity of the skin, decrease in the amount of subcutaneous tissue and atrophy, brittleness, and slow growth of the nails. Hair growth is poor, and hair may be shed from the affected extremity. Sweat glands atrophy, and the foot appears dry and scaly. Ischemia on elevation occurs because of increased venous return and retarded arterial inflow due to the effect of gravity. In hanging the limb down, rubor appears due to pooling of blood in the capillaries and insufficient force at the arterial end of the loop to keep movement rapid. The rapidity of the development of this rubor and its degree are also good indications of the degree of arterial ischemia. A foot which rapidly develops a deep rubor is one with a severe degree of arterial block. A small area of gangrene or infection may be the first signal of arterial disease. These usually result from trauma of some sort to the foot and point a moral that the arteries of every foot should be examined properly and patency assured before an elective procedure is done on the foot, even to the cutting of an ingrown toenail.

Treatment.—

1. Care of the feet: General measures to prevent minor trauma to the feet will obviate many of the disasters that may occur. The well-known paint advertisement, "save the

surface and you save all," applies here; washing is obligatory, as is the wearing of soft wool socks. The feet should be oiled with lanolin or cold cream every second day. Shoes should be pliable and loose fitting to prevent corns and blisters. Toenails need frequent cutting to prevent digging in at the base, the best method being to cut the nails straight across and to scrape the convex surface of the nail that tends to be ingrown. Fungus between the toes should be energetically treated if it develops.

2. Complete bed rest is indicated if gangrene or local infection is present. Active exercises should be carried out to prevent muscle atrophy where prolonged immobility is necessary. Dry dressings only should be used on open areas, and antibiotics are useful where infection is present.

3. Ethyl alcohol is a useful vasoconstrictor agent and is also an aid in analgesia if pain is present. Liberal amounts should be used.

4. Heat should not be applied to the affected part but can be used to good effect if employed to produce a state of general vasodilatation. This is accomplished by hot water baths (110°-115° F.) or a baker to the distal area, producing a state of general vasodilatation with benefit to the circulation of the legs.

5. Vasodilating drugs have not proved to be of much benefit because of their uncertain and transient effect. It is not reasonable to expect a badly damaged vascular bed such as that in advanced arteriosclerosis obliterans to respond to much vasodilatation. Also it must be remembered that these drugs have a generalized effect which will be major in the lesser involved vessels elsewhere in the body and so can possibly decrease the peripheral blood flow because of their action on the remainder of the body. Many supposed good results of vasodilator drugs are really the result of the adaptive capacity of the vascular system. After a period of months following the partial or complete occlusion of a main vessel, the collateral circulation gradually dilates because of the greater volume of blood forced into it. Peripheral circulation improves and, if any of these drugs were coincidentally given

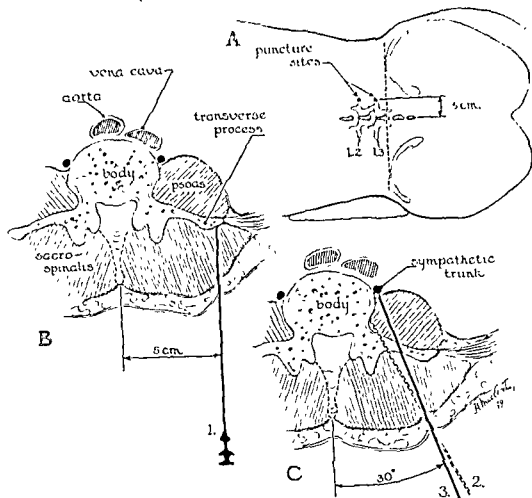


Fig 486—Technique of lumbar block

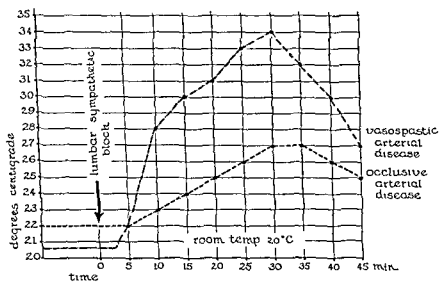


Fig 487—Typical skin temperature rises

improvement is credited to it. For example, ligation of the superficial femoral artery in a normal individual results in a loss of pedal pulses for only 1-3 months.

Many vasodilating drugs of variable usefulness are at present on the market. Their popularity is evanescent and it is beyond the scope of this book to discuss each separately. Many more will undoubtedly appear in the near future. Those currently in vogue are nicotinic alcohol, Priscoline, and diergocorine.

that it is not a cure but only a method of improving the circulation to the leg. The resulting degree of improvement will depend upon the existing circulation. In those cases with severely involved vessels, the amelioration would be slight and does not warrant the procedure. The operation is indicated where arteriography has revealed that the arterial lesion is not suitable for the replacement by graft but where a relatively good collateral circulation is demonstrated by a rise in foot

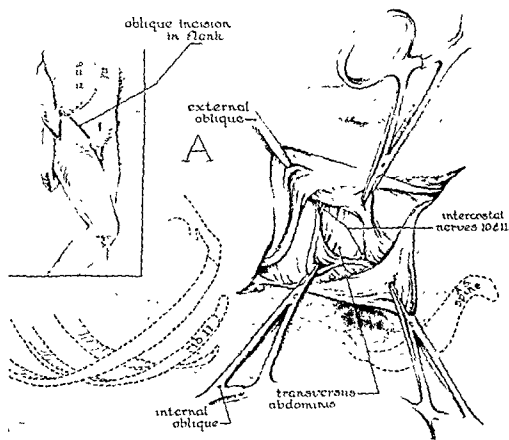


Fig. 488.—Technique of lumbar sympathectomy. (See continuation on next page.)

6 In arteriosclerosis the collateral vessels may still show potential vasodilatation. This can be determined if a rise in skin temperature of the foot follows procaine block of the lumbar sympathetic chain. If such a true rise occurs of over 1°C following these tests, then maximal permanent vasodilatation can be accomplished by the operation of lumbar sympathectomy.

This operation carries a low morbidity and mortality, but the patient should be warned

skin temperature following lumbar procaine block. In such a case, an indolent ulcer will heal and ischemic neuritis improve. Intermittent claudication in the calf is rarely improved by lumbar sympathectomy unless the arterial occlusion is in the popliteal artery. Sympathectomy is also useful in association with minor amputations to improve wound healing.

7. *Arterial grafts.* One of the dramatic surgical advances of the past 5 years has been

the use of grafts to replace occluded or partially occluded vessels and aneurysms. This advance has been made possible by the widespread use of arteriography, which reveals the exact state of patency of the arterial tree, and by the development of arterial graft banks. The techniques of arterial replacement have advanced to the point where any well-trained surgeon should be capable of performing these procedures.

It is our practice to perform femoral or aortic arteriography on all cases of arteriosclerosis obliterans which show evidence of arterial blockage in the femoral artery or higher. A surprisingly high percentage will show evidence of a segmental occlusion with good or relatively good vessels proximal and distal to the occlusion. It is this group of cases which is suitable for arterial grafting. Segmental occlusions are found even where toe

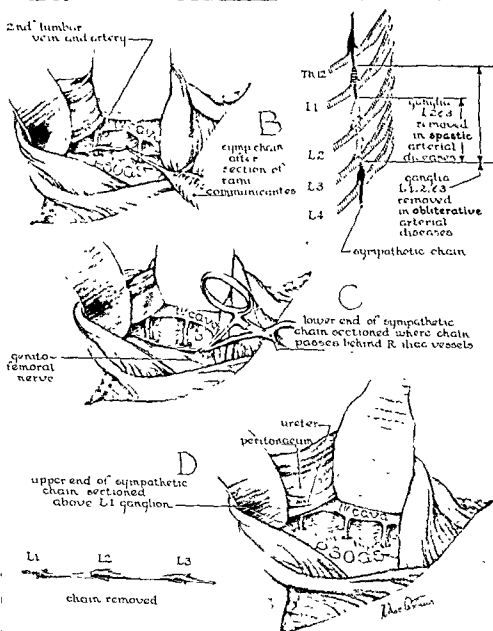


Fig 488 (cont'd).—Technique of lumbar sympathectomy.



Fig 489—Femoral arteriogram, with accompanying diagram, showing segmental femoral artery occlusion. This is a suitable case for a by-pass graft to restore normal circulation (From Luke, J C. *Postgrad Med* 22: 10-20, 1957)

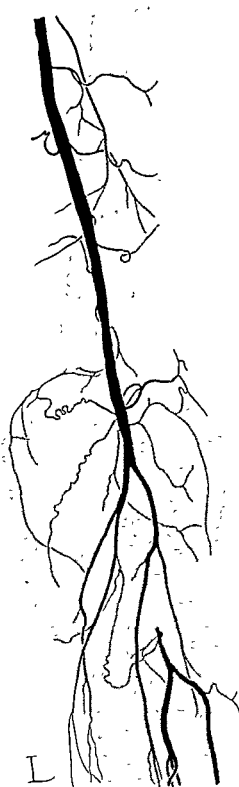


Fig. 490.—Femoral arteriogram, with accompanying diagram, showing popliteal artery occlusion. This case was not suitable for grafting, but the patient's intermittent claudication was relieved by lumbar sympathectomy. (From Luke, J. C.: *Surgery* 41: 165-171, 1957.)

or foot gangrene is present, but usually in such cases arteriosclerosis is more advanced than in those with intermittent claudication.

Criteria for Selection of Cases

- 1 Good quality femoral or aortic arteriograms
- 2 Evidence that the arteriosclerotic process is not severely widespread in the arteries under consideration
- 3 Evidence that the outflow distal to the occluded segment is of good caliber
- 4 No major arteriosclerotic manifestations in cerebral, cardiac, or renal areas
- 5 Availability to an arterial bank or, as second choice, the use of plastic cloth prostheses
- 6 Plenty of operating time, a steady hand, and an equable temper

The segmental occlusion has usually been slowly progressive, allowing the development of a relatively good by-passing collateral circulation, and consequently no severe ischemic changes occur in the foot. However, with this

main vessel occlusion, blood flow to the distal muscles on exercise is insufficient, and typical intermittent claudication is present. As mentioned previously, the muscles involved by the claudication will depend on the level of main vessel block. Leriche's name is given to the syndrome in which claudication pain is felt in the low back and buttocks, leg atrophy and weakness are present, and impotence occurs in the male. This results from segmental occlusion of the abdominal aorta below the renals. Blockage of the common iliacs is usually associated. Occlusion can occur at any point from this level distally, the most common site being in the femoral artery commencing at the adductor canal level.

These blocked segments of artery can be treated by two different techniques, *i.e.*, arterial grafting or thromboendarterectomy. The former is more commonly used because of its



Fig 491 —Thrombotic occlusion of the lower aorta and common iliac arteries (Leriche syndrome).
Note the patent normal-appearing distal vessels

wider applicability. The grafts employed are chiefly preserved homologous grafts stored in an artery bank. However, plastic cloths woven to appropriate artery size are also used and appear to be just as effective as homografts in major arteries such as the aorta or iliacs. In smaller arteries such as the femoral, the woven grafts appear to have a higher rate of thrombosis. Autogenous veins may be used as grafts but are only suitable for smaller arteries as they are not strong enough to withstand the pressure within the major arteries. Thrombo-

endarterectomy (Fig 496) is a procedure in which the occluding material, including the thickened intima, is removed, with repair of the arteriotomy incision and thus restoration of main vessel flow. This is a better procedure as the end result is the equivalent of an arterial autograft and obviates the disadvantage of placing foreign material in the arterial tree. At the present time thromboendarterectomy is chiefly employed in cases of short segmental occlusion of major vessels and in those where a neglected embolus is present.



Fig 492.—Postoperative aortogram of patient shown in preceding figure. The main vessel continuity has been restored by the operation of thromboendarterectomy.

When grafts are used, the blocked artery can be resected with end-to-end anastomosis between the host artery and the graft. This technique is usually employed where major vessels are involved. Where smaller arteries such as the femoral are occluded, a better method is the end-to-side technique in which the end of the graft is sutured to an opening in the side of the host vessel above and below the involved segment. The occluded segment is not removed, and consequently no collateral

vessels are disturbed. The success rate with this by-passing technique has been much higher than in the older end-to-end methods.

Grafts applied to major arteries are almost invariably successful in restoration of normal blood flow, but where smaller grafts are used a higher rate of thrombosis occurs. In our series of 70 femoral by-pass grafts, the immediate and late failure rate has been about 35%. In the more advanced cases where considerable widespread arteriosclerosis is present



Fig. 193—Aortogram revealing arteriosclerotic occlusion of lower aorta and common iliac arteries. The patent distal main arteries indicate suitability for the operations of thromboendarterectomy or resection and grafting.

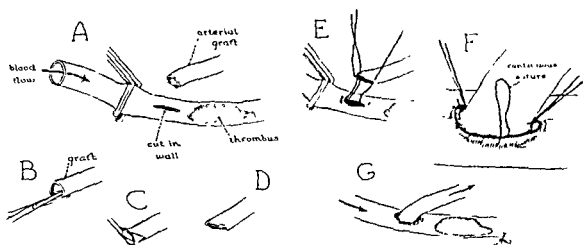


Fig 494—Technical steps in end to side anastomosis

with associated gangrene, we have been successful in saving, by grafting, 50% of the limbs which would otherwise have required major amputation. Fig 495 illustrates another use of the by-pass graft which was used to relieve impending gangrene of the right leg in a 72-year-old woman with widespread arteriosclerotic involvement of all the vessels.

The use of arterial grafts in the arteriosclerotic vessel is a palliative procedure. The original disease is not checked and usually

progresses with further occlusions and consequent late failure of the graft or the development of coronary or cerebral manifestations. However, from our follow-up data to the present time, these grafts can function for many years with relief of symptoms. The long-term fate of the plastic and homograft is not known, as it is too early for a final evaluation.

8. Amputations are the eventual result in a relatively large number of cases of arteriosclerosis obliterans due to irreversible ischemia

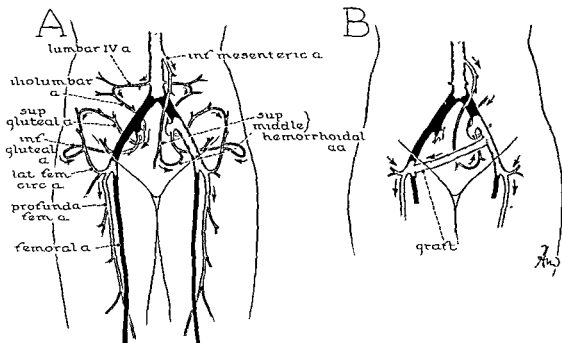


Fig 495—By-pass homograft inserted from left external iliac to right femoral artery to relieve gangrene of the right foot in an elderly, very arteriosclerotic woman (From Luke, J. C.: *Postgrad Med* 22: 10-20, 1957.)

PERIPHERAL VASCULAR DISEASES

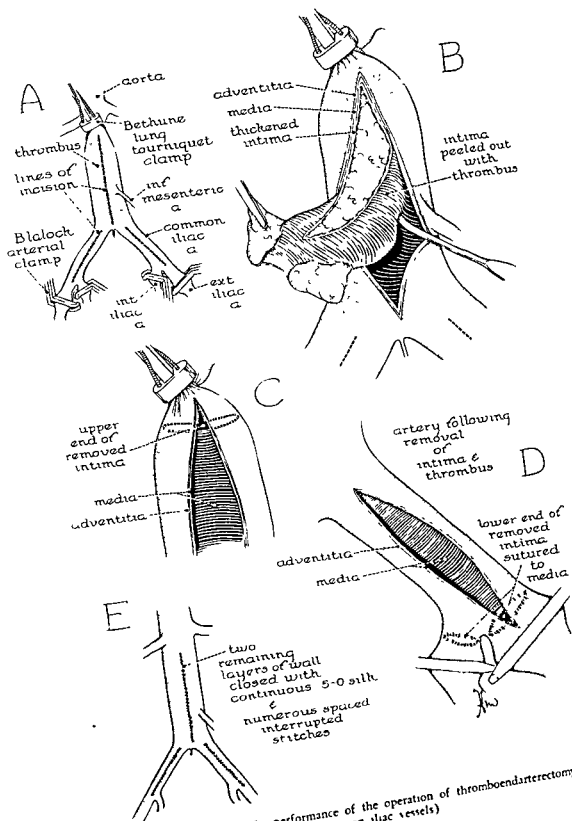


Fig 496—Technical steps in the performance of the operation of thromboendarterectomy (lower aorta and common iliac vessels)

and gangrene. The majority of such amputations are done at the lower thigh level because of the poor distal circulation which mitigates against healing of the operative wound. At the Royal Victoria Hospital from 1936-1946, 141 major amputations were performed of which 101 were done because of the results of arterial disease. Eighty-five were done through the mid or lower third of the thigh, 13 were end-bearing amputations at the level of the knee, and 3 were at the site of election. These figures should not indicate that minor amputations are not employed, healing will take place in many local amputations of toes where the circulation has been properly and previously assessed.

Aneurysms

An *arterial aneurysm* may be defined as a dilated segment of an artery resulting from trauma or disease. It is in direct communication with the main vessel and presents as a pulsating tumor. Two types of aneurysms are recognized, the *saccular* where the enlargement has occurred from one wall of the vessel and the *fusiform* where the entire arterial lumen and walls are diffusely enlarged. A third variety has been labelled as an aneurysm but is not truly so, being more a variety of blood vessel rupture. This is the so-called *dissecting* aneurysm.

Arterial aneurysms are lesions of major importance due to their continuing tendency to gradual enlargement and rupture. The syphilitic aneurysm has the worst prognosis with rupture occurring in about 75% of cases within 3 years from the onset of symptoms. Those of arteriosclerotic origin are more slowly progressive, but most of this variety will enlarge and rupture or thrombose within 10 years. However, many of these individuals will succumb from other manifestations of their disease before these catastrophes take place. Until recently, treatment of these lesions has been mainly conservative, but in the past few years the advances in vascular surgery have included surgical removal with a consequent likelihood of cure.

Etiology.—Prior to adequate antisyphilitic therapy, syphilis was the most frequent cause

of aneurysm formation. Now with this disease being detected early and adequately treated, this variety has shown a marked decrease. Syphilitic aneurysms occur chiefly in the aorta, especially in the aortic arch. They are generally saccular and arise from any area of the aortic arch but are commonest in the ascending portion. Enlargement may occur in any direction, resulting in the symptoms of hoarseness of the voice due to interference with the recurrent laryngeal nerves, tracheal tug due to pressure on the trachea, and pain, the result of mediastinal pressure and encroachment on neighboring bony structures. Bony erosion occurs when continued pressure on the thoracic cage exists and erosion and rupture outside the chest wall has been reported. Syphilitic aneurysms have been reported in lesser arteries, but the majority of these are arteriosclerotic in nature with a positive Wassermann reaction as an incidental finding.

Arteriosclerotic aneurysms are the commonest variety found in countries where proper diagnosis and control of syphilis is present. In this type, the aneurysm is chiefly fusiform in shape, and the peripheral vessels are more commonly involved than the aorta. The popliteal artery is the one chiefly affected, and a pulsating tumor in the popliteal space in an elderly individual is not uncommon. The author has seen three separate aneurysms in a 74-year-old female—one involving the left popliteal, a second involving the left superficial femoral, and a third the right common femoral artery. She was brought to hospital because of rupture of the left superficial femoral mass, which necessitated a high left thigh amputation. A common area for aneurysmal dilatation of the arteriosclerotic type is also in the lower aorta and iliac vessels, but the danger of rupture in this variety is not quite so great as in the peripheral types. Abdominal pain and the palpation of a pulsating mass in the abdomen or the finding of a partially calcified tumor on plain x-ray will lead to a correct diagnosis.

Aortography done on elderly arteriosclerotic patients will frequently reveal small unsuspected aneurysms of the lower aorta and iliac



Fig 497 —Angiocardiogram showing fusiform aneurysm of anterior wall of ascending arch of aorta



Fig 498 —Popliteal arteriosclerotic aneurysm with two proximal areas of arterial dilatation treated by resection and replacement by a venous autograft (Courtesy Dr D I C Bingham)

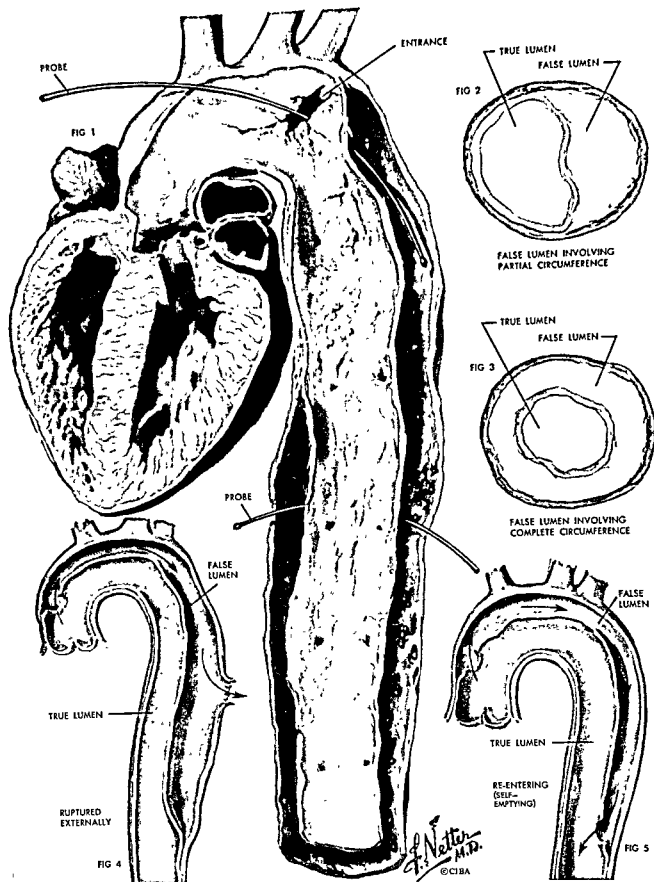


Plate 52.—Dissecting Aneurysm.

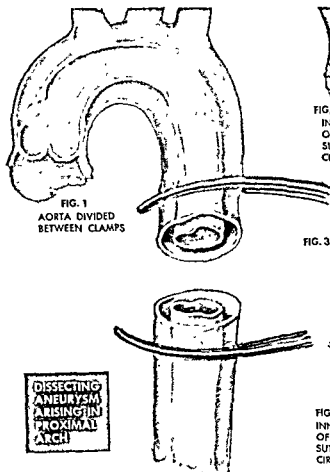


FIG. 1
AORTA DIVIDED
BETWEEN CLAMPS

DISSECTING
ANEURYSM
ARISING IN
PROXIMAL
ARCH



FIG. 2
INNER AND OUTER WALLS
OF PROXIMAL SEGMENT
SUTURED FOR PART OF
CIRCUMFERENCE;
WEDGE RECTED
FROM INNER WALL

FIG. 3



FIG. 4
INNER AND OUTER WALLS
OF DISTAL SEGMENT
SUTURED FOR COMPLETE
CIRCUMFERENCE

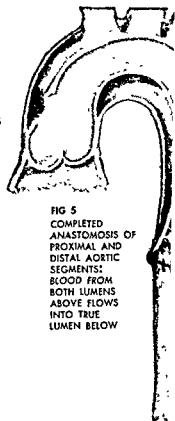


FIG. 5
COMPLETED
ANASTOMOSIS OF
PROXIMAL AND
DISTAL AORTIC
SEGMENTS;
BLOOD FROM
BOTH LUMENS
ABOVE FLOWS
INTO TRUE
LUMEN BELOW

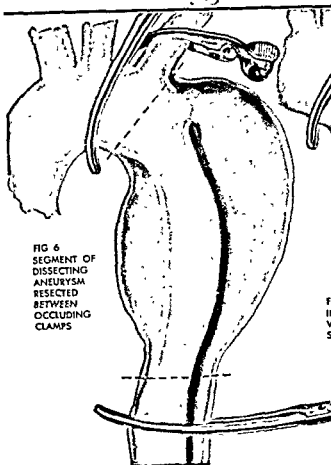


FIG. 6
SEGMENT OF
DISSECTING
ANEURYSM
RESECTED
BETWEEN
OCCLUDING
CLAMPS



FIG. 7

FIG. 8
INNER AND OUTER
WALLS IN DISTAL
SEGMENT APPROXIMATED



DISSECTING
ANEURYSM
ARISING IN
DISTAL ARCH

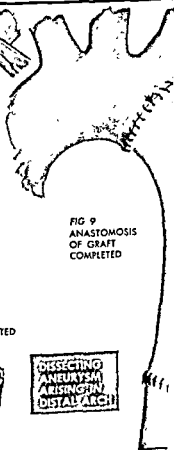


FIG. 9
ANASTOMOSIS
OF GRAFT
COMPLETED



FIG. 1
EXPOSURE AND
MOBILIZATION OF
ANEURYSM

LEFT RENAL VEIN
AND ARTERY

VENA CAVA

FIG 2
ANEURYSM BEING
EXCISED BETWEEN
OCCLUDING CLAMPS

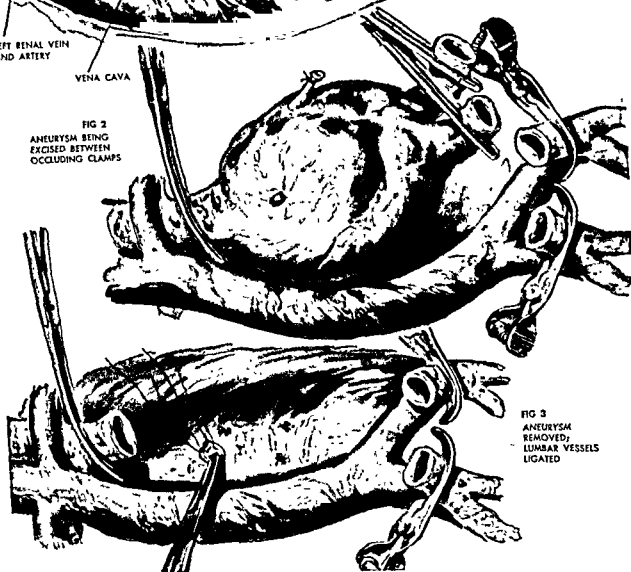


FIG 3
ANEURYSM
REMOVED;
LUMBAR VESSELS
LIGATED



FIG. 1
ANASTOMOSIS BEGUN
WITH TWO SUTURES ANCHORED
POSTERIORLY

FIG. 2
FIRST SUTURE BEING
BROUGHT ANTERIORLY

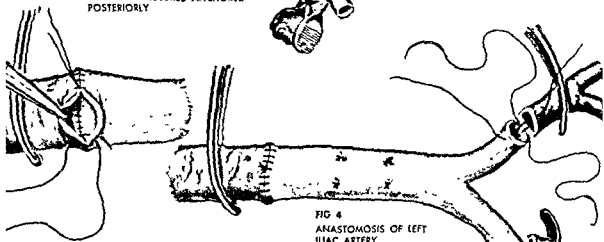
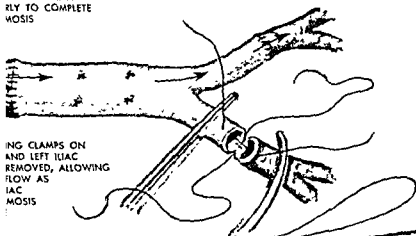


FIG. 4
ANASTOMOSIS OF LEFT
ILIAC ARTERY

SUTURE BEING BROUGHT
ANTERIORLY TO COMPLETE
ANASTOMOSIS



ING CLAMPS ON
AND LEFT ILIAC
REMOVED, ALLOWING
FLOW AS
TAC
MOSIS

FIG. 6
ANASTOMOSIS COMPLETED,
LIGAMENT OF TREITZ
AND PERITONEAL EDGES
BEING APPROXIMATED

vessels. A systolic bruit is usually present over the aneurysm.

Mycotic aneurysms are the result of weakening of the blood vessel wall, following continued blood stream infection. These occur chiefly in association with subacute bacterial endocarditis and result in small pea-sized or larger aneurysms, usually involving smaller arteries. This variety is rare but is also prone to rupture. They are rarely diagnosed prior to rupture except in the head where even their small size may produce sufficient neurologic signs and symptoms to necessitate investigation

with confirmation of the diagnosis by cerebral arteriograms.

Dissecting aneurysms result from a weakening of the intimal layer of the arch of the aorta usually just above the aortic valves. They occur on an arteriosclerotic basis, but there have been numerous reports of cases resulting from trauma, especially crushing injuries of the chest of the "steering-wheel" variety. The intimal tear is clean cut, and blood forces its way between the intima and media and may dissect in this plane along the entire length of the aorta to the bifurcation. When the



Fig 499—Arteriosclerotic fusiform aneurysm of the abdominal aorta and separate smaller aneurysm of left common iliac

blood penetrates through the media to the adventitia, rupture is imminent, as the latter coat will not contain the pressure for long. The usual termination is rupture into the thorax or into the abdominal cavity. Dissecting aneurysms have been reported to reconnect with the aortic lumen at the lower end, resulting in spontaneous cure but with the development of a "double-barrelled aorta." Just distal to the left subclavian is the common site of origin for the traumatic saccular type. The symptomatology of dissecting aneurysms is protean and depends on the location of the involved area. Their rupture (usually preceded by leakage) presents the picture of an acute thoracic or abdominal emergency. Diagnosis is difficult and is seldom made before death, but there are a few reports of operative survival from repair done in the thoracic aorta.

For a description of traumatic aneurysms see page 945

Treatment.—Conservative therapy of aneurysms is notoriously unsuccessful in attempting to halt the progress of this type of lesion. Syphilis requires treatment by high penicillin dosages, but otherwise little can be done. Surgical extirpation of these arterial weaknesses has been gaining momentum, and at present all varieties of aneurysms have a possibility of surgical cure.

Peripheral aneurysms such as the popliteal may be treated by proximal and distal ligation with excision or obliteration of the sac, provided that the collateral circulation is adequate. However, in the elderly arteriosclerotic patient this fact is difficult to assess, and lumbar sympathectomy is an additional safeguard in this respect. The treatment of choice is excision of the aneurysm with restoration of main vessel continuity by use of an autogenous vein graft or preserved homologous arterial graft.

Aneurysms of the proximal aorta are suitable for surgical excision only when saccular in type. The sac neck is clamped, and sutures are placed before the sac is excised. In this way the aortic continuity is not lost. The use of hypotensive drugs reduces the chances of cutting through and facilitates suture in the descending thoracic

by lateral aortorrhaphy, as mentioned above, or resection and replacement by a suitable homograft. Hypothermia (artificially reduced body temperature) is a great aid in preventing paraplegia, a complication of too long an interruption of the aortic flow. Aneurysms of the lower aorta below the renal vessels should be excised in the suitable case with replacement by a preserved homologous graft or a plastic (orlon) prosthesis. A sufficient number of these have now been done to indicate the safety of this procedure, the mortality being 10-15%. Previous methods of treatment, including internal wiring, external wrappings with dicycl cellophane, cutis grafts, and polyethylene, have not produced worthwhile results. If a dissecting aneurysm can be diagnosed clinically, surgical therapy is also effective in this type. The thoracic aorta is transected and the double barrel arranged to flow into a normal single barrel. (See Plate 53.)

Acute Arterial Occlusion

The two main causes of this sudden and frequently disastrous condition are arterial thrombosis and arterial embolism. The differentiation between them is often difficult, but such a differentiation should be made if possible because surgical removal of an impacted embolus is indicated in the early case of arterial embolism but surgery is rarely effective in acute thrombosis. However, only certain sites of lodgment of emboli need surgery, the most common being the bifurcation of the common femoral artery. Embolism of the upper limb rarely needs surgical removal as the collateral circulation is adequate when the blockage is distal to the axillary artery.

Both acute arterial thrombosis and embolism are of sudden onset, but the latter is the more acute. As a rule, acute pain is experienced in the limb with the rapid development of numbness and semiparesis depending on the degree of ischemia. When total ischemia is present, blanching of the limb is followed by a mottled cyanotic discoloration. The distinctive color changes above findings occur in the and consequently a delay, 1 to 2 hours in diagnosis is not unusual.



Fig 500—Gangrene of the foot subsequent to a neglected iliac embolus

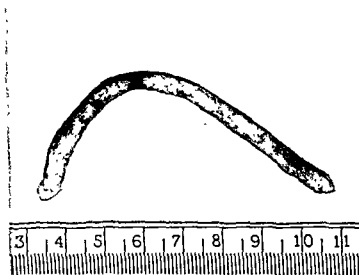


Fig 501—Embolus and tail thrombus removed by the technique of thromboendarterectomy in the case shown in Fig 500

treated, anesthesia develops in the distal part of the limb, gradually progressing centrally. The calf muscles become firm to the touch and tender, with loss of all contractile power, and the rigidity of the powerful ankle flexors gives a plantar flexion of the foot. The distal extremity is cold, and, on palpation, a zone of transition between warm viable tissues and cold ischemic ones can be found. Visual evidence of demarcation is also present in this area. Later frank gangrene of the ischemic parts develops.

The main points of differentiation between acute thrombosis and embolism are that embolism usually has a more sudden onset than

brain. More rarely, a portion of a mural thrombus can be dislodged from an atherosclerotic plaque in the aorta or from a proximal aneurysm. Such an embolus will progress distally and become arrested at a site, depending on the size of the embolus. Rapid narrowing in size of an artery occurs at the sites of major branches or bifurcation, and emboli become lodged at such points.

Treatment.—The diagnosis of acute arterial occlusion is urgently necessary as is also the determination of the cause of occlusion so that therapy can be instituted before irreversible ischemic changes occur in the limb. The results of treatment vary directly

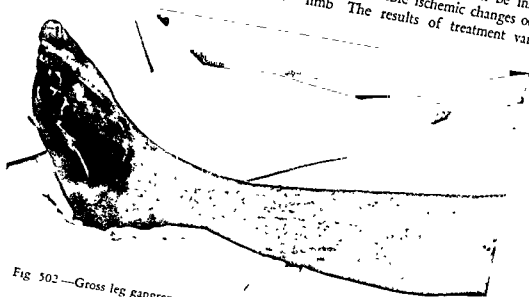


Fig. 502.—Gross leg gangrene, subsequent to neglected common femoral embolus

thrombosis. The effect of an embolus lodgment is like a bolt out of the blue versus that of thrombosis which is somewhat more gradual and takes several hours to develop. In the case of embolism, there must be some focus from which an embolus could have originated. The focus is most commonly a mural thrombus in a fibrillating heart (left atrium or atrial appendage) or a similar thrombus which has formed following a recent cardiac infarction from coronary thrombosis. Again, a portion of valve vegetation may be dislodged from a case of bacterial endocarditis. However, in bacterial endocarditis the emboli are small in size and block lesser arteries, producing the typical petechiae of this disease. Such a blockage therefore is not severe in effect except in the

with the length of time the occlusion exists and consequently it is imperative that the medical specialist and general practitioner who first see the patient are impressed with the need for speed. After 6 hours of ischemia there is a sharp decline in the number of legs that can be saved, and gangrene and subsequent amputation is the rule.

When first seen, and, particularly if less than 6 hours from onset, conservative therapy is indicated in both conditions. Priscoline is given in a 50 mg dose intravenously, the patient is transported to hospital, and anticoagulant therapy is begun. The leg is kept cool, and procaine block of the appropriate sympathetic chain is carried out to release the spasm of the remainder of the main vessel and the

collaterals which usually occurs as a result of the acute blockage. If signs of improvement in the distal circulation of the limb do not occur within an hour of this therapy, then embolectomy is indicated in those cases where this diagnosis is beyond question or where reasonable certainty exists that such a diagnosis is correct. The diagnosis of embolism must also include a correct level of lodgment of the embolus. Such an assessment is made by digital palpation of the vessel pulsations, the site of pain and paresthesia, the determination of where the level of ischemia commences, and

thrombus is found occluding a major artery, as, for example, the superficial femoral, and the surgeon is certain that the block is segmental, then the techniques described (pp. 956-962) are indicated. Acute segmental thrombosis producing severe distal ischemic effects, however, is a great rarity, and the usual finding is that the main vessel is thrombosed over a long area. Occasionally this long thrombus can be removed by opening a distal artery such as the popliteal or posterior tibial and flushing the thrombus upward to the femoral arteriotomy opening. The results of this technique are uncertain, especially in the advanced arteriosclerotic patient, and all too commonly major amputation is necessary. In summary, then, one can say that surgery is indicated in a diagnosis of arterial embolus but has little to offer in acute arterial thrombosis.

Certain site of lodgment of emboli or sites of acute thrombosis do not require surgery and will respond well to conservative therapy. The anterior and posterior tibial arteries when blocked will not result in severe ischemia, and the same can be said for the radial and ulnar arteries. Popliteal and brachial embolectomy is also seldom required.

ARTERIAL CONDITIONS SUBSEQUENT TO AUTONOMIC IMBALANCE

Raynaud's Disease and Raynaud's Phenomenon

Under this heading is included a group of closely allied conditions, the result of arteriolar spasm rather than organic occlusion. All the varieties resemble one another but in different degrees. Classic Raynaud's disease consists of attacks of arteriolar spasm, most commonly affecting the hands and rarely the feet. Women preponderate in the ratio of 4 or 5:1. Cold is a precipitating factor in the majority, but emotional stress may also bring on attacks, indicating the fact that the emotionally unstable individual is more prone to this disease than the stable, well-adjusted person. True Raynaud's is differentiated from Raynaud's phenomenon by the fact that the attacks are clear cut, relatively abrupt in origin, and consist of a period of intense vasoconstriction lasting

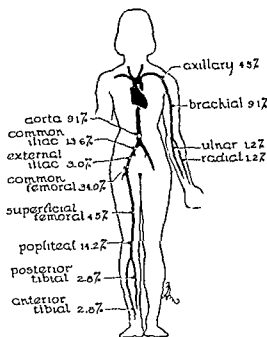


Fig 503.—Frequency of embolus lodgment

the use of the oscillometer. The sites of bifurcation of the main arteries should be kept in mind, as an embolus is most likely to plug an artery where an abrupt change in its lumen results from the sudden giving off of a large branch.

It is frequently difficult to distinguish clinically between acute embolism and thrombosis. An arteriogram can be readily performed and will be of great benefit in giving the correct diagnosis. In a dubious case, if conservative therapy has not shown improvement in the distal circulation, then surgery should be attempted in an effort to save the foot. If a

5-20 minutes, when the fingers are white and numb, followed by deep cyanosis and finally a burning erythema of the involved digits. In Raynaud's phenomenon, the same etiology is present but the effects are slightly different. Blanching is not so dramatic and the return to normal color is gradual and does not show the stage of cyanosis or erythema. There is the occasional case where the etiology appears to be due to the use of vibrating tools such as the pneumatic hammer.

In 1933 a cold precipitable globulin in the blood was first described. Since then this work has been confirmed on many occasions, and this variety of globulin has been called *cryoglobulin*. It appears in small quantities in many diseases but is especially marked in multiple myeloma. In other conditions in which no definite disease entity is present, its appearance is labelled as essential cryoglobulinemia, and it manifests itself in many ways, such as cold sensitivity, Raynaud's phenomenon, purpura, and livedo reticularis. Superficial gangrene of the extremities has been reported on four occasions. This abnormal protein precipitates in the blood vessels on exposure to cold, the precipitant acting as a foreign protein with resulting surrounding tissue reactions, thus explaining the many manifestations recorded. This also explains the localized areas of skin necrosis and ulceration, localized gangrene, and localized attacks of Raynaud's phenomenon encountered. Therefore cryoglobulinemia should be suspected in any bizarre case where the manifestations are multiple and resemble those of autonomic imbalance out of the ordinary.

Precipitation of an elastic translucent gel occurs when the patient's serum is exposed to cold and electrophoresis, indicating that cryoglobulin migrates to the gamma globulins. ACTH and cortisone apparently suppress this globulin and produce remission of symptoms either temporarily or, rarely, permanently.

Diagnosis.—The history in such a case is the most important point in establishing a diagnosis. Attacks of blanching of a finger or fingers, gradually increasing in severity and possibly gradually involving more and more of the hand, are characteristic. The presence of the "three-color phase" in such a case is pathognomonic of Raynaud's disease. The

preponderance in women, involvement of both upper extremities, relatively early onset (aged 20-40 years), and the precipitating factors of cold and emotional tension should give a clear diagnosis in the absence of any signs at the time of examination. During an attack (which frequently can be precipitated at will by exposure to cold), varying degrees of blanching are seen from dead white to a yellowish hue. The fingers feel numb and clumsy. The radial pulses are normal so far as palpable pulsations are concerned because the condition is at the arteriolar in type Raynaud's attacks can be initiated by the conditions which produce as cervical rib and scalenus anticus syndrome. Less intense attacks of Raynaud's phenomenon occur also in rheumatoid arthritis and Boeck's sarcoid. Such causes should be ruled out before a diagnosis of idiopathic Raynaud's disease is made.

In the occasional severe case with frequently repeated severe episodes of spasm, organic changes appear in the digital arteries. These consist of thickening of the arterial walls and occasionally thrombosis. In such a case, it is not uncommon for infections to occur about the finger tips, chiefly about the nails. These are chronic and painful and may result in local patches of superficial gangrene.

Scleroderma of the fingers and hand is occasionally seen as a complication to Raynaud's disease. The skin becomes atrophied and tightly stretched, and the deeper tissues are fibrosed even to the joints. Circulation is further decreased by this process, and organic vascular occlusion occurs with resultant trophic changes in the digits. Gangrene and chronic ulceration frequently supervene.

Treatment.—It must be kept in mind that the great majority of cases of Raynaud's disease and Raynaud's phenomenon are mild and therefore the individuals never seek medical advice. They have discovered for themselves that if they protect their hands from cold, handling cold objects, they will be relatively comfortable. The removal to a warm climate in the winter may be necessary. Emotional states are harder to control, but reassurance and careful medical guidance are of benefit. Medical management also includes vasodilator

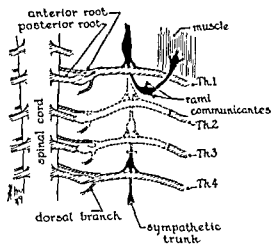


Fig 504—Diagrammatic illustration of technique of preganglionic upper dorsal sympathectomy

drugs and estrogen therapy because of its vasodilating effects. In the severer cases and especially where organic changes are occurring in the finger tips, surgical treatment is indicated. Preganglionic section of the sympathetic fibers to the arm gives spectacular initial results

According to Smithwick's technique, the upper dorsal sympathetic chain is sectioned below the 3rd thoracic ganglion and the white and gray rami to T2-3 are sectioned, the former being removed intraspinally by intraspinal section of the somatic nerves T2-3. The proximal portion of the sympathetic chain is then removed as far as possible from the field by suture of its free distal end to the muscles of the back.

Results from this operation unfortunately are not permanent, and about 60-70% of cases tend to recur to some degree after 6 months. The reason for this recurrence is not clear; local arterial sensitization to circulating Adrenalin has been cited as a factor. Sensitization of the arterioles to cold or regeneration of the interrupted sympathetic pathways has also been blamed for recurrence

More recently a return to the older surgical technique of removal of the stellate and 1st and 2nd dorsal ganglia has become popular. It has been shown that better long-term sympathetic denervation of the upper limb can be achieved by this method. The only disadvantage to this operation is the production of a Horner's syndrome

Acrocyanosis

Acrocyanosis is a vascular imbalance resulting in a primary loop with peripheral cyanosis. The etiology is obscure but is associated with the same disturbance. The difference, however, is that the hands and/or feet are deep cyanotic blue. The fingers are reddened somewhat in the deeper blue in cold. This will result in the fingers becoming maroon in color. It is more involved than Raynaud's disease and most frequently occurs in women.

The hands and feet are cold and show the characteristic cyanosis. The patient usually complaining of numbness and other symptoms.

Acrocyanosis, with its characteristic blue color of the veins, shows a reversal of the normal color change into the red when the patient is on a hot bath. The symptoms are most frequent in women.



veins in-

Investigation of a proper assessment of the affected area, termination of the procedure occurs. Upon question of treatment tests have been de-

Fig 505
on 1

phyton infection may be troublesome due to the excessive sweating of the feet

Treatment.—Treatment is unsatisfactory, and the condition tends to persist although decreasing somewhat with age. Lumbar sympathectomy may be indicated in the severe cases, especially where marked sweating and Epi-dermophyton infection are associated.

Livedo Reticularis

This variety of neurovascular imbalance is characterized by patches of cyanotic reddish mottling on the feet and legs, extending to the knee. The hands are also frequently involved. The vessel changes are similar to acrocyanosis but are patchy and more widespread. The patient may complain only of the appearance, but coldness, clamminess, puffiness, and some numbness may be mentioned.

The majority of patients do not require any specific therapy beyond reassurance and an explanation of their condition. In the rare case where symptoms are more severe, lumbar sympathectomy will provide relief, although the bluish, mottled areas merely turn to pink. However, the coldness, wetness, and puffiness are relieved.

VASOMOTOR CHANGES RESULTING FROM TRAUMA AND INFECTIONS

When tissue is damaged by trauma or an infective process, the resultant healing is dependent to some extent on a normal nervous control of the reparative process. In the occasional rare case there appears to be an abnormal nervous control which may show a preponderance of the vasoconstrictive (sympathetic) tone or, rarely, vasodilator preponderance. The degree of such abnormality varies greatly, and the lesser degrees are transient and pass off before serious effects are noticed. However, in the occasional case, a chronic state of either vasoconstriction or vasodilatation of the vascular bed results. Such a continued effect leads to an abnormal process which has a widespread result in the limb and shows, in the spastic type, clinical evidence of vasoconstriction and coolness of the limb, increased sweating, pain, and vague, poorly defined sensory changes in the limb. The peripheral pulses appear to be reduced, edema

may be present, and the joints become stiff and painful. The vasodilatation type shows a limb which is warm, painful, dry, and edematous, and which later may show a typical spotty decalcification of bone known as Sudeck's atrophy. The pain in both types is typically causalgic in nature, and a case may begin by showing the vasodilatation phase which later (2-3 months) changes over to the vasoconstriction type.

Trauma of a mild degree, such as that resulting in a sprain, may be the initiating factor, and the author has seen cases resulting from burns, minor fractures, and even minor surgical wounds such as an incision for drainage of an abscess. Infections in the limb, such as cellulitis or phlebitis, may be the originating cause. This whole condition is somewhat vague and poorly understood, and the exact nerve pathways taken by these abnormal reflexes is not clearly known. Why one person will be affected and not another from identical trauma is not known, but it appears as though this condition is more likely in those persons who show instability of the nervous system, especially the autonomic portion.

Treatment.—The fact that a neurovascular imbalance is present is utilized in treatment, and interruption of some portion of the reflex arc is logical therapy. The sympathetic supply to the limb is the efferent arc to this reflex, and it is simple to interrupt. Procaine block of the appropriate sympathetic chain will often give dramatic temporary relief of symptoms which lasts for a progressively increasing length of time after each block, and after the second or third the curative effect may be permanent. If a temporary effect only is noted after each block, then a sympathectomy will give a permanent effect. In the later cases where osteoporosis is severe, edema has been present for a long time, and joint stiffness is present, long-continued physiotherapy is a necessity in producing recovery.

DISEASES OF VEINS

Varicose Veins

A varicose vein may be defined as an abnormally dilated vein, and, for practical purposes, the legs and lower abdomen are almost solely involved. These veins show an increase

an caliber, sacculations due to patchy thinning of segments of the vein wall, elongation and tortuosity, and fibrous thickening of other portions of the wall. A loss of valvular function takes place, and at times calcification of the vein wall may occur.

Etiology.—There are two main types of varicose veins from the causative standpoint. The *primary* ones develop in early adult life and probably arise on the basis of a congenital defect in the structure of the vein. This structural defect is hereditary, as a tendency to varicose veins is often seen in certain families. Such a defect is likely a weakness of the wall and valves of the veins. Also a decreased initial number of valves may be present, the normal number being 6-8 in a competently functioning great saphenous vein. This congenital and hereditary background is probably the reason that pregnancy will cause the appearance of varicose veins in one woman and not in another, or why one shopgirl who stands all day will develop varicose veins, whereas another will not.

Secondary varicose veins are those that develop following increased venous pressure acting upon them from the main venous trunks. These follow a previous deep phlebitis where increased venous pressure can occur in the damaged deep vein from direct transmission of increased intra-abdominal pressure. This increased pressure is transmitted to the superficial veins and structural defects in the valves and vein walls occur with the development of true varicose veins. Another example of this mechanism is the development of hemorrhoids and esophageal varices in portal hypertension. The rapid development of varicose veins in a middle-aged individual should raise the possibility of the secondary type and can arise from large intra-abdominal tumors, ascites, and cardiac failure.

Signs and Symptoms.—In some cases, well-developed varicose veins result in no symptoms whatsoever, and patients present themselves for treatment from the cosmetic standpoint. However, the usual complaints are those of tiredness, heaviness, fullness, and aching of the leg after prolonged standing and therefore worse toward the close of the day. Menstruation usually aggravates the complaints. Symptoms of uncomplicated varicose veins should

be completely relieved by recumbency, with the possible exception of nocturnal muscle cramps. To give the above symptoms, the veins should be well developed and should show a positive Trendelenburg test (a reversal of flow in the vein when the patient assumes the upright position). Beware of falling into the common error of blaming leg symptoms on a few varicose veins, especially where the symptom complex is at variance with that mentioned above!



Fig 506.—Extensive primary varicose veins involving both legs

An essential part of the investigation of a case of varicose veins is the proper assessment of the degree of incompetency of the affected veins and valves, and a determination of the level at which the incompetence occurs. Upon this knowledge the entire question of treatment depends. A number of tests have been designed to answer this point, and all are modifications of the original Brodie-Trendelenburg test. This test is done by having the patient in a horizontal position, with the affected leg lifted. The blood drains out of the superficial

veins, assisted by massage centrally with the hand. With the veins collapsed, a rubber tourniquet is placed around the upper thigh sufficiently taut to compress the superficial veins only. The patient stands with the tourniquet on, and the veins below the tourniquet should remain collapsed up to 20 seconds if the communicating veins and small saphenous vein distal to the tourniquet are competent. Release of the tourniquet will now give rapid filling of the distal varices from above when incompetence of the valves of the saphenous vein only is present. If with the tourniquet on the thigh and the patient standing, the distal

varices fill immediately, this indicates an incompetent valvular mechanism between the deep and superficial venous systems below the level of the tourniquet, and the small saphenous vein can be included in this group of communicating veins. The site of the incompetent communicating vein or veins can be determined by repetition of the test using multiple tourniquets placed at different levels or by moving the single tourniquet gradually more distal until a level is found where the distal varices do not immediately fill but only fill when the tourniquet is released. At this level one is therefore just below the point of the most distal incompetent communicating vein.

Treatment.—

Primary Veins.—Where small dilated veins are present with no symptoms and the patient shows no reverse flow in the veins on performing the Brodie-Trendelenburg test, then local injections of a sclerosing fluid are indicated if the patient wishes treatment for cosmetic reasons. When reverse flow occurs in the great saphenous vein alone and there are no incompetent communicating veins, a choice of surgical procedures can be employed. Ligation of the great saphenous vein as close to the femoral junction as possible will interrupt the incompetent vein at its source. The vein can then be obliterated by the injection of sclerosing solutions either at time of operation or subsequently. These solutions must be used with care because of several possible complications. Allergic manifestations are not too rare, especially in those persons who have an allergic tendency. Preoperative testing for sensitivity should be done in all cases before injection. Neglect of this precaution has resulted in many cases of sudden death from anaphylactic shock. Another danger in the use of sclerosing fluids is the fact that when injected into a superficial vein a certain proportion of the fluid inevitably gets into the deep veins. This may occasionally result in deep venous thrombosis with crippling damage to the leg and the occasional case of pulmonary embolism.

Stripping of the great saphenous vein is undoubtedly a better procedure in all cases of incompetent varicose veins, as removal of the diseased vein results in a reduced tendency

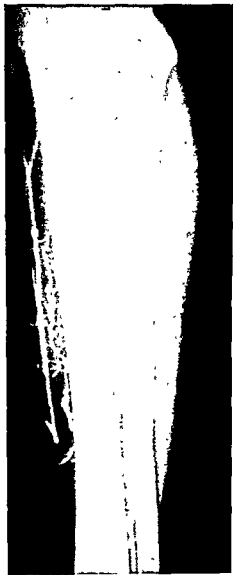


Fig. 507.—Pronade venogram in a normal individual. Although patient is erect, the contrast medium ascends in the narrow, straight-walled veins.

to recurrence. Stripping should be the only surgical procedure in those cases that show evidence of incompetent communicating veins because of the high rate of recurrence in cases when the ligation and injection technique is employed.

In about 20% of cases the small saphenous vein shows incompetent valves, and this vein will also need surgical attention. It should be ligated as high in the popliteal fossa as possible and then distally injected or stripped according to the preference of the surgeon.

Varicose veins are not diseased tissue that can be obliterated or removed with any degree of permanency. The student should realize that he is dealing with a gradually progressive condition involving all the superficial (and possibly to some extent the deep) veins of the legs and that removal of one set of veins will not prevent enlargement of remaining ones. Some evidence of new varicose veins following surgery occurs in most cases, but the degree of the recurrence will depend to a great extent on the excellence of the job done

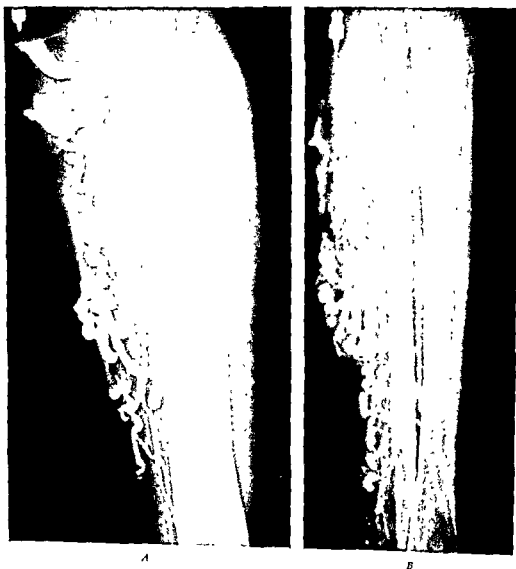


Fig 508—*A*, Retrograde venogram showing the contrast medium descending rapidly in widely dilated varicose veins
B, Lateral view taken 25 seconds later. The contrast medium is now escaping into the deep veins

flow on standing, and therefore a positive Trendelenburg test, then these damaged veins only aggravate the venous stasis and should be corrected.

The method of correction should be by the stripping technique as the use of sclerosing fluids is dangerous due to the already damaged deep circulation. The patient should be warned prior to the operation that this procedure is not totally curative but will improve to some degree the venous stasis in the damaged leg. The instructions necessary for the care of the postphlebotic leg must still be carried out because of the continuing deep vein damage

(See p. 989.) The use of the elastic stocking as outlined will in many cases obviate the need for the surgical correction of these secondary varicose veins.

Complications of Varicose Veins.—Venous stasis, whether from incompetent superficial varicose veins or from deep veins following a previous phlebitis, results in the occurrence of several abnormal physiologic states. Increased venous pressures are present in the upright position and particularly after straining, with elevations up to 400 mm. of water from a normal of 50-100. This allows stasis of the capillary loop, resulting in cyanosis, and



Fig. 510

Fig. 510—Retrograde venogram of common femoral vein which has been occluded centrally by digital pressure. Valves show excellent competency (patient recumbent).

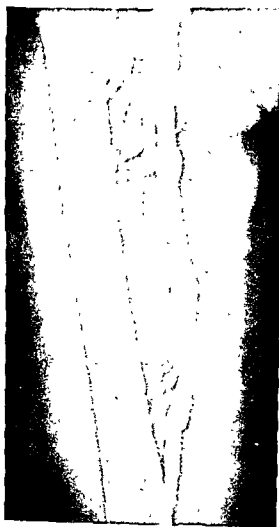


Fig. 511.

Fig. 511—Retrograde venogram of superficial femoral vein. Valves which are located at the bulbous dilatations show marked incompetence.



Fig 512—Typical varicose ulcer

also anoxemia of the endothelium allowing increased permeability and edema. This edema together with some decrease in O_2 and increased CO_2 tensions, also occasionally increased NPN values, results in tissue devitalization and paves the way for the complications.

Varicose Ulcer—When ulceration is present in association with varicose veins, these varicosities must be of a relatively severe degree and show valvular incompetence with reversal of flow on standing; otherwise such an ulcer is not a true varicose one. Varicose ulceration is usually preceded by the preliminary changes of chronic venous stasis. This results in brownish skin pigmentation, the result of the deposition of hemosiderin subsequent to the breakdown of extravasated red cells, also thinning of the skin with fibrosis, and devitalization of the underlying fatty tissue. Chronic edema is frequently present, aiding in the development of these changes and encouraging the presence of chronic low-grade periphlebotic infection and consequent induration.

With such devitalizing influences present, it is not uncommon, spontaneously or following a slight trauma, that an ulcer develops which persists as long as the chronic venous stasis is allowed to remain. This ulcer is most commonly located just above the medial malleolus but may be anywhere in the lower half of the leg if trauma has been the initiating cause. Each time the ulcer recurs following inadequate treatment, further fibrosis is added to the former ulcer area, rendering the tissues more devitalized and so more liable to further ulceration.

Treatment consists of correcting the underlying cause of the venous stasis by removal of the varicose veins. The ulcer is treated by some type of compression bandage to remove edema. The best dressing is still Unna's paste boot. Bed rest, hot dressings, and the use of antibiotic drugs will be necessary in the acutely inflamed ulcers.



Fig 513—Hypertensive ulcer showing the indolence and surrounding skin devitalization associated with skin infarction resulting from hypertensive cardiovascular disease

To many practitioners any chronic leg ulcer is diagnosed as being varicose in origin. This is incorrect in many instances and leads to improper treatment. Many saphenous ligations or strippings have thus been done unnecessarily. The problem of the chronic leg ulcer is badly managed by the profession as a whole, with undue emphasis being placed on local treatment and insufficient thought as to the etiology.

The following list indicates, in order of frequency, the different causes of chronic leg ulcers, and these causes should be kept in mind in determining the correct etiology so that the appropriate therapy may be instituted:

- 1 Varicose ulcer
- 2 Postphlebotic ulcer
- 3 Ischemic (hypertensive) ulcer
- 4 Syphilis (tertiary)
- 5 Tuberculosis (erythema induratum)
- 6 Squamous cell carcinoma
7. Associated with splenic diseases (splenic anemia, Gaucher's, etc.)
- 8 Chronic pernio
9. Blastomycosis and other allied conditions

Varicose Eczema.—Another of the manifestations of chronic venous stasis is that of eczema. This usually consists of a patchy involvement of the medial ankle region but may be severe enough to involve most of the lower leg. It is manifest by a red, exuding, scaly involvement of the skin, and the patient complains of edema, burning, and itching of the affected part. Again, it should be pointed out that, to be true varicose eczema, incompetent varicose veins must be present. A previous deep phlebitis, diabetes, or sensitivity reaction should be ruled out. Treatment consists of support to the leg, the same as for varicose ulceration, plus the correction of the incompetent varicose veins.

Superficial Phlebitis.—Varicose veins are more prone to the development of phlebitis than normally functioning ones because of the relative venous stasis present in addition to the fact that their exposed position renders them more liable to trauma. Such a phlebitis is diagnosed by the signs and symptoms of pain, redness, soreness, and hardness in varicosities previously soft and painless.

Minor degrees of this complication are readily controlled by a pressure bandage such

as Unna's paste boot or elastic adhesive dressing. There is no need for the patient to be immobilized. Major degrees involving a large stretch of vein, especially where extension toward the saphenofemoral junction is taking place, are best treated by high saphenous ligation at the saphenofemoral junction. This has the merit of preventing the rare case where the saphenous thrombus extends into the femoral vein with therefore possible embolus formation; also it corrects the basic fault in the original varicose veins. The inflammatory thrombus present obviates the need for the use of sclerosing fluid, and the same ultimate effects of obliteration of the varices take place. This method of therapy allows the patient to be ambulatory, corrects the underlying venous pathology, and so prevents recurrent attacks. A pressure dressing is applied to the leg after operation to reduce the pain, stiffness, and edema resulting from the phlebitis.

Venous Thrombosis

Thrombosis of blood in the venous tree is a complication fraught with danger to the patient's life and may result in varying degrees of mild to severe disablement. There are three principal factors which result in venous clotting:

1. Increased coagulability of the blood which may follow trauma or be secondary to anemia or polycythemia or may be present for some undetermined reason.
2. Slowing of venous blood flow which may occur secondary to heart disease, dehydration, or prolonged immobility.
3. Damage to the intima of the vein resulting from trauma or infection.

The degree, extent, and type of the venous thrombosis vary according to the degree of associated inflammation and may vary from the bland, quiet phlebothrombosis to the severe septic thrombophlebitis.

Bland Thrombosis (Phlebothrombosis)

When the inflammatory element is mild and the intimal damage is minimal, blood clotting in the vein will take the form of a soft, loosely attached clot which gradually propagates centrally. Such a process most frequently originates in the veins of the lower extremities be-

cause in these veins the above-mentioned factors predisposing to thrombosis are most frequently met. Those of the sole of the foot, the calf, and the adductor region of the thigh are most commonly originally involved. Propagation of the clot from these regions may take place into the main venous channels of the leg and progress centrally even as far as the vena cava.

The thrombus originates as a collection of platelets which adheres to the intimal endothelium at some area where intimal damage from whatever cause has taken place. Leukocytes and fibrin adhere usually in layers, and a white thrombus is formed. Further clotting is mixed with red cells, and a red thrombus is formed which progresses centrally with little or no attachment to the vein wall. Such a loosely adherent or freely floating clot attached only distally is known as a "tail" thrombus and consequently is easily detached to act as an embolus. Such a fragment finds lodgment in the lung to produce the typical pulmonary embolus, and the amount of pulmonary artery occluded will depend directly on the size of the thrombus detached, those from a small distal vein will produce minor emboli and those from a major vein may block the origin of the pulmonary artery, producing sudden death.

Bland thrombosis may originally be associated with only a mild degree of inflammation of the vein wall but such an inflammatory involvement tends to increase in some cases and consequently an original case of phlebothrombosis may progress to a full-blown thrombophlebitis.

Signs and Symptoms.—It must be emphasized that this condition can occur both in medical and surgical diseases but probably is more common after surgery and childbirth. Symptoms may be completely absent, the first indication of the lesion being that of a pulmonary embolus. Many of these emboli are massive and produce the tragic occurrence of sudden death during an apparently uncomplicated convalescence. Signs and symptoms, however, usually occur first in the leg where the majority (95%) of such thromboses originate. Some aching in the foot, the calf, popliteal region or thigh will be noticed by the patient, and the leg will

have the sensation of fullness and stiffness. Such symptoms usually occur or are aggravated when in the upright position (Luke's sign.) Mild fever is present and the pulse is usually slightly elevated. Tenderness in the sole of the foot, the calf, popliteal region, or medial thigh may be found, and slightly increased warmth of these parts is usually present. A sense of fullness of the muscle mass of the calf is a very constant finding. A feeling of tightness or pain in the calf is produced in the majority of cases by forced dorsiflexion of the foot with the knee extended (Homan's sign). Mild edema of the



Fig. 514.—Phlebogram in a case of phlebothrombosis. The contrast medium flows along the sides of the loosely attached clot, forming a "mantle shadow." This patient had suffered a pulmonary embolism.

ankle and lower leg is present, and the foot may show some cyanosis on dependency. The sedimentation rate is elevated.

It cannot be emphasized too often that the condition of bland thrombosis and that of thrombophlebitis are part of the same process and differ only in the degrees of in-

flammatory involvement. Bland thrombosis has been described as an entity chiefly because of its minimal signs and symptoms and its great tendency to embolize (30% of cases). Consequently, with the possibility of such a serious complication, every effort should be made to prevent this development or to recognize it in its early stages. The condition should be constantly kept in mind and looked for in every postoperative patient, and a serious view taken of even the mildest leg complaint in any patient so that early therapy can be instituted and a possible pulmonary embolus averted.



Fig. 515.—Pronade venogram showing occlusion of a major branch of the popliteal vein by phlebotrombosis. Only a small stub remains patent.

Thrombophlebitis

As mentioned above, thrombophlebitis presents the picture of venous clotting associated with marked inflammatory involvement of the vein and, to some degree, the perivenous structures. The same etiologic factors pertain as in bland thrombosis, consequently it may occur following childbirth or a surgical operation, especially where infection is associated.

The incidence is about 2% following surgical operations and is highest in kidney and bladder cases, major gynecology, or such abdominal lesions as a perforated appendix. As in bland thrombosis, it occurs between the 5th-15th postoperative or post-partum day and takes place in the leg veins in the great majority of cases, though primary involvement of the pelvic veins may occur. The veins of the portal system occasionally are affected from some suppurative lesion in the abdomen, but this condition appears to be much less common than formerly due to the improvement in surgical technique and the use of antibiotics. Certain medical diseases show a relatively high incidence of thrombophlebitis of the leg, such as typhoid fever, pneumonia, Buerger's disease, and ulcerative colitis. Occasionally thrombophlebitis is seen where no definite predisposing factor can be determined, but in such cases some infectious process such as an upper respiratory infection has usually preceded the onset. When such a spontaneous thrombophlebitis exists, the possibility of an undiagnosed malignancy in the abdomen should be considered. It has been shown that cancer of the body and tail of the pancreas has associated episodes of leg thrombophlebitis in about 30% of cases.

Signs and Symptoms.—In the acute form, the patient will complain of a relatively sudden onset of a severe ache in the involved leg, variously localized in the calf, popliteal area, or thigh. The temperature climbs to 101°-103° F. Some cyanosis of the leg, even when horizontal, is usually present, and severe edema soon appears involving the lower leg only when the process starts distally or involving the whole leg and buttock when the femoral and iliac veins are affected. Acute tenderness is present over the involved area; the part is hot and full to the touch and pain results from muscular movement.

All gradations of thrombophlebitis occur from the fulminating type of acute massive venous occlusion to the lesser degrees resembling bland thrombosis. Luke's sign also applies to thrombophlebitis and is an early diagnostic indication. A fulminating variety of thrombophlebitis is occasionally seen where the degree of involvement is sudden and severe and is associated with a major extent of arterial

vasospasm to such a degree that the picture of acute arterial thrombosis or arterial embolism may be simulated. However, the leg edema and cyanosis will usually indicate the true picture. The arterial spasm may be of such a degree that true gangrene develops, and such a lesion demands energetic treatment to obviate such a possibility. This consists of high elevation of the leg to at least 45 degrees, frequent active or passive exercises of the involved leg, antispasmodic drugs and the rapid institution of anticoagulant therapy.

Pulmonary Embolism

As previously indicated, the origin of most pulmonary emboli is from the leg or, rarely, the pelvic veins and is usually the sequel to a case of bland thrombosis because of the looseness of the attachment of the clot consequent to the minimal inflammatory involvement of the vein wall. In thrombophlebitis where inflammation of the vein wall is extensive, embolus production is uncommon (5%). Detachment of a fragment of clot occurs where a large tributary connects with the blocked main vein, the flow of blood from this tributary, being sufficient to break off the "tail" thrombus. Sudden increases in intra-abdominal and consequently intravenous pressure may also produce the same result; this is the reason for the sudden death of patients in the act of using the bedpan or getting up for the first time.

The degree of pulmonary artery blockage depends directly on the size of the embolus, and the size of the embolus depends on the caliber of the vein from which it originates. A piece of clot from the veins of the lower leg will give a minor degree of pulmonary infarction, whereas one from the common femoral or iliac may produce sudden death. Another factor which contributes to the degree of symptoms in pulmonary embolism is spasm of the bronchial tree and pulmonary vessels incident to the sudden lodgment of an embolus, this being similar to the effect of a peripheral arterial embolus. Another similar result is the fact that thrombosis can progress distally from the site of occlusion in pulmonary artery and give a fatal termination to a patient who survived the catastrophe. The rapid drop in h

associated with pulmonary infarction of major degree is a further cause of death in this condition.

Signs and Symptoms.—Any person suffering from any inflammatory disease, after an operation or childbirth, or who has been confined to bed for any period of time and who complains of a sudden pain in the chest should be suspected of having pulmonary embolism. Minor emboli may be manifested only by a "stitch in the chest" and be so mild that the initial one is not recognized. Pathognomonic is the triad of a sudden pain in the chest aggravated by respiration, breathlessness, and later, hemoptysis. The greater the amount of lung infarcted the more marked are the symptoms. Pallor, cyanosis, collapse, sweating, loss of consciousness, lowered blood pressure, and rapid thready pulse are the signs of a major embolus. Differentiation of this picture from that of an acute coronary occlusion may be difficult, and many patients have died of so-called acute heart attack when the real reason was an embolus. The discovery of the site of the peripheral thrombus, the later spitting of blood, the development of a friction rub, and the x-ray appearance of a dense area in one lung will give the differentiation.

Treatment of Venous Thrombosis and Pulmonary Embolism

Two relatively recent methods have been added in the treatment of this condition with marked improvement in the morbidity and mortality results. These are the employment of the anticoagulant drugs and the judicious use of vein ligation. The aim of present-day treatment is to make a diagnosis at the earliest possible moment and then institute active therapy to prevent extension of the process centrally. It is the emboli from the larger veins which kill or, in thrombophlebitis, it is the involvement of the major veins such as the common femoral or iliacs that produce the crippling sequelae in subsequent years of chronic swelling of the leg, eczema, ulceration (postphlebotic leg).

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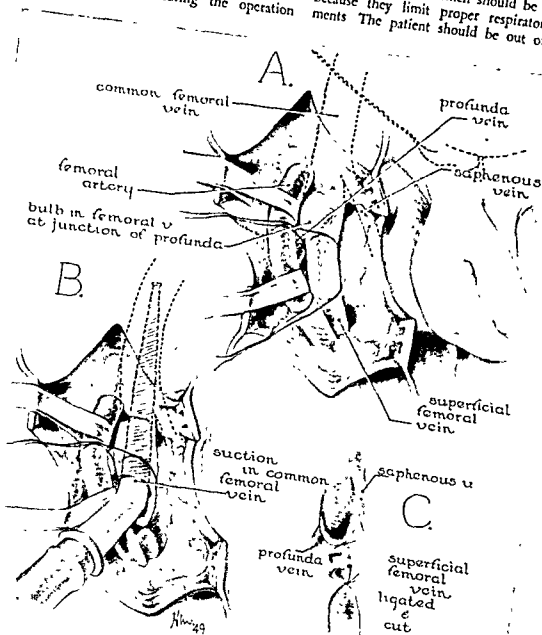


Fig 516—Surgical technique for removal of thrombus in the common femoral vein

reduces the amount of tissue damage and consequently the amount of thrombokinase liberated. Active movements of the legs should be carried out many times daily according to a definite schedule, and turning from side to

early as possible and encouraged to walk and not just sit in a chair. In a case of an elderly patient where extensive surgery has been done or where an increased clotting tendency is suspected by a history of previous episodes

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However, the best method of treatment is prophylaxis, and prevention of this complication can be attained in great measure

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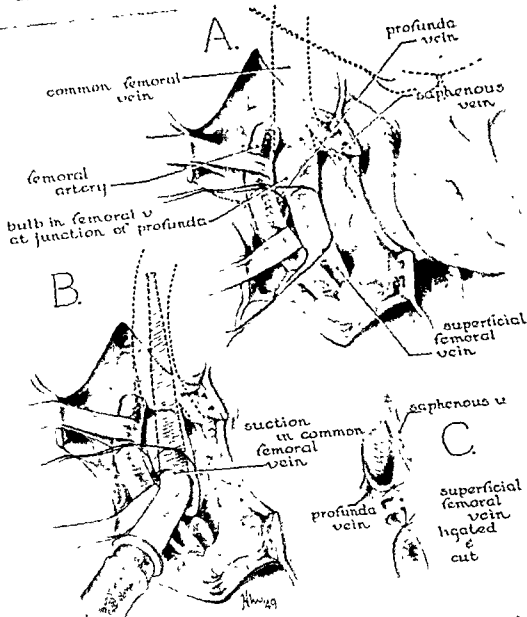


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DISEASES OF VEINS

by careful attention to detail in the patient before and after operation. Anemia, dehydration, and alterations in the albumin-globulin ratio should be corrected prior to operation. Gentleness during the operation

side should be done hourly. Fowler's position should not be used, and tight binders or dressing on the abdomen should be discarded because they limit proper respiratory movements. The patient should be out of bed as

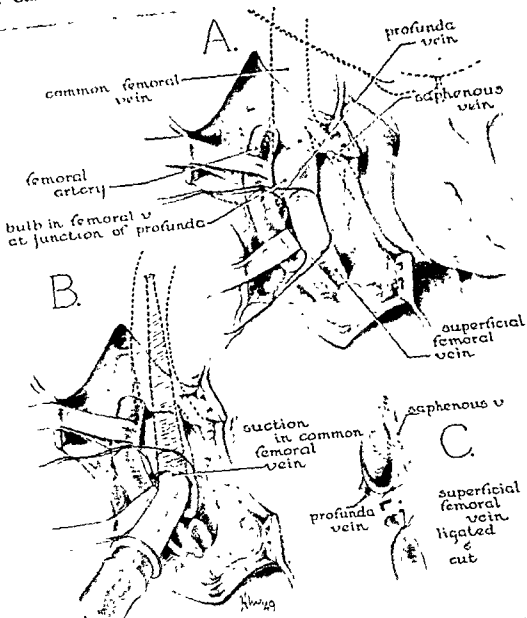


Fig 516—Surgical technique for removal of thrombus in the common femoral vein

reduces the amount of tissue damage and consequently the amount of thrombokinase liberated. Active movements of the legs should be carried out many times daily according to a definite schedule, and turning from side to

early as possible and encouraged to walk and not just sit in a chair. In a case of an elderly patient where extensive surgery has been done or where an increased clotting tendency is suspected by a history of previous episodes

of thrombosis, anticoagulant drugs should be used as a prophylactic measure. The legs should be inspected and palpated daily in every case of major abdominal surgery and treatment instituted at the first suspicion of venous thrombosis. Prophylactic ligation of both superficial femoral veins just distal to the profunda branch has been proposed by some groups prior to major abdominal surgery but the results achieved have not been impressive and do not justify the additional surgery.

The use of suitable vein ligation in the treatment of phlebothrombosis and thrombophlebitis has recently been through a phase of popularity but is now on the wane. The rationale of this procedure was to trap the thrombus distally by a suitably placed proximal vein ligation or, if thrombus was found in the vein when the superficial or common femoral vein was opened, then to suck out the central extension of the thrombus. The ideal site for such a vein exploration and ligation is the superficial femoral just distal to the profunda branch. Ligation at this level does not lead to leg edema or postphlebotic leg changes, and the common femoral up to the common iliac can be cleared by a suction tip.

This reasoning is good in theory but is not so sound in practice. On many occasions the author has found on opening the superficial or common femoral veins that clot is present which cannot be removed because of its internal adherence. In such a case clot reformation is inevitable. Other cases have occurred where a thrombus has formed central to the site of ligation, and resulted in embolism. Again it is frequently impossible to tell from which leg an embolus has originated; consequently a bilateral operation is necessary. Or again it is impossible to say that the thrombus has not originated in the pelvic veins.

At the present time it is the author's opinion that there are only two indications for ligation in venous thrombosis. The first is when the patient continues to throw multiple emboli to the lungs despite treatment by adequate anticoagulant therapy. His life is endangered and further steps must be taken. In this case, femoral ligations are usually inadequate, and ligation of the vena cava below the renal veins is the site of choice.

The second indication is the ligation of the superficial femoral vein just distal to the profunda in the early but severe case of thrombophlebitis when the signs and symptoms are still localized below the knee. The rationale here is not to prevent embolization but to prevent the central extension of the process into the common femoral and iliac veins by dividing the extending pathway. If the process is kept distal to the major veins, convalescence is shortened, but most important, the sequelae of the postphlebotic leg are prevented. It should be emphasized that all patients with deep venous thrombosis treated surgically should have coincident anticoagulant therapy.

Anticoagulant drugs will not dissolve an existing thrombus, but, if correctly used, should prevent its extension. If immediate anticoagulant effect is desired, heparin is used by the intravenous drip method or by intermittent intravenous or intramuscular injection. Dicumarol (200-300 mg.) is started at the same time but, as its effect is not manifest until 36-48 hours, the heparin should be used for this period of time. Newer anticoagulant drugs, *Tromexan* and *Marumar*, have a similar action to Dicumarol, but their effect is more rapidly obtained. The dosage control, by determination of the prothrombin time, is similar to that of Dicumarol. The great majority of cases of venous thrombosis are at present treated by anticoagulant therapy and the below-mentioned adjuvant measures.

When severe pain and edema are present in thrombophlebitis, it is an indication of more than usual arterial and venous spasm. In such cases, blockage of the appropriate lumbar sympathetic chain will relieve this vasospasm and aid recovery. One to four such procaine blocks lessen pain, reduce the edema, and appear to shorten convalescence. Other adjuvant methods in the treatment of acute thrombophlebitis are elevation of the leg as high as is comfortable and active contraction of the muscles of the limb, the first to aid in draining off the edema and the second to aid also in this respect and to prevent muscle atrophy during convalescence.

When a minor embolus occurs, the treatment consists (as outlined above) in energetic attempts to prevent a second. How-

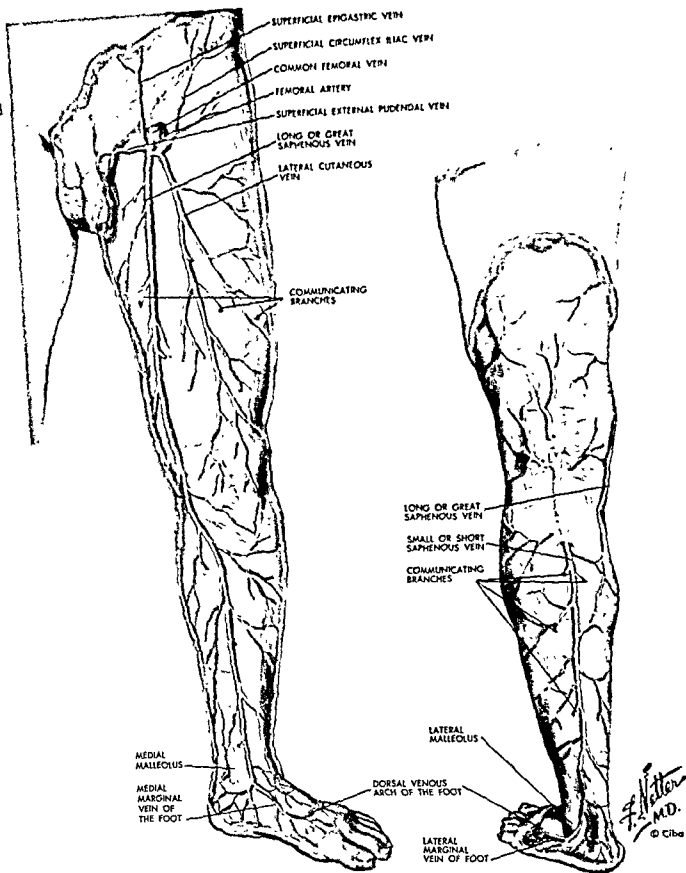
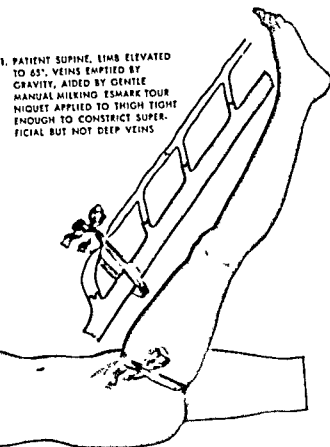
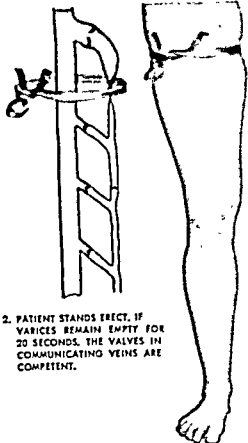


Plate 56.—Superficial Veins of the Leg.

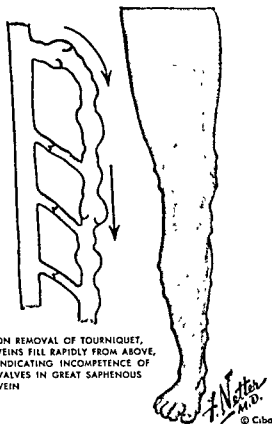
1. PATIENT SUPINE, LIMB ELEVATED TO 65°. VEINS EMPTIED BY GRAVITY, AIDED BY GENTLE MANUAL MILKING. ESMARK TOURNIQUET APPLIED TO THIGH TIGHT ENOUGH TO CONSTRICT SUPERFICIAL BUT NOT DEEP VEINS



2. PATIENT STANDS ERECT, IF VARICES REMAIN EMPTY FOR 20 SECONDS, THE VALVES IN COMMUNICATING VEINS ARE COMPETENT.



3. ON REMOVAL OF TOURNIQUET, VEINS FILL RAPIDLY FROM ABOVE, INDICATING INCOMPETENCE OF VALVES IN GREAT SAPHEOUS VEIN



4. IF VEINS FILL RAPIDLY WITH TOURNIQUET IN PLACE, THERE IS INCOMPETENCE OF VALVES IN COMMUNICATING VEINS, INCLUDING THE SMALL SAPHEOUS VEIN

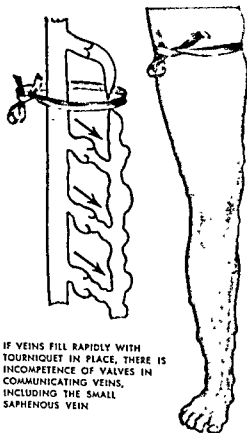
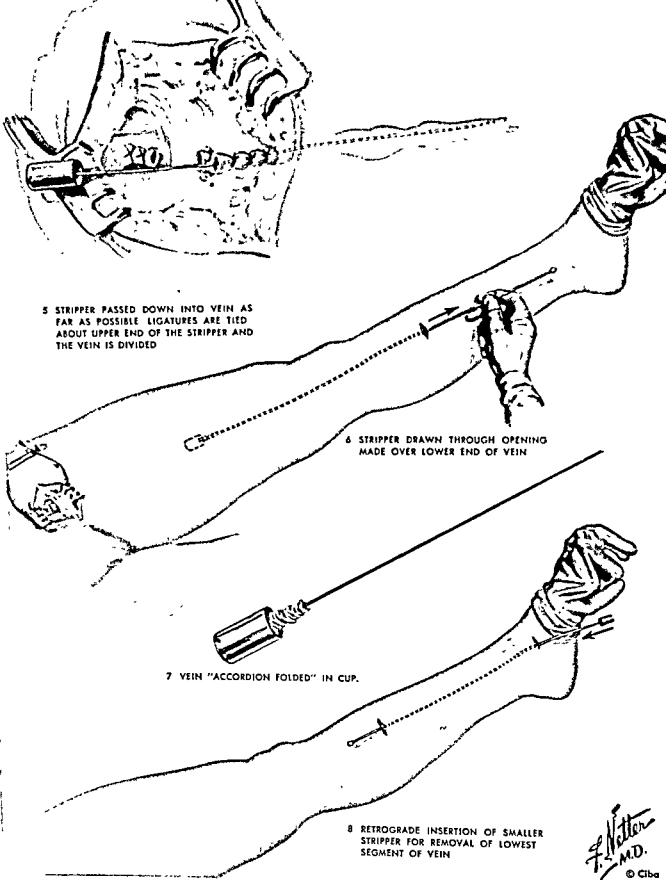


Plate 58.—Trendelenburg Test.



F. Netter
M.D.
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Plate 60.

Courtesy Luke, Josephus C. CIBA
CLINICAL SYMPOSIA 5: 99, 1953



Plate 61

Courtesy Luke, Josephus C. CIBA
CLINICAL SYMPOSIA 5 99, 1933

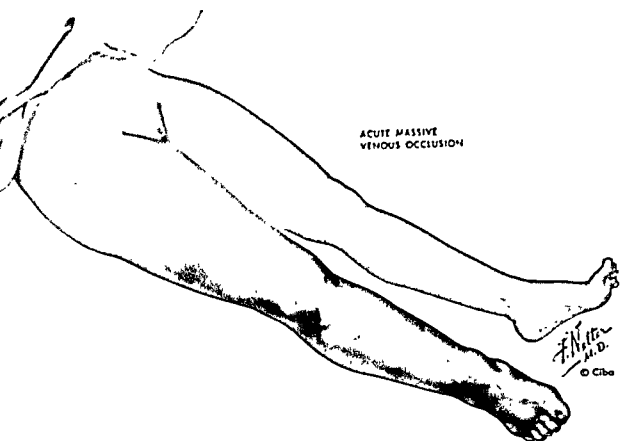


Plate 62.

Courtesy Luke, Josephus C. CIBA
CLINICAL SYMPOSIA 5: 99, 1953



Fig 518 —Retrograde venograms of the femoral system showing valvular destruction, partial recanalization, and collateral dilatation following previous deep thrombophlebitis

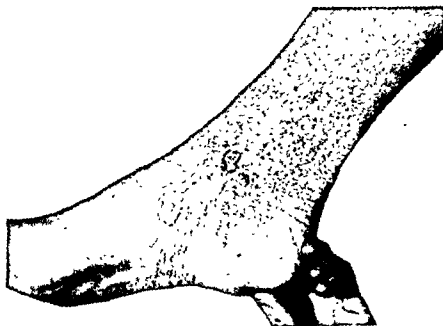


Fig 519 —Postphlebotic ulcer with surrounding eczema and tissue fibrosis

the interval. One of the complications most frequently encountered is that of chronic ulceration. This is seen most commonly just proximal to the medial malleolus. This ulceration should be easily differentiated from the true varicose ulcer by the history of previous deep phlebitis, the absence or paucity of varicose veins, and the other soft tissue changes of deep phlebitis. Other complications are those of eczema involving the lower leg (stasis dermatitis), areas of induration, pigmentation, and subacute inflammation, and the development of secondary varicose veins.

Prevention of the complications of the postphlebitic leg should be the primary aim of treatment. The patient who has recovered from a severe thrombophlebitis should have his condition explained fully. It should be stressed that the leg will never be normal again and that complications can occur if the chronic edema is neglected. A strong elastic stocking should be worn for an indefinite period, employment entailing prolonged standing should be avoided, and the patient should get the habit of elevating the leg whenever possible. Once an ulcer has developed, bed rest with leg elevation and moist hot compresses will result in healing, but recurrence is likely in the future unless the following instructions are carried out.

Instructions for the Continual Care of the Leg Damaged by Phlebitis

- 1 Wear your elastic stocking from the time you get out of bed until you retire, with the exception of bath time. The stocking should be renewed every 3 months, and it is best to have two stockings that can be alternated for cleaning purposes.

- 2 Do not stand for more than 30 minutes without sitting down for 15 minutes and elevating the leg on another chair. When standing, get into the habit of flexing the toes in your shoes and frequently rising on tip toes.

- 3 Plan your day so that you can lie down for 2 to 3 half-hour periods and elevate your leg to a 45 degree angle. The back of a small straight-backed chair is useful for this purpose.

- 4 Whenever you sit down, elevate your leg on a footstool, chair, or chesterfield.

- 5 At night raise the foot of the bed on blocks about 6 inches.

- 6 Apply a bland cold cream to the affected skin at night about every second day.

- 7 Avoid irritation to the involved leg, especially in respect to sunburn and hot-water bottles.

- 8 Be extremely careful to prevent bumping, bruising, or scratching the affected leg.

In the lesser degrees of ulceration, ambulatory treatment with Unna's paste boot or one of its modifications will result in healing, but, again, permanent care of the leg is necessary.

The occasional case of postphlebitic ulceration will be encountered when the ulcer is so large, fibrotic, and long standing that excision and skin grafting are necessary for initial healing. Also some degree of sympathetic dystrophy is seen occasionally in this type of case, and a lumbar sympathectomy will speed ulcer healing and minimize the possibility of recurrence. As mentioned previously, the removal of secondary incompetent varicose veins in the postphlebitic leg will also accomplish the same result. But it cannot be too often emphasized that all treatment is merely partial, the underlying etiologic pathology still remains, and recurrence is almost inevitable unless care is taken to prevent edema and injury to this unhealthy leg.

Phlebitis Migrans

This variety of venous thrombosis is relatively uncommon and consists of multiple waves of thrombophlebitis in short segments of superficial veins of the limbs and occasionally the trunk. This thrombophlebitis occurs in normal veins as distinct from that occurring in varicose veins. The patient complains of hard, reddened tender subcutaneous cords in the location of the superficial veins. One vein segment after another becomes involved, and the condition may last for months. Such a lesion occurs in 30% of cases of thromboangitis obliterans, and a careful examination of the arterial tree is indicated for many months to rule out this possibility. However, most cases of this condition do not show any immediate or late evidence of thromboangitis and therefore constitute separate entities. The etiology is not clear, but the author has had many cases where a careful search for focal infections and their removal have resulted in cure. The tonsils are the most important focus and should be removed on the slightest suspicion. Anticoagulants are of little use because of the chronic nature of the disease.

VASCULAR LESIONS DUE TO COLD

(See Chapter 6, Injuries Due to Physical Agents.)

COSTOBRACHIAL COMPRESSION SYNDROME

There is a group of anatomic or developmental anomalies of the shoulder girdle which by compression of the components of the brachial plexus and/or the subclavian artery can cause a variety of signs and symptoms involving the upper extremity. These conditions are cervical rib, scalenus anticus syndrome, costoclavicular syndrome, and abnormalities of the 1st thoracic rib.

affected is the ulnar because its origin from C8 and T1 is most subject to compression due to its anatomic position. The degree of involvement varies from paresthesias to partial motor and sensory loss. The vascular symptoms include attacks of arteriolar spasm resembling Raynaud's phenomenon. These attacks probably are caused by pressure irritation of the sympathetic outflow to the limb, this outflow running with the medial cord of the brachial plexus and with the subclavian artery. Evidence of organic arterial occlusion is sometimes present, which may be severe due to thrombosis of the subclavian artery, the result of prolonged trauma. Or the picture may be that of occlusion of digital arteries to the

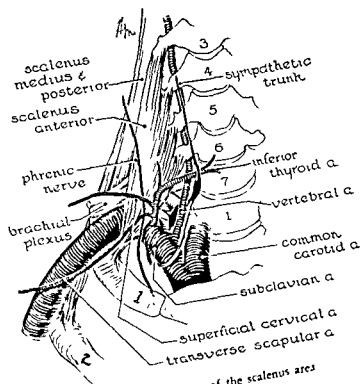


Fig 520—Anatomy of the scalenus area

Signs and Symptoms.—These are common to all the above conditions and can be divided into two main groups, those with evidence of brachial plexus pressure and those with indications of interference with the vascular supply to the limb. A combination of both types may be present. The neurologic picture consists of the symptom of pain localized to the deltoid area or down the arm, most commonly in the distribution of the medial cord of the brachial plexus. The individual nerve most frequently

affected is the ulnar because its origin from C8 and T1 is most subject to compression due to its anatomic position. The degree of involvement varies from paresthesias to partial motor and sensory loss. The vascular symptoms include attacks of arteriolar spasm resembling Raynaud's phenomenon. These attacks probably are caused by pressure irritation of the sympathetic outflow to the limb, this outflow running with the medial cord of the brachial plexus and with the subclavian artery. Evidence of organic arterial occlusion is sometimes present, which may be severe due to thrombosis of the subclavian artery, the result of prolonged trauma. Or the picture may be that of occlusion of digital arteries to the

fingers, presumably on the basis of prolonged spasm. Cold bluish-red fingers result with pain and trophic changes which may be severe enough to produce digital gangrene. When arterial occlusion is present in the subclavian artery, the distal pulses will be palpable or weak, depending on the efficiency of the collateral circulation. The blood pressure in this arm will be low or unobtainable.

The patient will volunteer the information in many cases that carrying heavy objects in

this hand will aggravate symptoms, also that he cannot sleep on this side at night because the hand and arm "go to sleep" and develop pins-and-needles sensations. He finds greater comfort by sleeping with his arms above his head. A definite test (Adson's maneuver) has been devised which indicates that subclavian artery compression is taking place. This is designed to indicate scalenus compression but applies equally well to the other varieties of shoulder girdle compression. A perusal of Fig. 521 will indicate the performance of these tests.

In the differential diagnosis of these compression syndromes, especially where pain and neurologic signs are present, certain other conditions should be considered and ruled out before a positive diagnosis of shoulder girdle

rib is of all varieties, from small stubs to a complete rib articulating with the 1st thoracic rib. Also seen is a short bony rib with a cartilaginous or fibrous cord articulating with the first rib in the region of the scalene tubercle. These accessory ribs are usually bilateral but frequently only one side gives symptoms. The medial cord of the brachial plexus, the sympathetic supply to the arm, and the subclavian artery have to pass over this rib before descending into the arm and consequently are subjected to pressure. Prolonged pressure on the subclavian artery by the cervical rib may cause the condition known as post-stenotic dilatation of the artery where an aneurysmal like dilatation of the vessel occurs distal to the point of compression. This unusual effect has been shown to be the result of

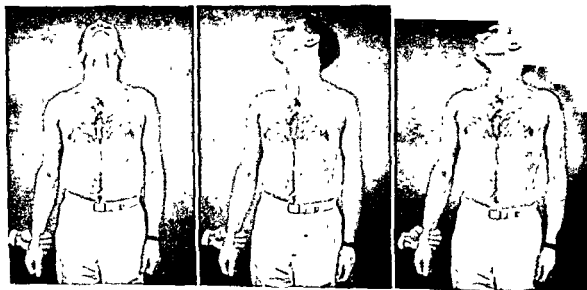


Fig. 521—Steps in the performance of the Adson maneuver

compression can be made. Prolapse of a cervical intervertebral disc is a common cause of this so-called brachial neuritis as is also nerve pressure following osteoarthritis, involving the intervertebral foramina. The medical conditions of neuritis subsequent to diabetes, alcohol, lead poisoning, and vitamin B deficiency should also be kept in mind.

Cervical Rib

About 0.5% of all persons have a cervical rib arising from the 7th cervical vertebra. This

marked disturbance in the hemodynamics at this area. The turbulent and slowed blood flow in this dilated portion predisposes to the occasional complication of thrombosis and the resultant ischemic changes in arm and hand. However, many persons have cervical ribs without symptoms, and, in many others, symptoms do not develop until middle life. The late development of symptoms in cervical rib is probably due to the fact that, as age advances, the posture becomes more stooped and the muscle tone of the shoulder girdle relaxes,

allowing the shoulder to fall forward and downward, thus giving a greater likelihood of compression.

At the Royal Victoria Hospital a series of 40 cases of cervical rib has been studied, of

pain and motor or sensory changes. Three showed pure vascular signs only, whereas 5 others had a mixed vascular and neurologic symptomatology. Fifteen of the 20 patients with symptoms required operation.

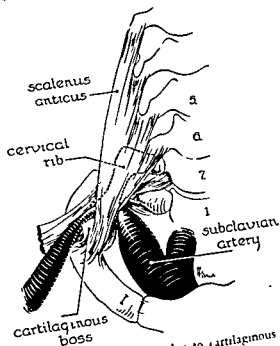


Fig 522 --Case of subclavian artery compression due to cartilaginous extension of cervical rib

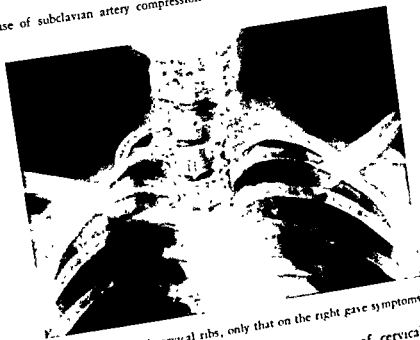


Fig 523 --Bilateral cervical ribs, only that on the right gave symptoms

these 29 were in females and 11 in males, 25 were bilateral and 15 unilateral, and only half of this number had symptoms. Neurologic manifestations preponderated, with 17 showing

The diagnosis of cervical rib is confirmed by x-ray, and surgical treatment consists of section of the scalenus anticus muscle. Resection of a part or the whole rib is, in most

cases, unnecessary. Surgical therapy is, however, reserved for the more severe cases, whereas physiotherapy designed to improve posture and muscle tone in the shoulder area is frequently sufficient in the milder manifestations



Fig 524—X ray of congenital abnormality of upper dorsal spine with elevation of the right 1st rib which acts similarly to a cervical rib

Scalenus Anticus Syndrome

A study of the anatomic drawing will show the anatomic arrangement of this muscle as it attaches to the 1st rib anterior to the brachial plexus and subclavian artery. Spasm or hypertrophy of this muscle narrows the tunnel through which the neurovascular bundle emerges into the arm. This occurs in two ways: the scalenus anticus can cause pressure between itself and the scalenus medius, especially if the latter is abnormally large and more medially placed and, second, subsequent to the muscle spasm the 1st thoracic rib is elevated, causing further compression of the outlet to the arm

Scalenus anticus syndrome is suspected by the previously mentioned signs and symptoms, local tenderness to pressure over the insertion of the muscle, positive scalene tests, and the absence of the other conditions mentioned in the differential diagnosis. An important confirmatory test in both cervical rib and scalenus syndrome is the finding of a systolic bruit over the subclavian artery in the neck which is accentuated during the performance of the Adson test

The treatment is on the same lines as that for cervical rib, with the exception that the surgical therapy required is only section of the scalenus anticus muscle (scaleneotomy) as close to its insertion into the 1st rib as possible.



A



B.

Fig 525—A, Normal venogram of the upper extremity
B, Axillary vein compression with stasis due to costoclavicular compression

Disorders Caused by Abnormalities of the First Thoracic Rib

These are uncommon and only occasionally result in symptoms. The abnormality which is concerned here is a broadening and thickening of the lateral portion of the rib over which the neurovascular structures pass. One feature seen in this variety and that of costoclavicular compression is varying degrees of obstruction to the venous return of the arm. Anatomically, the subclavian vein cannot be compressed by a cervical rib or the scalene muscles, but in abnormalities of the 1st rib or in costoclavicular compression, the vein is affected with the neurovascular bundle.

Surgical treatment may require removal of this section of rib or, more frequently, scalenotomy is all that is necessary. Total removal of the clavicle is also effective.

Costoclavicular Compression

Pressure of the clavicle when pulled downward and backward can cause neurovascular compression against the 1st rib. This factor is rare and is to be suspected only when typical compression symptoms are present and all other causes have been excluded. Treatment is similar to that of scalenus anticus syndrome. Recently, total resection of the clavicle has been advocated so as to remove the upper compressing bone.

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procureable From</i>
Aortic Graft for Arteriosclerosis Obliterans (Illustrates resection of aortic bifurcation in patient with occlusion of this vessel due to arteriosclerosis and replacement with freeze-dried homograft) (1955)	14 min	Sound Color	A. W. Humphries, MD 2020 E. 93rd St Cleveland 6, Ohio
Diagnostic Tests in Peripheral Arterial Disease (1940) (By Norman E. Freeman, M.D., San Francisco, and Hugh Montgomery, M.D., Philadelphia)		Silent Color	American Heart Association, Inc. Film Library 13 E. 37th St. New York 16, N. Y.
End to side Bypass Homograft Without Resection in Segmental Arterial Occlusion (1956) (By Harold Laufman, M.D., Robert Hohlf, M.D., Victor Bernhard, M.D., and Otto Trippel, M.D., Chicago)	12 min	Silent Color *	Harold Laufman, M.D. 720 N. Michigan Ave Chicago 11, Ill
Femoral Grafts for Arteriosclerosis Obliterans (Shows three separate techniques of grafting end to end replacement of segmental block, bypass and an alternate method of bypass using bifurcation graft at distal end of occlusion) (1955)	17 min	Sound Color	A. W. Humphries, MD 2020 E. 93rd St Cleveland 6, Ohio
The Postphlebotic Syndrome (1954) (By Darrell A. Campbell, M.D., Robert W. Gillespie, M.D., Eloise and Merle M. Musselman, M.D., Omaha)	34 min	Silent Color	Darrell A. Campbell, M.D. Wayne County General Hospital Eloise, Mich
Surgical Treatment of Varicose Veins (1955) (By Grza deTolats, MD Chicago)	25 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.
The Treatment of Aneurysm of the Abdominal Aorta by Resection and Aortic Homograft (Illustrates the technique employed in resection and repair of abdominal aorta in an elderly patient with an arteriosclerotic fusiform aneurysm involving the abdominal aorta and its bifurcation) (1953) (By Michael E. DeBakey, M.D., and Denton A. Cooley, M.D., Houston)	26 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn

Chapter 34

Lymphatic System

Josephus C. Luke, MD

EMBRYOLOGY

The lymphatic system has been described as being the "third system" in the body's fluid transportation mechanism and is concerned with the return of tissue fluids through various filtration stations back to the general blood stream. The origin of the lymphatics is still not specifically known, but it is generally agreed that the lymphatics primarily develop from the venous system. The histology is very similar, the vessels are valved, and, anatomically, the two systems are in close approximation.

According to Sabin, the lymphatic system commences from a series of sacs situated close to the confluence of the principal large veins. From these sacs the lymphatic vessels sprout distally and so form the peripheral lymphatic system. The primary lymph sacs are initially formed from a confluence of venous capillaries and are situated on either side of the base of the neck as the jugular sacs, in the retroperitoneal area as the celiac sac, and in each inguinal region as the inguinal sacs. All these become connected, and from them develop not only the lymphatic channels but also the principal masses of lymph nodes. The thoracic duct originates from intercommunication between the celiac and left jugular sacs and receives the communications from both iliac (inguinal) areas. The right lymphatic duct develops from the right jugular sac.

An alternate theory is that the lymphatic vessels have a distal origin from the venous

system and progress centrally until they fuse into the above-mentioned sacs and intercommunicators. A practical application of lymphatic embryology is the fact that the condition known as cystic hygroma, found almost invariably in the neck, is probably the result of a remnant and maldevelopment of one of the jugular sacs.

ANATOMY

Most tissues of the body contain lymphatic capillaries, the exception being those organs or structures which do not contain a normal vascular system. For example, endothelial linings such as the peritoneum or pleura, cartilage, the cornea, and the membranous labyrinth do not contain lymphatic vessels. The central nervous system is devoid of lymphatic vessels because the lymph formed drains directly into the cerebrospinal fluid and, in fact, composes this fluid. Lymphatic capillaries are numerous in proportion to the blood supply to the part, being, therefore, more frequent in such structures as the skin than in the subcutaneous fat. These capillaries are in general larger than the corresponding vascular ones, are lined by endothelium, have a somewhat beaded appearance, and possess bicuspid valves in the larger radicles.

In the skin the lymphatic capillaries are so numerous that no trauma to the skin or injection into the skin can take place without opening numerous lymphatic channels, and so it should be assumed that any injection, be it

intra-dermal, subcutaneous, or intramuscular, is absorbed via the lymphatic system. In the leg a somewhat unique construction of lymphatic vessels occurs with two separate systems (the superficial and deep) only connecting at two locations, the popliteal region and fossa ovalis. No intercommunication occurs through the deep fascia except at these points. This fact is important in an understanding of the relatively frequent condition of lymphedema of the lower extremity.

Situated along the return lymph drainage are strategically placed collections of lymph nodes whose function it is to filter the incoming lymph and so remove foreign material, including bacteria. The nodes contain large numbers of phagocytes that play an important part in the destruction of entering bacteria and

cemia. It is believed that infection reaches the blood stream almost entirely by the lymphatics, this including the viruses whose passage through the lymph nodes is relatively unobstructed.

It can be seen that a detailed knowledge of the various channels of lymph drainage from an organ or region of the body is of prime surgical importance not only from the point of view of the spread of infection but also for a knowledge of the spread of tumor growth. In the majority of malignant tumors, extension is primarily by the lymphatics, and consequently the route of extension can be relatively accurately predicted. Removal of the involved lymphatics and the related nodes is therefore of paramount importance in seeking the cure of a carcinoma.

Lymph Nodes and Vessels

As indicated above, the lymph channels of the lower extremity consist of the superficial and deep sections which communicate only at the popliteal region and through the fossa ovalis in the upper thigh. The superficial channels of the lateral aspect of the foot and leg follow the course of the small saphenous vein, emptying into the deep system at the popliteal space. Before entry into the deep channels, the lymph is filtered by 2-3 popliteal lymph nodes. The medial aspect of the foot and leg and most of the thigh drains into channels which follow the course of the great saphenous vein and join the deep system through the fossa ovalis. Here they pass through the femoral (subinguinal) group of lymph nodes. The corresponding side of the scrotum or labia also drains through the femoral nodes. This group is commonly affected by chronic and acute inflammations in the foot and leg.

Drainage from the buttocks, external genitalia, anal region, vagina, and cervix passes to the inguinal nodes which are situated parallel and superficial to the inguinal ligament. These nodes are frequently involved in venereal infections, skin infections in the drainage area, and cancer of the genitalia, including the cervix. Both the femoral and inguinal nodes drain to the iliac nodes and thence to the main lymphatics of the pelvis, retroperitoneal area, and receptaculum chyli.

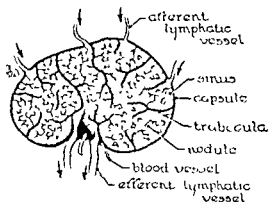


Fig. 526.—Filtration mechanism of a lymph node

also are undoubtedly concerned in antibody production. Enlargement of the lymph node occurs during the filtering process (catarrhal lymphadenitis). Occasionally the bacterial invasion is sufficiently large or virile that the lymphadenitis goes on to necrosis and abscess formation. Or, again, if the infection reaching the nodes is not too virulent and is continuous though mild in type, a state of chronic hyperplasia and fibrous tissue reaction occurs in the nodes and results in permanent palpable nodes (chronic lymphadenitis). These filters can be sufficiently overwhelmed by a virulent infection so that not only does suppuration of the node occur but also the infection continues on past one or more nodes to reach the blood stream and so produce a bacteremia or septi-

LYMPHATIC SYSTEM

In the abdomen, the nodes are very numerous both in the mesenteric area and retroperitoneal spaces. The lymphatic vessels collecting nodes follow the blood supply closely and are located in the mesenteric attachments of the organs. In the stomach the

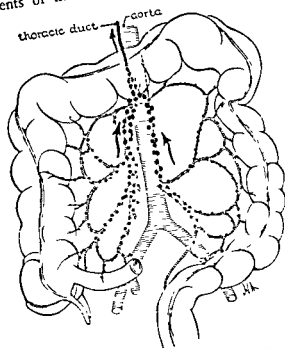


Fig 527—Lymphatic drainage of colon

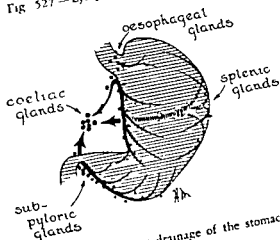


Fig 528—Lymphatic drainage of the stomach

nodes are clustered on either curvature of the stomach, about the lower esophagus, and in the region of the first part of the duodenum. Those draining the small intestine are located in the mesenteric leaves, and frequently the lymphatic vessels leading to these nodes show as small white threads in the bowel wall due

to the milky appearance of the lymph because of the high fat content. This milky lymph (chyle) is also seen in the receptaculum chyli and the thoracic duct, because of the large amount of the lymph collected from the intestinal tract. The degree of milkiness will, of course, vary with the food intake.

The hilar region of the lungs and the mediastinum are especially well supplied with lymph nodes which are a mottled grayish-black color due to the phagocytosed carbon particles which have reached them from the lungs. Also, these nodes are frequently the final resting place of lesser degrees of tubercle infection which has entered through the lungs and which has been overpowered at this first

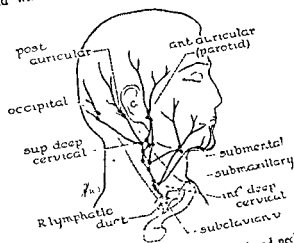


Fig 529—Lymphatic drainage of head and neck

line of defense. As in the abdomen, these arrested tuberculous nodes frequently reveal themselves as serrated calcified spots. Efferent drainage from the mediastinal nodes is mainly to the thoracic duct, but some from the right lung empties into the right lymphatic duct.

The lymphatic drainage of the breasts is of great surgical importance because of the frequency of breast cancer (see Plate 9).

The head and neck have a superficial and deep set of vessels and nodes. The scalp drainage, as in the remainder of the head and neck, is similar on both sides, and the vessels drain sectionally to specific sets of lymph nodes. The occipital, posterior auricular, and parotid nodes receive the scalp lymph, and their

efferents connect with the superior deep cervical nodes. The lymphatic return from the parotid gland, nasopharynx, mouth, lips, and tongue travels through the submaxillary lymph nodes, and that of the tip of the tongue and central part of the lower lip and mouth travels through the submental nodes. Further drainage thence is to the superior deep cervical or directly to the inferior deep cervicals and then to the blood stream via the right lymphatic duct on the right or directly into the internal jugular-subclavian vein junction on the left in association with the thoracic duct.

The upper extremity has few lymph nodes in comparison to its bulk. One node is placed just proximal to the medial epicondyle and receives the drainage from the ulnar aspect of the forearm and hand including roughly the 4th and 5th fingers. The remainder of the superficial and deep lymphatic drainage progresses directly to the axillary group of nodes which lie in the subcutaneous tissues of the axillary space and are readily palpable when enlarged.

PHYSIOLOGY

Lymph is a collection of tissue fluid and, as such, closely resembles blood plasma. The red cell count is understandably low, varying from 500-10,000/c mm. The white cell (mainly lymphocyte) count is usually double this amount. This is so because of the discharge of lymphocytes into the lymph during its passage through the lymph nodes. The protein content is lower than that of blood but varies markedly, depending on the lymphatic area studied and especially (where thoracic duct lymph is being studied) on the time, whether before or after a meal. As a rule, the protein content is about half that of the plasma. The fibrinogen and prothrombin content is low and, consequently, lymph clots but more slowly than blood. Most of the other constituents such as nonprotein nitrogen, chlorides, sugar, urea, uric acid, and calcium have much the same content as the blood.

The lymph pressure and rate of flow are subject to many variables. Studies on thoracic duct fistulas indicate that 1-2 ml. of lymph are lost per minute. Peripheral lymph has a greater pressure than that in the main chan-

nels, the pressure decreasing following passage through a lymph node. The pressure in a normal peripheral vessel rarely exceeds 50 mm of water.

Certain abnormal conditions increase the flow and pressure of lymph. Heat increases production and flow of lymph and, along with the resultant hyperemia, is the reason for the beneficial effect in the treatment of infections. Heat is probably also an accessory factor (in association with prolonged standing) in producing ankle edema in many persons during the heat of summer. Muscle activity increases lymph flow. Also, any factor producing edema obviously increases production and return flow. Examples of this latter group are increased venous pressures and anything that damages the capillary endothelium, allowing increased capillary permeability. This applies particularly to inflammatory edema.

A decrease in the flow and pressure of lymph results from cold and particularly immobility. Congenital malformation of the peripheral lymphatic collecting system, such as those probably present in Milroy's disease and lymphedema praecox, produce decreased flow and varying degrees of lymph stasis with consequent dilatation of the peripheral lymphatic channels. Surgical removal of main lymphatic trunks, their blockage by cancer cells, or infection also gives rise to clinical evidence of lymphatic obstruction with edema.

ACUTE LYMPHANGITIS

In acute inflammations the lymphatic vessels are rapidly affected because of the marked number of cutaneous and deep lymphatics, and extension, local or distant, takes place mainly via the lymphatic system. This spread can be of the local diffuse type when the massive cutaneous lymphatic network becomes involved with coagulated infected lymph and cellular debris. The picture is that of a red, warm, tender, edematous area surrounding the local portal of entry, and the picture is known clinically as cellulitis. Some degree of cellulitis is always present following a surface infection, but the degree varies. Erysipelas is a type of cellulitis but rapidly extending and massive and the result of streptococci. Local edema is the result of the increased tissue fluid produced

is indistinguishable from that of acute appendicitis and consequently the diagnosis is usually made at operation done for a supposedly acute appendix. Red, swollen, and markedly enlarged nodes up to the size of a walnut are found with little evidence of any inflammation of the terminal ileum or appendix. Rarely does suppuration with abscess formation occur, and the usual outcome is rapid subsidence of the condition following appendectomy and the use of the antibiotic drugs.

Chronic Mesenteric Lymphadenitis.—In common with all lymphatic tissue, the lymph nodes in childhood and adolescence are more hyperplastic than in later adult life. The mesenteric nodes share in this involvement and, as a result, produce symptoms due to their rigidity where mesenteric movement is involved. The patient is usually a child 3-13 years of age who complains of intermittent spasms of abdominal pain. The spasms may be momentary or persist up to 15-30 minutes. The pain is not typical colic but is described as an intermittent knifelike pain centered about the umbilical region. Nausea, vomiting, and anorexia are occasionally associated. Vague tenderness is present in the right lower quadrant or mid-abdomen, but splinting is minimal or absent. A mild degree of fever occurs in most cases, but the leukocyte count is little if any elevated. The local point of tenderness is found either in the right lower quadrant or subumbilical region frequently shifts to the left when the patient is turned on the left side (Brennan's sign). The significance of this is the positional shift of the mesentery with its offending enlarged nodes. Such a shift does not take place when the cecum and appendix are involved.

The diagnosis is difficult to distinguish from chronic appendicitis, but the above-mentioned points should tend to indicate the possibility of chronic mesenteric adenitis. Active therapy is necessary only if symptoms persist and interfere with the activities and nutrition of the child. Normally the complaints will disappear with time, especially after puberty. However, when of a disabling nature or when chronic or subacute appendicitis cannot be excluded, then appendectomy is indicated.

On opening the abdomen a somewhat increased quantity of straw-colored peritoneal fluid is encountered. The lymph nodes in the small bowel mesentery are numerous and enlarged up to the size of a bean. Biopsy of one of these nodes invariably shows a catarrhal lymphadenitis. Removal of the relatively normal-looking appendix is all that is indicated in these cases and appears to bring about a complete regression of signs and symptoms in most cases. Formerly it was considered that these enlarged lymph nodes were a chronic hyperplastic form of tuberculous adenitis, but pathologic examination of many nodes removed for biopsy failed to confirm this idea, also bacteriologic study of the removed node always fails to yield any growth.

Specific Forms of Lymphadenitis

Tuberculous Lymphadenitis.—This form of lymph node involvement is much less frequent than formerly in North America because of the strict control of the public sale of milk. Pasteurization has reduced this disease to a minimum, especially among city dwellers, and the cases now seen usually come from the country in areas where pasteurization is not used. In England, Scotland, and Wales less stringent milk control still produces a large number of node, bone, and joint tuberculosis.

Tuberculous adenitis in the child is almost invariably the result of the bovine type of bacillus acquired from infected milk, but in the occasional adult case the organism can be either bovine or human in type. The lymph nodes of the neck are most frequently involved, the germs entering the lymphatic system from a tonsillar focus or through one of the pharyngeal lymph plaques, infected tooth, or other break in the lining of the oral cavity. The organism can come via infected milk or sputum coughed up from a pulmonary lesion.

The mesenteric lymph nodes are next most commonly involved, becoming infected from organisms absorbed from the gastrointestinal tract originating either in infected food and drinks or the swallowing of tuberculous sputum. The ileocecal region is the most frequent portion of the gastrointestinal tract to be involved. An x-ray of the abdomen taken for other reasons frequently shows an irregular

roundish patch of calcification about 1.25 cm. in diameter. These are tuberculous nodes in which the process has finally been conquered but not before node destruction and later calcification of the caseated node have occurred. This finding can be associated with no past or present signs or symptoms of the disease, and such a finding is common also in routine chest radiography.

Nodes draining one of the extremities are rarely involved by tuberculosis unless the organism gains entrance to the distal tissues through a small abrasion. Such an accident is most common in persons such as nurses whose work brings them in contact with open cases of tuberculosis. A localized, firm, tender, painful nodule develops in the skin at the site of accidental entry of the germ. A small, shallow, painful ulcer usually develops at this site, and, later, enlarged, tender regional nodes appear. The local finger lesion was formerly known as a butcher's wart because of the frequency of this condition in those who handled infected meat.

Pathology.—When the bacillus of tuberculosis is conveyed to a lymph node by the lymphatics, the reaction in the node depends on the degree of invasion and the resistance of the host to the invader. All degrees of node response may occur from single node hyperplasia to that of caseation and abscess formation. In the lesser degrees, typical tubercle formation may be hard to find, making the determination of the correct diagnosis difficult even on pathologic section, therefore the diagnosis may be easily mistaken for chronic non-specific lymphadenitis.

Clinically, the nodes are initially discrete and elastic but later become matted into masses due to the development of periadenitis. An actively infected or caseating node may rupture into the blood stream and result in the military generalized form of the disease. In the more advanced cases tubercles are numerous, areas of caseation are obvious, and the typical "cold abscess" of tuberculosis may result. Incision and drainage of such an abscess under the mistaken impression that it is due to pyogenic organisms invariably produce a fistula which remains as a discharging sinus indefinitely because of the underlying infected

node and the inevitable secondary infection incident to the draining wound.

The moral to be emphasized in this connection is to suspect all abscesses which have developed in a subacute manner, especially in the neck. In the tuberculous case, the constitutional reaction is minimal, the origin of the infection is obscure, and the abscess shows minimal signs of inflammation, tenderness, and surrounding induration. When doubt exists, the pus should be aspirated and cultured, and if tubercle bacilli are found, then incision of the infected abscess is indicated with removal by curettage of the underlying infected node or nodes.

Formerly, extensive surgery including block dissection of all the neck nodes was the accepted procedure in tuberculous adenitis, but at present such an extensive degree of the disease requiring such treatment is rarely seen. Improvement in the medical management has also decreased the need for surgery. Control of the milk supply, removal of infected tonsils, improvement in general health subsequent to adequate rest, nourishment, vitamins, and heliotherapy will result in cure in most instances. As indicated above, the role of surgery in tuberculous adenitis is the evacuation of abscesses and the local removal of caseating abscessed nodes. Streptomycin combined with either para-aminosalicylic acid or one of the isoniazid group of drugs is the most recent successful aid to treatment.

Syphilitic Lymphadenitis.—Involvement of lymph nodes is seen in both the primary and secondary stages of syphilis. In the former, the nodes adjacent to the chancre become involved in 5-14 days following inoculation. The inguinal nodes are therefore most commonly involved, and the nodes present as firm, discrete, tender nodules. The adenitis is due to a combination of the *Spirochaeta pallida* and other secondary invaders from the chancre. The diagnosis is not difficult when the chancre is seen and is confirmed by dark-field examination of the secretion. The secondary stage of syphilis is frequently associated with a generalized lymphadenopathy, the nodes again being discrete, rubbery, and nontender and do not go on to suppuration. Tertiary gumma in lymph nodes is extremely rare and will be less common in the future because of the decrease in incidence,

improved detection, and improved treatment of this disease

Lymphogranuloma Venereum (Lymphogranuloma Inguinale).—This condition has been called the fourth venereal disease and is caused by a filtrable virus. The genitalia are the most common site of entry, but of course digital and oral origins can occur. The initial surface lesion rarely is as large as a chancre and may be so insignificant as to go unnoticed. Several weeks after contamination, the regional nodes become swollen, indurated, and painful. Necrosis of the node results and suppuration occurs. This pus tends to burrow to the surface or surrounding structures unless aspiration is carried out. In the inguinal region, ulceration and sinus formation with later extensive scarring are the usual sequelae. In some cases extension to the perirectal tissues with perineal or rectal fistula takes place. The incidental scarring subsequent to this type of spread may result in extensive stricture formation of the lower rectum. Such a sequel appears to be more common in females, and these strictures were formerly supposed to be syphilitic in origin because the pathologic picture is that of a nonspecific granuloma.

The diagnosis should be suspected from the clinical picture of the inguinal bubos or the presence of a rectal stricture. Carcinoma and posthemorrhoidectomy strictures should be excluded. The Frei test gives confirmation to the diagnosis and consists of an intradermal injection of diluted pus taken from one of the lesions and autoclaved at 56° C. A positive test is indicated by a central papule at the site of injection with more than a 1 cm. ring of surrounding erythema. Treatment consists of aspiration of abscesses and the use of massive doses of Aureomycin and penicillin.

Hodgkin's Disease

Definition.—In 1832 Hodgkin described a series of glandular swellings associated with splenomegaly and made the first description of the disease which bears his name. It is likely that he described other lesions in his original paper, but four of the cases appear to fit closely with what we now know as Hodgkin's disease. The etiology of this condition has not been clearly settled because it shows not only

many of the features of an infective granulomatous lesion but also those of a malignant tumor, being particularly invasive in its final stages.

Jackson and Parker have made a differentiation of varying stages of this disease and have called the initial stage *Hodgkin's granuloma*, indicating that the pathologic picture is close to but not typical of Hodgkin's. This is an important differentiation because of the pathologic difficulties in labelling a case one of true Hodgkin's disease. The author has several cases in which a pathologic probable diagnosis of Hodgkin's disease has been made and a poor prognosis given, only to have the patient continue in good health or with further episodes of symptomless glandular enlargements for more than 15 years. It is obvious that such longevity indicates features not in keeping with true Hodgkin's disease in which the prognosis is fatal in 1-4 years. This latter fatal group is called, by Jackson and Parker, *Hodgkin's granuloma*. The malignant, invasive variety of Hodgkin's disease which appears to progress from the granulomatous stage has been called, by Ewing, *Hodgkin's sarcoma* and appears mainly in the older age group. Therefore it would appear wise to divide this disease into the three above stages, each of which has to be settled by a combination of clinical and pathologic criteria.

Diagnosis.—The sex incidence appears higher in males (3:1), and the disease may occur at any age. The paraganuloma is seen mainly in postpubertal adults, the granuloma in the early adult age, and the sarcomatous type in the older age groups. The patient usually presents himself with a gradually progressive enlargement of various lymph nodes particularly those of the neck. In the early stages (paraganuloma and early granuloma stage) the constitutional symptoms are absent but later weight loss, anorexia, anemia, and pressure symptoms from the node masses are present. An unusual variety of unexplained fever (Pel-Ebstein) is occasionally associated with this disease. The temperature rises over a few days to a level of 101°-103° and is maintained at this level with minor variations for 7-14 days, after which it returns to normal. Recurrent bouts of this pyrexia usually occur.



Plate 64.—Hodgkin's Disease.

The spleen is moderately enlarged in about 70% of the cases, and blood examination shows, in the later stages, a definite anemia, occasionally eosinophilia, and an increase in large mononuclear leukocytes. These blood changes are seldom sufficiently characteristic to be diagnostic. Primary involvement of the gastrointestinal tract can occur.

Pathologic Appearance.—The lymph nodes are enlarged but remain discrete and rubbery until the late stages. They have an elastic consistency unlike the rocky hardness of secondary cancer deposits in nodes. On section the node shows a gray, translucent appearance, markedly different from the cephaloid appearance of secondary cancer. In microscopic section, the characteristic points are the replacement of the normal lymphoid tissue by a multitude of different cell types. The most prominent of these is a large, pale epitheloid cell, but most characteristic are the giant cells. The latter are mononucleated or multinucleated and are called after their discoverers, Reed and Sternberg. This characteristic cell is found both in the paragranuloma and in the granuloma. Other cells present include eosinophils, lymphocytes, and monomorphonuclear and polymorphonuclear leukocytes. The above picture is also seen scattered in other organs, especially the spleen, liver, and bone marrow. Hodgkin's sarcoma usually shows direct extension of the disease into lung, bone, liver, and portions of the gastrointestinal tract.

Treatment.—Treatment is a combination of surgery and radiotherapy. Surgery is indicated mainly for the purpose of obtaining biopsy material, but it is wise to remove the entire enlarged node at time of operation rather than a mere portion of the node. X-ray therapy produces remarkable regression of the enlarged nodes and is used in different locations as new node masses arise. In the paragranuloma, the regression following x-ray therapy may last for years, but once the disease is well established, the benefit from this therapy is only temporary as other manifestations soon arise. However, there is no doubt that x-ray therapy is the best therapeutic agent available, but unfortunately recurrent enlargement of the original nodes following x-ray

responds less favorably on secondary irradiation. Teroplerin and nitrogen mustard may be useful in the terminal stage to give some alleviation of symptoms but are purely palliative.

Lymphosarcoma (Lymphoblastoma)

Definition.—This very fatal disease can be considered a primary neoplasm of lymphatic tissue. It has the characteristic of appearing in scattered areas of lymphatic tissue almost simultaneously. There is considerable clinical resemblance between this form of lymphatic abnormality and that of Hodgkin's disease as the initial complaint is that of a lump in one of the lymph node areas. As in Hodgkin's disease the neck is the commonest site of origin, although the groin and axilla are also frequently primarily involved. The individual lymph nodes rapidly become fused from local extension of the tumor and even may ulcerate through the skin.

Diagnosis.—There are no specific symptoms of the disease, the patient seeking attention because of a lump or symptoms which are the result of pressure and involvement of surrounding structures. The spleen may be found to be somewhat enlarged. Similar to Hodgkin's disease, the blood changes are not characteristic but usually show a leukocytosis which can be caused by either an increased lymphocyte or polymorphonuclear count. In the former type, confusion may result from the resemblance to lymphatic leukemia. Anemia and some fever are frequently associated with the late cases and generalized metastases may be spread throughout solid organs. Lymphatic leukemia may develop in a case of lymphosarcoma and vice versa, especially in childhood when the greatest incidence of the disease takes place.

Not only lymph nodes but other lymphoid structures are primarily the seat of the disease. These include the tonsils, the adenoids, and the gastrointestinal lymphoid tissue. In the gastrointestinal tract the stomach is the organ most commonly affected, and the preoperative clinical x-ray picture is indistinguishable from carcinoma. The ileum with Peyer's patches may also show the first signs of the disease, whereas the rectum and the colon are next in fre-

quency. At the Royal Victoria Hospital 23 cases of primary lymphosarcoma of the gastrointestinal tract were found in a total of 100 cases of this disease.

Cases of Lymphosarcoma.—

Generalized spread	56
Primarily retroperitoneal	21
Gastrointestinal	23
Stomach	6
Duodenum	3
Jejunum	2
Ileum	3
Cecum	3
Colon—other parts	3
Rectum	3
Total number of cases	100

Pathologic Features.—The gross appearance of the cut node is not characteristic. It is whitish gray to pink in color and is softer than the corresponding node of Hodgkin's disease. Fusion of contiguous nodes occurs early in the disease.

Microscopically, two main varieties of pathologic types are distinguished. The more common is that in which the node appears to have been replaced in its architecture by a diffuse collection of small round cells resembling small lymphocytes. Occasionally a larger type of lymphocyte is seen similar to the appearance of a lymphoblast. This variety shows a uniform pathologic appearance differing markedly from the pleomorphic cell types of Hodgkin's disease. Giant cells do not occur in lymphosarcoma. In the second variety, the reticuloendothelial elements appear to be primarily involved, and consequently it has received the name of a *reticulum cell sarcoma*. It is argued by some authorities that this is not a variety of lymphosarcoma but a separate tumor which arises from the reticuloendothelial system of lymph nodes. Especially does this concept hold when it is known that this type of tumor can be primary in bone and other tissue where lymphatic tissue is minimal. In the lymph node, the structure is destroyed and is replaced by large, pale staining cells of endothelial type, arranged loosely and with a more pronounced reticulum than in the lymphoid type.

It must be borne in mind that diagnosis of one of the lymphomas on pathologic appearance only may contain a margin of error be-

cause of the considerable variations in the picture from case to case. At times, a simple hyperplastic lymph node may be difficult to differentiate from the lymphatic variety of lymphosarcoma, consequently diagnosis should be made in combination with a suggestive clinical picture and a progressive clinical course. It is likely that patients with lymphosarcoma who survive many years have not had authentic cases of the disease.

Treatment.—Treatment is similar to Hodgkin's disease, with surgery being limited to the obtaining of material for biopsy. Radiation therapy produces a rapid melting away of the nodes and relief of symptoms, but, unfortunately, the disease usually recurs in other areas. Radiation therapy can be used as a diagnostic test, especially in those cases in which the mediastinal nodes are involved. The obtaining of biopsy material is not practical in such cases, and if the symptoms rapidly decrease and the chest picture rapidly improves under treatment, it is likely that lymphosarcoma is the cause of the glandular enlargement. As in Hodgkin's disease, nitrogen mustard is of some palliative benefit in the advanced cases. Cortisone and ACTH therapy has so far been disappointing in the treatment of lymphoma.

Brill's Giant Follicular Hyperplasia (Giant Follicle Lymphoma)

This variety of lymph node enlargement is of importance because of its potentialities. Pathologically it is characterized by the large number of hypertrophied lymphoid follicles seen in the node. These can often be seen grossly in the freshly cut node. The enlarged follicles are packed with actively proliferating lymphocytes sufficiently neoplastic as to invade the node capsule. As a rule only a few nodes are involved, and it is rarely that the disease becomes generalized.

The importance of this variety of lymphoma is the frequency of its progression (about 65%) to one of the more malignant lymphomas. Lymphosarcoma, either lymphocytic or reticulum cell type, is the most frequent, but Hodgkin's disease has also been reported to arise from this focus. Radiation therapy is the best treatment, but constant observation must be made for possible future developments.

THE THYMUS

Although generally considered a portion of the lymphatic system, the thymus is composed of two separate elements. The medulla is derived from the 4th branchial cleft and is composed of epithelial cells derived from the cleft. These are mainly collected in cell aggregations known as Hassall's corpuscles whose function is not fully known. The thymic cortex is composed mainly of lymphocytes, and the variations in size of the organ during disease states and in varying ages are mainly due to the increase or decrease of the lymphocyte content.

In disease states, the thymus decreases in size due to loss of lymphocytes, whereas in sudden death the gland size corresponds to the normal. This latter finding was formerly mistaken to indicate a pathologic increase in the size of the gland, especially in infants and children, with sudden unexplained death. The terms *status thymolymphaticus* and *thymic death* were coined in an attempt to describe these cases, but it has been shown that these so-called enlarged thymus glands were either normal or little enlarged. At birth the gland weighs about 13 Gm., and increases to 35 Gm. at puberty, gradually becoming atrophied in adult life. The thymus rarely is sufficiently enlarged to produce direct pressure symptoms on the trachea but should be kept in mind in cases of unexplained moderate dyspnea. X-ray, including anteroposterior and lateral projection, will give the clue to such a condition. In diseases which result in lymphatic hypertrophy, the thymus is also enlarged, including lymphatic leukemia and Graves' disease.

Tumors

As the thymus consists of the two separate elements, cortex and medulla, it is heir to rare tumors involving the cortical lymphatic tissue or the epithelial-celled medulla. The former type resembles lymphosarcoma either of the lymphocytic or reticulum-celled type and is known as *malignant thymoma*. The epithelial cell type is a true *carcinoma* arising from Hassall's corpuscles. Both varieties are highly malignant and rapidly fatal, yielding only briefly to x-ray therapy. Local compression and

invasion of the chest structures and lymphatic node spread occur early. General metastases sometimes are found.

PERIPHERAL ABNORMALITIES OF THE LYMPHATIC SYSTEM

Tumors of Lymphatic Origin

Tumors of the lymphatic system are relatively rare if the dilated lymph spaces associated with acquired or congenital lymphedema are excepted. True lymphatic tumors can be divided into the simple, the cavernous, and the cystic. The *simple lymphangiomas* are usually congenital and are manifest as small warty tumors on the skin or mucous membranes. They are elastic to the feel and white to pink in color and consist of a meshwork of dilated lymph spaces and lymph vessels set in a thin reticular network. The *cavernous type* is also congenital in origin and is frequently combined with hemangiomatous tissue. Large areas of the skin and subcutaneous tissue may be involved and result in a tumor-like mass of dilated vesicles containing lymph with a spongy mass of grayish tissue in which are mingled spots of hemangiomatous tissue. These tumors are most commonly seen in the upper extremity and shoulder region, the lips, eyelids, and tongue. Surgical excision is the best therapy, but recurrence will take place if not totally removed. X-ray and radium can be used but are probably most effective where excision has not been total or where recurrence has taken place.

Cystic lymphangioma (cystic hygroma) is also congenital and develops from endothelial cell rests remaining from the formation of the embryonic jugular sacs from which the lymphatics of the head, neck, and arms develop. They are most common at the base of the neck but may occur in the situations of the other primary embryonic lymph sacs (inguinal and celiac). Cystic hygromas may be well developed at birth and cause dystocia or may develop in early childhood. The typical one is a large, soft, cystic swelling in the supraclavicular area, lobulated, and containing large quantities of lymph. Well-marked transillumination occurs. In common with other lymphatic collections, they become infected easily, and

treatment should be carried out early to prevent this complication. Radical surgical excision is the best therapy.

LYMPHEDEMA

The development of the lymph vessels is closely akin to that of the veins and they resemble veins in being endothelial-lined tubes containing valves. They closely follow the main vascular channels. The lymph in these channels is strained through various sets of lymph nodes and is collected in the main lymph ducts which empty into the venous system. In consequence of their similarity to the venous system, lymphatic vessels are heir to many of the abnormalities that affect veins. In the extremities there are two separate sets of lymph channels, those of the superficial tissues and those of the deep tissues. In the leg, the superficial ones follow the great and small saphenous veins and communicate with the deep vessels only in the popliteal and groin areas.

Lymphedema is most common in the lower extremity due to the upright posture and the frequent anomalies of the vessel walls and valves which occur. In this respect, the parallel with the venous system is marked, one producing lymphedema and the other varicose veins. Any abnormality that results in lymphatic obstruction or stasis can result in lymphedema. Such abnormalities are manifested mainly in the superficial lymphatics because of the lack of support in the subcutaneous tissues. These derangements include congenital weakness of lymphatic walls and valves, block dissection of lymph nodes and channels, x-ray obliteration of these channels, or their occlusion by neoplastic cells or fibrosis following multiple waves of lymphangitis and lymphadenitis. This occlusion and subsequent stasis result in dilatation of the distal lymph spaces to all degrees from mild swelling to the huge leg of elephantiasis. The collection of lymph in numerous dilated lymph channels in an extremity results in gradually progressing fibrosis as the high protein content of the lymph is a powerful stimulant to fibroblastic proliferation. This fibrosis in an extreme degree produces the

warty excrescences, the thickened inelastic skin, and doughy subcutaneous tissue of the elephantine leg seen in long-standing lymphedema. Infection in this static lymph is also a frequent accompaniment, the streptococcus being the most common organism. Recurrent attacks of cellulitis develop, each of which causes further lymphatic obstruction by its associated lymphangitis and lymphadenitis.

Lymphedema Praecox

This is a variety of lymphedema of the leg and rarely of the arm, which comes on most frequently about the time of puberty but may be seen either earlier or later than this period. Females are more commonly affected



Fig. 530 -- Lymphedema praecox, left leg, in woman aged 20 years

than males. A gradual enlargement of one leg, and possibly later the other, occurs beginning in the foot and ankle and slowly progressing up the leg. The origin is probably analogous to that of varicose veins where a congenital deficiency of the lymphatic system is present involving the lymph valves and possibly the vessel walls. These defective ves-

sels gradually fail to function normally, and dilatation of the lymph vessels to lymph sacs occurs in the most dependent part of the limb. Lymph stasis with enlargement of the limb occurs, this enlargement originally disappearing with elevation of the limb, but later, due to the increased lymphatic enlargement and the accompanying fibrosis, the enlargement only partially subsides on elevation. The patient originally reports for cosmetic reasons, but later complains of tiredness, heaviness, and aching of the enlarged leg. Subsequent waves of erysipeloid-like inflammation of this lymph-soaked tissue may result

Obstructive Lymphedema

Actual occlusion of lymphatic pathways can be produced in the various ways mentioned above. The degree of lymphedema will depend on the extent of this obliteration and resembles the hereditary variety in being confined to the skin and subcutaneous tissue. The swollen arm after radical mastectomy for carcinoma is the result of lymphatic vessel obliteration by carcinoma, chronic infection, x-ray, or radical removal of the pathways, whereas a swollen leg is a late complication of extension of carcinoma of the cervix for the same reasons. However, surgical removal of lymphatic pathways seldom produces the degree of edema seen in the stenosing lesions caused by cancer or infection.

One of the more common causes of lymphedema of the acquired type is a stenosing lymphangitis and lymphadenitis, the result of multiple attacks of lymphangitis usually produced by the streptococcus. The history of such a case is of many attacks of erysipeloid infection in the extremity associated with chills, fever, and general malaise. Each attack is followed by increasing degrees of lymphedema in the limb. This chronically edematous limb appears to harbor the offending organism in a quiescent form between attacks, and the infection is reactivated by more than usual overuse of the leg. This variety of lymphatic stenosis leads to the immense legs of elephantiasis. The word *elephantiasis* is purely a descriptive term and does not denote the etiology. In tropical climates elephantiasis is more common due to the filaria Bancrofti which settle mainly in lymph channels and nodes and set up a chronic irritation (frequently in association with the streptococcus) and so produce an extreme degree of lymphatic occlusion.

Swelling of an extremity due to lymphedema has several characteristics. The enlargement is usually unilateral and the swelling subsides only slightly on recumbency in the well-established case. The extremity is whitish in color and the edema is doughy and pits only on prolonged pressure. Varicose veins are absent and there is no history of previous involvement of the deep venous system. The arterial circulation is normal. The symptoms



Fig 331—Lymphedema praecox in man aged 26 years

Milroy's disease is almost identical to lymphedema praecox, but, to conform with the original description of Milroy, it must be both hereditary as well as congenital and therefore present in other members of the same family.

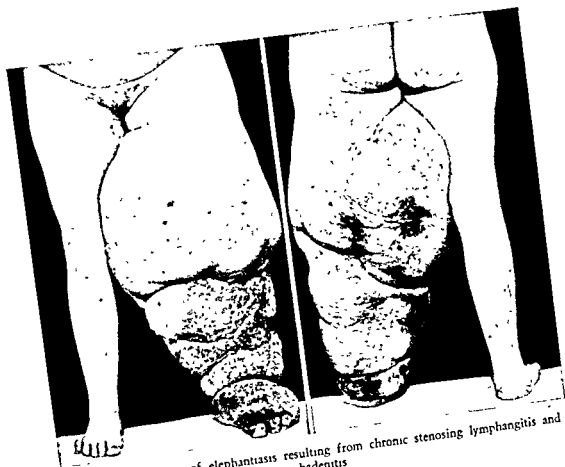


Fig 532—Severe degree of elephantiasis resulting from chronic stenosing lymphangitis and lymphadenitis

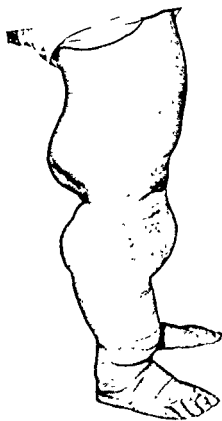


Fig 533 Elephantiasis resulting from multiple previous attacks of leg erysipelas and associated lymphatic blockage

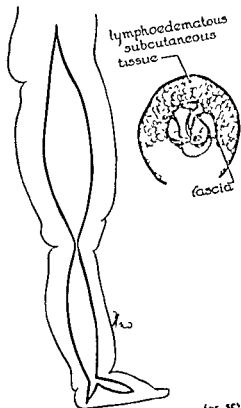


Fig 534—Kondoleon procedure for severe degrees of lymphedema

are those due to the result of the increased size and weight of the limb.

No reliable method has yet been devised to compensate for blockage of the lymphatic return. Consequently treatment is mainly palliative and consists, in the early stages, of elevation of the limb as much as possible and the wearing of compression bandages, the best being a strong elastic stocking or strong elastic or rubber bandage. If the degree of swelling is great and is not controlled by conservative measures, then surgical therapy is indicated. This consists of the Kondoleon procedure and its modifications which remove as much as possible of the lymph-soaked skin, subcutaneous tissue, and deep fascia of the limb. This can usually be done in two or three stages to complete the circumference of the leg. The original plan in this procedure was to establish a communication between the superficial and deep lymphatics whereby the former could drain into the latter by the development of new channels made possible by removal of the deep fascia. However, the major benefit probably results from the mechanical removal of the lymphedematous tissue. Following this method, a strong elastic stocking must still be worn to prevent swelling, as the original reason for the blockage still persists.

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Chapter 35

Amputations

Josephus C. Luke, M.D

It is a distressing fact that despite the recent advances in surgical diagnosis and treatment, the number of limbs requiring amputation is increasing rather than decreasing. The reasons for this are those of the changing modern life with its high degree of mechanization, and its increased life span allowing for a greater frequency of the circulatory ailments of advancing age, as well as two destructive world wars in the first half of this century. Modern surgery has saved many lives in the amputated group, increasing the number of amputees, but has failed in most instances in devising means of resuscitating an ischemic limb. Therefore the need for amputation arises sufficiently often that every surgeon should be completely familiar with the indications and technique

INDICATIONS FOR AMPUTATION

Trauma.—The only true indication for amputation following injury is irreparable damage to the blood supply to the limb and development of complete ischemia of the distal part. Such an injury is usually associated with major nerve damage and multiple fractures.

Arterial Occlusion.—As mentioned previously, the most common indication in civilian life is occlusive arterial disease. The increasing age of the population and the prevalence of arteriosclerosis with or without diabetes necessitate the removal of many limbs because of gangrene. However, in the past 5 years the

increasing use of arteriography has demonstrated many cases where the employment of arterial grafts has saved legs from major amputation. Buerger's disease, frostbite, acute arterial spasm, acute arterial thrombosis, and embolism are less common conditions leading to the same result.

Infection.—It is rare in this era of antibiotics for a limb to be sacrificed because of acute or chronic infection. The only exception is the case of widespread gas gangrene not localized to a single group of muscles, and it is not too much to hope that this type of infection will soon be controlled by new chemotherapeutic agents.

Malignancy.—Amputation of the limb well above the lesion is still the chief method of therapy in sarcoma involving the extremity.

Deformity and Uselessness.—Amputation for these reasons is becoming less and less practiced because of the improvement in orthopedic methods and the use of superior orthopedic appliances. This cause finds its greatest frequency in the removal of useless fingers and toes.

SELECTION OF LEVEL OF AMPUTATION

Where amputation is necessary following acute trauma, as much of the limb as possible should be saved. This means that amputation is carried out at the upper limit of the site of trauma. This should be done regardless of

whether the stump is going to be final or require revision. This rule is even more important in battle than in civilian casualties, because, in the latter group, continuous uninterrupted treatment is possible. The reason for this selection of amputation level is that contamination or actual infection is always associated with open wounds and hence the original amputation level is unlikely to be a properly healed wound, especially if done by the guillotine method. An unsightly or unsuitable stump can always be revised at leisure when the initial wounds have soundly healed.

The same ruling applies in cases of frostbite when the gangrene is always more extensive on the surface than in the deeper tissues. Extreme conservatism is indicated to allow a proper line of demarcation to develop. After the dead tissue is removed, the use of suitable skin grafts will save many a foot or hand which originally appeared doomed.

In those cases where amputation is indicated because of deficient blood supply, the result of main artery occlusion, no hard and fast rules can be laid down as to the proper level of amputation. The fact should be kept in mind that amputation is being done for an arterial lesion which is usually progressive and that a successful low amputation may break down months hence because of progression of the disease. This observation applies especially to Buerger's disease. Another point to be remembered is that a lower level stump, though healed, may not be sufficiently supplied with blood to withstand the trauma incident to wearing an artificial limb. Each case is individual, and a careful preoperative study is necessary to determine the efficiency of the existing collateral flow and to ascertain whether this flow can be improved by suitable measures.

It is our practice to carry out the following investigations prior to every amputation indicated because of occlusive arterial disease. Palpation of the pedal and leg arteries is of extreme importance, because it can be stated somewhat dogmatically that primary intention healing (in the absence of infection) will take place after toe amputations if the dorsalis pedis and posterior tibial arteries are felt to pulsate and almost invariably if only one of

the two is actively functioning. Similar findings apply to a transmetatarsal amputation. Healing of a below knee site of election amputation will take place when the popliteal artery is found to be patent, and a good result will follow a lower third of thigh amputation when the common femoral artery is patent.

Frequently a lower amputation can be done successfully in the absence of the above criteria where it can be shown by sympathetic nerve block that the circulation at the proposed amputation level is capable of further dilatation, this being indicated by increased skin temperature readings. Where such improvement is seen, a lumbar sympathectomy done at the time of amputation will ensure a successful lower level of amputation. When infection is associated with the gangrene, a marked local inflammatory response will indicate good collateral circulation and aid in the decision for a minor amputation. The lack of chronic ischemic changes in the soft tissues of the foot also indicates an adequate set of collateral vessels. The histamine flare test and the use of intravenous fluorescein are aids in the determination of the level of adequate collateral circulation and therefore successful healing. Arteriography is also indicated in the selection of those cases suitable for the use of arterial grafts.

In gas gangrene, amputation levels should be above the area of visible diseased tissue. If, at operation, certain muscle groups are found to be involved higher than was anticipated, these groups can be excised and so allow for a longer stump than otherwise would be the case.

In limb sarcoma, the level should be as high as is practicable in order to reduce the possibility of further extension, but it is unfortunately too common that distant metastases have already occurred via the blood stream before amputation is done. A mid-thigh amputation is indicated for growths involving the foot and lower leg or hip disarticulation in tumors of the lower femur. The same levels apply to the upper extremity and a forequarter amputation should be carried out for the neoplasms involving the shoulder region or hind-quarter ablation in those involving the hip area.

REFRIGERATION ANESTHESIA FOR AMPUTATIONS

The use of refrigeration as the complete anesthetic for a limb amputation is occasionally indicated. The person concerned is usually elderly, diabetic, and in poor general condition, coming from a slum district where his previous medical attention has been sketchy or nonexistent. His diabetes has been out of control and becomes more so in association with the severe infection of the foot and leg. He is semicomatose, irrational, and running a high fever. Local examination reveals a blue, foul-smelling, swollen, crepitant foot and leg, and it is obvious that this leg will result in his death from anaerobic infection if not soon removed. He will not stand a general or spinal anesthetic, and time will not allow much in the way of expectant treatment.

The leg to the upper thigh is encased in chopped ice to which salt has been added, and supportive intravenous and diabetic care is commenced. After 2 hours a tourniquet is applied to the thigh just distal to the upper refrigeration level, and the refrigeration is continued. One hour later painless low thigh amputation can be performed. Occasionally a slight spasm is felt by the patient when the sciatic nerve is severed, but otherwise the operation is painless and not shock producing.

The disadvantages to this form of anesthesia are the delay in wound healing due to prolonged tissue cooling and the increased incidence of wound infection, but it is undoubtedly lifesaving in the occasional indicated case.

LOWER EXTREMITY AMPUTATIONS

Toes

Toe amputations are indicated chiefly for deformity, trauma, or deficient circulation. Removal of part of the toe should be reserved for the first two indications mentioned and is done, if possible, by a long plantar and short dorsal flap. Circulatory ailments demand removal of the complete toe; this is carried out by an elliptical incision with lateral flaps. The big toe is the most important one, being an essential factor in proper walking and bal-

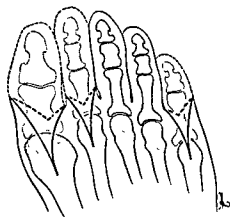


Fig 535—Amputation of toes

ance, and consequently every attempt should be made to conserve as much of this toe as possible.

Transmetatarsal Amputation

Transmetatarsal amputation is frequently used in severe crushing trauma to the forefoot and in those cases of peripheral arterial disease where preoperative tests in a case of painful or gangrenous toes indicate that healing will likely occur at this level. This amputation has supplanted the time-honored Lisfranc and Chopart types where, in the former, disarticulation of the tarsometatarsal joints was done and, in the latter, the foot was removed by section through the midtarsal joint. In the transmetatarsal variety, a longer plantar than dorsal flap is fashioned because of the thinness of the soft tissues on the dorsum. This applies to cases done because of trauma or occlusive arterial disease and is the only exception to the dictum that equal flaps are indicated when the amputation is done for arterial occlusion. The metatarsals are sawn through as close to their bases as possible and all loose tendons are excised. The flaps include skin, subcutaneous



Fig 536—Transmetatarsal amputation

tissue, muscle, and fascia. The only disadvantages to this operation are the interference with balance of this foot and the tendency of the foot to inversion resulting from pull of the tibialis anticus attachment

Syme's Amputation

The only advantage of Syme's amputation over a site of election removal is that end weight-bearing can be obtained with almost the full length of the leg. However, a prosthesis must be worn for walking and it is of necessity an unsightly one with a bulbous ankle and lower leg corset, making it unsuitable for women. This amputation is absolutely contraindicated in occlusive arterial disease because of the poor blood supply to the long plantar flap, and therefore it is used only in

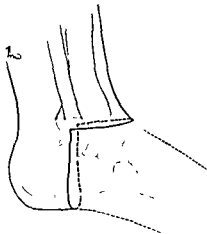


Fig 537—Syme's amputation

cases of trauma. During World War I, the greatest number of Syme's amputations were performed, but the majority of these had to be reamputated at the site of election because of painful or ulcerated stump. However, it should be said that many Syme's amputations have secured lasting excellent service, and it is claimed by the proponents of this type of amputation that the poor results are subsequent to improper selection of cases or incorrect surgical technique.

The tibia and fibula are divided just above the ankle joint through a transverse incision passing anterior to the ankle, and a long plantar flap including all the soft tissues of the heel is swung forward to cover the bone ends and to become the end-bearing surface

Site of Election

This level is probably the most useful in all respects for a leg amputation. It allows a functioning knee joint, and the prosthesis is easily worn and gives a good cosmetic result. Unfortunately, it has a limited application in cases of arterial disease because of the attenuated blood supply below the knee, but the occasional successful result follows a careful preoperative evaluation as outlined in the paragraph on selection of amputation levels.

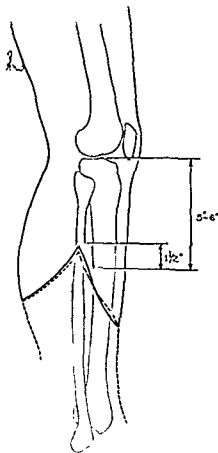


Fig 538—Site of election amputation

It has its greatest usefulness in cases of severe foot trauma. However, the prosthesis must be carefully fitted and the skin over the tibial end must be movable to prevent the frequent complication of pressure sores over this area.

The length of the stump should be measured from the level of the insertion of the hamstring muscles on the tibia and fibula. Between 5-7" of tibia is the ideal, but a useful

stump can be obtained as short as 2". Less than this makes for great difficulty in having the below-knee section activate the prosthesis. When the amputation level is less than 3" of tibia, it is preferable to excise the entire fibular fragment. This prevents springing of the fibula away from the tibia and the more successful achievement of a conical stump. In traumatic cases a longer anterior than posterior flap is cut, but in amputations due to

long anterior and short posterior flaps make for circulatory troubles in the longer flap, resulting occasionally in sloughing of the edges and consequently a delay in healing. Bony union between the cut surface of the patella and the cut distal end of the femur in the Stokes-Griffith type sometimes fails to occur, and in all types the stump end is bulbous due to the

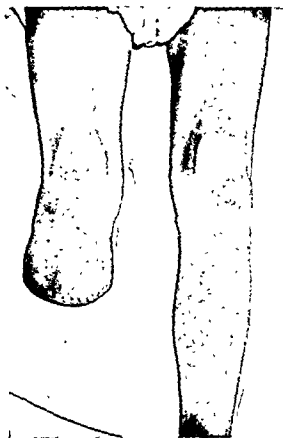


Fig. 539—Recent below-knee amputation because of gangrene of the foot due to Buerger's disease

arterial disease, equal flaps are the rule. These flaps as in all amputations, should be slightly larger than the diameter of the limb.

End-Bearing Amputations at the Knee

With the improvement in the construction of artificial limbs, the necessity of end-bearing stumps for proper walking is becoming markedly diminished. Although eminently satisfactory as a functioning stump, this type of amputation has several disadvantages. The

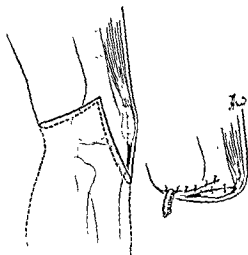


Fig. 540—Stokes-Griffith amputation

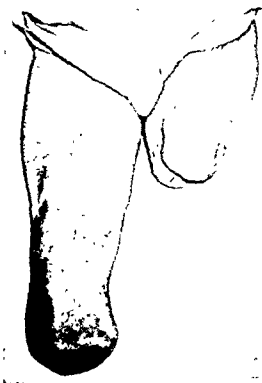


Fig. 541—Previous left midhigh amputation under refrigeration anesthesia and recent right below-knee amputation in a case of diabetic gangrene

flare of the remaining portion of the condyles. This fact added to the longer thigh resulting from the artificial knee joint makes this type of amputation unpopular with the limb maker.

The two main varieties of end bearing above-knee amputations are the Stokes Gritti and the Callander types. In the former, the femur is divided at the upper level of the condyles and the articular surface of the patella is removed. A long anterior and a short posterior flap are necessary. The patella is retained in the anterior flap and the bare surface of the patella is fixed to the cut surface of the femur. Bony union between the two is a necessity. The Callander type is somewhat the same except that the patella is removed from the quadriceps expansion and the cut end of the femur is covered by this expansion.

Thigh Amputations

It is the opinion of most surgeons interested in peripheral vascular disease that when a good site of election amputation is not feasible, the lower third of the thigh is the next best choice. At this level the arterial supply is adequate, long flaps are unnecessary, and wound healing is thereby improved. A good functioning prosthesis can be easily fitted. The majority of amputations done for the complications of obliterative arterial disease will be done at this level.

The optimum level is $2\frac{1}{2}$ " above the knee joint, but an adequate stump is obtained from the middle of the middle third of the femur to this level. Equal flaps can be used, but a slightly longer anterior one is preferable. The subsequent shortening of the stronger hamstring muscles pulls the suture line posteriorly, even when the flaps are initially equal. Suturing of the cut muscle over the bone end is not advisable when it is likely that a prosthesis will be worn because the resulting bulky end of the stump makes for difficulty in fitting a proper socket. However, in patients who are unlikely to wear an artificial limb, this point has advantages in cushioning the stump end. The author prefers to cut the muscles at the level of bone section and retain only the deep fascia and a small amount of muscle with the skin flaps. The nerves are cut as high as possible to keep them clear of the scar. The

sciatic is the only one which should be ligated at the time of division because of the artery that runs within.

The least amount of thigh possible necessary to guide a prosthesis is 6" from the great trochanter, consequently every effort should be made to achieve this length. If a higher amputation is necessary because of trauma or malignant disease, the head and neck of the femur should be retained to preserve hip contour. This is more desirable and is easier than hip disarticulation.



Fig. 342.—Supracondylar amputation.

Hindquarter Amputation (Interinnomino-Abdominal)

This heroic amputation is indicated only in primary malignancies involving the head and neck of the femur or the pelvic bones on one side. It consists of removal of the limb and half the pelvic girdle, after division of the symphysis pubis and ligation of the common iliac vessels on that side. A portion of the sacrum may need removal if the position of

the tumor so demands. The cure rate is understandably low when the operation is done for osteogenic sarcoma but is better in chondrosarcoma and fibrosarcoma.

UPPER EXTREMITY AMPUTATIONS

Fingers

Whether a finger should be amputated after trauma of a crushing type depends upon many individual factors. No hard and fast rule can be laid down, but, in general, it should be

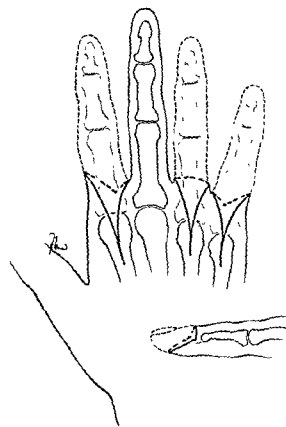


Fig. 543 - Amputation of fingers

said that if there is any possibility of obtaining a moderately functioning finger, then conservatism is indicated. A poor result can always have a subsequent amputation. Because of the frequency of poor results, most large hospitals have organized a Hand Group, who, with their teamwork, especially between the physiotherapist and traumatic and plastic surgeons, achieve better results than those usually obtained. It is in the segregation of these cases under the care of such a well-trained group that the hope of improvement in results rests.

In general, it can be said that amputation is indicated when, due to injury, pain, or sepsis, the finger becomes a menace to the individual, it is useless or actively in the way, or it interferes with the use of the hand. (See also *Amputating Injuries of the Hand and Figs. 641-643, Chapter 39.*)

Following are other points to be kept in mind:

1. The working man requires a strong mobile hand. Stiff fingers interfere with his manual labor and must be avoided. The cosmetic factor is of secondary importance. Therefore, in general, immediate finger amputation is more often indicated in trauma than are prolonged plastic procedures.

2. In children, the most conservative approach is taken because of their wonderful powers of functional restoration.

3. In men and women of the executive type, the cosmetic factor is most important and great strength is a secondary consideration.

4. Tendons should never be sutured over the amputated ends of fingers because this interferes with the function of the other digits.

5. When amputation takes place through joints, the cartilage should be removed to prevent a bulbous and sensitive stump. Cartilage is also more subject to infection, fistula, and sequestration than bone.

6. If amputation is in the region of the nail bed, the nail matrix should be totally removed to prevent the development of horny nail growths.

The level of removal of a damaged digit is open to considerable controversy, and it should be emphasized that the length of a finger should not be sacrificed to obtain skin flaps because such skin deficiencies can be easily remedied by skin grafting. In general, the following statements are widely accepted:

The thumb is by far the most important digit, but it requires parts of the other digits for proper opposition. Therefore the most conservative attitude is required in treatment of its injuries, all possible length is preserved, using skin grafts. All possible length of the other damaged fingers is also indicated for the best possible opposition. (See also Fig. 641.)

The index finger is next in importance. Traumatic amputation of the tip is best treated

by skin grafting to a pedicled graft lifted from the skin of the thenar eminence, or pectoral region, or by a split-thickness skin graft. If more than the distal one and one half phalanges are destroyed, amputation is best through the metacarpal to give optimum cosmetic appearances. This latter point applies only when the thumb is undamaged and should be considered only as a secondary procedure in the laborer.

The middle finger can be amputated at any point because any length of stump adds strength to the hand. Therefore, in the manual worker, conserve as much as possible of all phalanges. In the white collar worker who has lost the majority of this finger, it is probably wise to remove the entire finger and the complete metacarpal. This allows the index finger to take over the function of the middle and gives a much better cosmetic result.

The ring finger can be treated on much the same lines as the middle.

The little finger is of least importance. Amputations are successful at any level, but when most of the finger is destroyed, the head and most of the shaft of the metacarpal should be removed to give best cosmetic results. The metacarpal head is best preserved in the laborer.

Forearm Amputations

Forearm amputations are indicated mostly as a result of severe trauma. Occlusive arterial disease rarely involves the upper extremity to the point of needing amputation. Weight-bearing and strain on the stump in the prosthesis are not factors to be reckoned with, consequently upper extremity amputations rarely are heir to later complications. Equal flaps are therefore indicated. The optimum levels are not more than 7" from the olecranon to a minimum length of 1" beyond the insertion of the biceps tendon. Less than this amount leaves insufficient length for the forearm stump to control the artificial elbow. A disarticulation at the wrist has little to recommend it and is inferior to a 7" forearm stump.

Upper Arm Amputations

The same observations as in the forearm apply to the upper arm. The optimum level

is 8" from the acromion process, and the minimum functioning stump is 1" from the anterior fold of the axilla.

Forequarter Amputation (Interseapulothoracic)

The indications for this procedure are the same as in the hindquarter removal. The arm, including the scapula and all or most of the clavicle, is removed along with the associated muscles, axillary fat, and nodes. The subclavian vessels must be tied early in the operation and the brachial plexus divided.

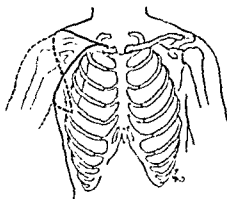


Fig 341—Forequarter amputation.

AFTERCARE OF AMPUTATIONS

It is the surgeon's responsibility not only to do the amputation and assure a good convalescence but also to be certain that the patient is physically and mentally prepared for his artificial limb. Loss of a limb is a great psychic shock to any individual, and the ability of the patient to adapt himself to his artificial limb will depend on his mental attitude toward it. The surgeon can do a great deal in promoting a healthy mental attitude of his patient toward his disability. Early postoperative mobility, occupational therapy, moving pictures of the activities of other amputees, and personal visits to the patient by other adapted individuals with loss of a limb will be a big boost to morale. Certain patients, however, never are capable of wearing a prosthesis, and expensive and misguided efforts in obtaining a limb for such a person should be discouraged. Many persons in the 6th, 7th, or 8th decade never acquire the balance or will power to use

crutches successfully, let alone learn to use an artificial leg. Such patients have usually been bedridden for months before amputation is finally done. Their musculature has wasted and their morale is poor. In most of these patients the remaining limb is also involved by arterial disease, and in about 30% its amputation is necessary within 3 years after the initial one.

Despite the possibility that an artificial limb may never be worn, all amputations should be treated postoperatively as though that possibility did not exist. The cardinal principles of aftercare of the stump are as follows.

- 1 Shaping of the stump by bandaging to control edema and prepare the limb for the prosthesis
2. Exercise of the stump to enable the amputee to use the artificial limb adequately
- 3 Prevention of stump contractures

CINEPLASTIC AMPUTATIONS

The plan of employing individual muscles or groups of muscles to activate a functioning part of the prosthesis was first conceived by Vanghetti in Italy at the close of the last century. A slow development of this technique has taken place but has not been widely accepted because of the inability of the limb maker to produce an artificial limb capable of properly utilizing this surgical advance. However, since the close of World War II, renewed interest has been aroused by the development of improved prostheses, and it is likely that the usefulness of this method will be more appreciated and more widely used.

The upper limb only is suitable for this modification because the finer movements achieved in an artificial hand are unnecessary in the leg. In general, the technique consists of utilization of a muscle or agonistic group of muscles to act on a skin tunnel around which the appropriate tendons have been fixed. This action is transmitted to a rod in the skin tunnel which is connected to activate the artificial fingers (thumb and index). Powerful and delicate voluntary movements can be achieved, allowing for the grasp of any object from that of a match to a hammer. The formation of the so-called "motor" loop can be accomplished at the time of the initial amputation, when infection is not a possibility,

or following the initial amputation. However, if done later, it must be ascertained that the individual muscle has sufficient length and still is an active contractile unit. Intensive training and use of the utilized muscles is a most important postoperative measure.

THE PROSTHESIS

No two amputations are of exact similarity even though of the same type, hence each limb has to be made to order. The correct artificial limb should be applied as soon as possible, but in most cases the stump is not adequately shaped before 4 months. The wearing of temporary "peg legs" or pylons is inadvisable because they lead to incorrect walking habits. Later corrections to the initial socket are always necessary as continued shrinkage occurs. Good fit, lightness, durability, and good repair service from the limb maker are essential points in an artificial limb. The most recent advance in limb fitting is the further development of the original German idea of the suction socket for thigh amputation. In these the stump is held in a close-fitting bucket in which a negative pressure is produced by exhausting the air in the socket through a distal valve when the stump is inserted. This allows more natural walking and dispenses with the pelvic band necessary in the conventional types. Cases suitable for this design must, however, be carefully chosen and must not be those done for occlusive arterial disease.

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
The Cineplastic Method for Arm Amputations (1947)	17 min	Sound Color	Director Armed Forces Institute of Pathology Washington 25, D.C. Attention: Chief, Medical Illustration Service
Amputation for Occlusive Arterial Disease (Amputation under freezing anesthesia) (1948) (By Gerald H. Pratt, M.D., New York)	22 min	Silent Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.
The Diabetic Foot (Transmetatarsal amputation with delayed primary closure) (1953) (By Angus D. McLachlin, M.D., London, Ontario)	23 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.
The Suction Socket Artificial Limb (Realistically appraises the potential benefits of the suction socket prosthesis) (1952)	22 min	Sound	Central Office Film Library Veterans Administration Vermont Ave. and H St., N.W., Washington 25, D.C.
Upper Extremity Prosthetics (Points out many advantages gained by wearing an upper extremity prosthesis) (1952)	22 min	Sound	Central Office Film Library Veterans Administration Vermont Ave. and H St., N.W., Washington 25, D.C.

Chapter 36

Fractures

H. Fred Moseley, DM

A *fracture* may be defined as a breach in the continuity of a bone which is complete or incomplete. Fractures are of many types, depending on the mode of classification:

A Traumatic—when due to an adequate force of:

- 1 Direct violence
- 2 Indirect violence
- 3 Muscular violence

B Pathologic—when largely due to disease in the bone caused by:

- 1 Congenital defects, *fragilitas ossium*, pseudoarthrosis of tibia
- 2 Metabolic errors, rickets
- 3 Generalized bone disease, Paget's disease, hyperparathyroidism
- 4 Generalized osteoporosis, old age, invalidism
- 5 Local inflammation, osteomyelitis
- 6 Neoplasm; benign or malignant

Fractures may be classified according to the absence or presence of a communicating skin wound into (1) closed (simple) and (2) open (compound).

The use of the word *simple* is probably misleading and should be replaced by the word *closed*. Very few fractures are simple to understand or treat. It is well to realize that a patient with a fractured tibia is best regarded as a *patient with a fractured leg* and the problem must be so assessed and treated. Too much emphasis must not be placed on the bone alone; adequate attention must be

given to the soft tissues of the affected part and the patient as a whole in relation to his work and family.

An *open* or *compound* fracture is one in which a wound in the skin and soft parts places the environment in communication with the fracture site.

There are two types of open fracture:

1. Direct—in which the direct violence compounds the fracture from without
- 2 Indirect—in which the indirect violence causes the fractured fragments to compound the soft tissues from within

Birth fractures occur in relation to the mechanics of parturition and may involve any of the long bones of the upper or lower limbs. The bones most commonly fractured are the humerus, clavicle, and femur. Epiphyseal displacements occur in relation to the lower end of the femur and both upper and lower ends of the humerus.

Greenstick fractures occur in children before puberty when the bones are elastic like green twigs and bend rather than break. When the fracture separates at the epiphyseal plate with or without a fragment of metaphysis, the injury is called an epiphyseal separation.

A sprain fracture results from a joint displacement where the ligament avulses its bony attachment in place of rupturing its fibers.

Fractures may be closed or open, complete or incomplete, or complicated by associated lesions of muscle, nerve, or blood vessels.

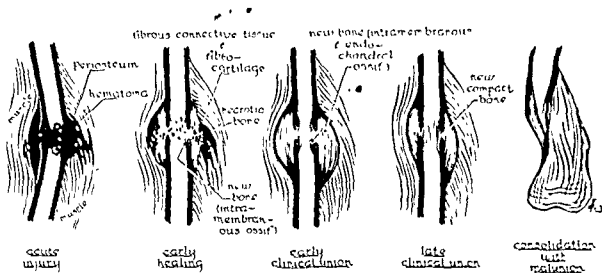


Fig 545—Stages in the healing of fractures (After Utter)



Fig 546—X-ray of clinical union

REPAIR OF FRACTURES

The process of repair follows immediately the occurrence of fracture. A hematoma develops around the broken fragments and injured soft tissues. Traumatic inflammation ensues with hyperemia, edema, resultant swelling, heat, local pain, and tenderness.

The fracture hematoma is widely considered the basis on which the healing process develops. The hematoma is replaced by granulation tissue from which callus, the healing cement of bone, arises. Into this callus, calcium and phosphate ions are precipitated from

periosteal and endosteal aspects, which permits the *permanent* or *definitive* bone to be laid down later in orderly fashion between the compact surfaces.

Clinical union may be said to be present when the fractured bone will move as a unit without pain or local tenderness. At this stage, guarded and graduated stresses and strains will accelerate the further consolidation of the healing by laying down the final architecture according to Wolff's law. As the permanent callus gains strength, the temporary callus absorbs except where necessary as a permanent



Fig. 547.—X-rays showing consolidation of fractured tibia sustained 6 years previously and also a fresh fracture-dislocation of the ankle.

the blood and from mobilized minerals of the bone ends. The two forces directing the course of repair are the vital activities of the osteoblasts in the early and late stages and, after a certain point, functional stresses and strains. The circulatory status of the healing tissues is very important.

The general method followed by Nature is to lay down first a *temporary* scaffold on the

buttress. X-ray, however, at this stage will not show bony trabeculae crossing the fracture line, and the line will still be visible.

Consolidation or final union requires a much longer period and is present when the x-ray shows the restoration of normal architecture with loss of the fracture line and trabeculation across this area completed. Consolidation or final union is therefore today an

x-ray interpretation. Function may be possible for months before this stage is finally reached in fractures of the long bones.

CLINICAL PICTURE

Patients with fractures present a variable picture, from the minor complaints that might suggest merely a sprain or bruise of bone to the severe symptoms of shock seen in open, complicated, or multiple fractures. All require a careful general as well as local examination, bearing in mind the possibility of severe internal injuries in the transport accidents of this age.

In fractures involving the limbs, a careful study of the circulatory and neurologic status must be made. This is especially true in open fractures and fractures associated with marked displacement or dislocation.

In evaluating the clinical picture, the following factors must be considered:

History of Accident.—A detailed account of the accident must be obtained. The exact hour, the method of handling at the time, the manner of transport to hospital, time of arrival in the ward, and operating room are significant from a medical and possibly a legal point of view. The severity of the injury gives an approximation as to what may be expected in bodily damage.

Local Symptoms and Signs of Fracture.

1. Pain—exquisite pain is felt in the region of the fracture. The muscles are in spasm and any movement augments discomfort.

2. Tenderness is noted on palpation of the fracture site.

3. Swelling may be present from extravasation of blood or from displacement of the bone ends.

4. Deformity occurs in the presence of angulation or overlap of the bone fragments.

5. Excessive mobility and bony crepitus will be noted on examination of a complete fracture.

6. There is loss of function.

7. X-ray examination is imperative and gives information regarding the exact injuries and displacement of the bony skeleton. It must always, however, be considered as an accessory to accurate clinical observation of the patient, although elicitation of crepitus and painful examination are rendered unnecessary

EMERGENCY TREATMENT

General.—Therapeutic measures will be determined by the conditions under which the patient is first seen. If attended at the site of the accident, assessment of the severity of the injuries is of prime importance as this will decide the urgency of treatment. In most cases, the ready accessibility of hospital facilities and the rapid means of transportation available permit the delay in definitive measures, such as transfusions, antibiotics, and antitetanic serum, until the patient has been admitted. The patient should be warmly wrapped in blankets and moved with great care. Morphine should be given to diminish pain and psychic trauma. In cases of severe blood loss and shock, transfusions can be started in the ambulance.

Local.—The most important treatment at the site of the accident is to splint the fractured limb securely. All unnecessary movements should be avoided. When gross deformity is present or when the fractured bone end projects from an open wound, it is best to reduce the deformity and replace the projecting bone end using traction and counter-traction. This will improve the circulation to the peripheral part of the limb, and later operative treatment will include removal of the contamination by wound cleansing and excision of devitalized tissue. The wound should be temporarily covered with the cleanest dressing available. When hemorrhage is present, a pressure dressing is applied and the limb elevated. A tourniquet is rarely necessary. If used it should be released every 15 minutes, and only pressure adequate to obliterate the pulse should be applied.

The splints employed will depend on the available materials. Plaster of Paris and Cramer wire can be readily adapted to the various regions requiring protection. A temporary padded cast can be quickly applied after the fracture has been placed in the best possible alignment. Plaster casts are most valuable as temporary splints for the lower arm, elbow, forearm, and wrist and for the lower leg, ankle, and foot. For the shoulder region and upper arm, an encircling bandage around the arm and chest applied with an axillary pad and sling is a comfortable at-

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arrangement for transportation. For fractures involving the hip, femur, knee, and upper two thirds of the tibia and fibula, a Thomas splint arranged for fixed traction with a circular bandage of flannel or plaster of Paris is excellent. An alternative method of splinting when transport over long distances is required would be a plaster spica. In cases of fractures of the lower leg, ankle, and foot, the pillow splint is readily applicable. On arrival in hospital, the patient should be prepared for definitive treatment at the earliest moment.

For the emergency treatment and transport of vertebral fractures, see Chapter 11, Neurosurgery, and Chapter 43, Disorders of the Vertebral Column.

ORGANIZATION OF FRACTURE TREATMENT

An organized approach is required to secure uniformly good results in fracture work. The principles of this organization are as follows:

- 1 Segregation of cases
- 2 Uniformity of treatment with standardization of methods and equipment
- 3 Supervision by the same personnel from the initial to final treatment
- 4 In all cases a planned program for each patient; should include physiotherapy, occupational therapy, and general rehabilitation

FUNDAMENTALS OF FRACTURE TREATMENT

The ideal of fracture treatment is to secure the maximum return of function in the minimum time.

Reduction.—Displacements are reduced to their anatomic position if possible.

Immobilization.—The fracture is immobilized by splinting until united.

Protection.—The part is protected during the period between clinical union and consolidation with graduated return to full function.

Functional Treatment of Soft Parts.—From the earliest moment function of the soft parts is encouraged. The patient is made ambulatory at the first opportunity. Return to light work precedes full duty, and rehabilitation should be an essential part of the treatment.

REDUCTION

Reduction means the restoration of the displaced fragments to as near the anatomic position as possible. Perfect reduction is the ideal, but this cannot always be achieved because of the nature of the injured surfaces, the interposition of soft parts, and the later influence of altered muscle pull, splinting, and gravity. For this reason a knowledge of what constitutes adequate reduction is of the greatest importance.

The age of the patient determines the plasticity of the bone. In infants and young children, the healing power of bone is very great, and often fractures uniting in positions of gross displacement will finally in the course of time be reformed to the shape of the normal bone in the area. This quality of bone diminishes rapidly from birth to old age, but at any age this fact should not influence our desire for perfect reduction. It should, however, be recognized that end-to-end reduction for fractures of the femur in the young is undesirable, as overgrowth in the length of this bone usually results. An overlap of $\frac{3}{4}$ " is ideal.

The Site of Fracture.—Fractures in the shafts of the long bones of the upper limb may heal with overlap and sometimes with angulation without gross interference to function of the limb as a whole. One joint may be disorganized by malunion, but the other joints usually increase their range of function to compensate for this defect. Shortening of the limb by 1" or more is not usually obvious. In the lower limb, however, shortening of degree over $\frac{1}{2}$ " results in inequality of leg length and requires compensation by correction to the shoe. Without this a limp may be present. Any alteration of the normal direction of force through the joints due to angulation is followed by abnormal stresses and strains which, because of the transmission of body weight, lead to wear and tear changes or traumatic arthritis.

If the site of fracture involves the joint surfaces, exact anatomic reduction is required, otherwise the smooth gliding action is lost and, as is found in machinery, rapid deterioration of the mechanism results. Joint injuries frequently require removal of loose fragments of bone in order to prevent the rapid development of traumatic arthritis.

over pulleys balanced by elevation of the foot of the bed, the expression *balanced traction* is employed.

The most common use for continuous traction is in displaced fractures of the thigh or leg. Either the Thomas or Braun splint is used

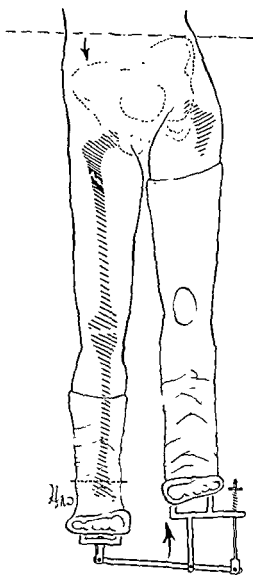


Fig 549—Well-leg traction

Sometimes traction to the affected extremity can be secured by upward pressure on the normal limb through the sole of the foot, by the use of the parallelogram of forces secured by the Roger Anderson apparatus.

This constitutes the principle of *well-leg traction* employed occasionally for fractures of the upper end of the femur.

Distraction and Impaction.—A variation of traction and countertraction methods where

complicated apparatus is involved is the method of mechanical distraction followed by impaction with the use of external fixation for immobilization. The two sets of apparatus most commonly used are those of Roger Anderson and Stader. This method requires considerable experience for successful use and has not gained great popularity because of this difficulty and the dangers of infected pin holes and joint stiffness.

Operative Reduction.—Operative reduction and the associated internal fixation of fractures were introduced by Lambotte and Lane at the beginning of the century. They were dissatisfied with the clinical results obtained in oblique fractures of the femur and tibia in adult patients. The method was correct, but its application until recent years resulted in serious complications due to infection, poor surgical technique, and foreign body reaction.

The present use of this method has been increased by the following considerations:

1. Improved standards of technique and asepsis on the part of the surgeon, his assistants and associated operating room personnel
2. The prophylactic and curative power of the chemotherapeutic and antibiotic drugs
3. The development of inert metal for plates and screws

It is not the method for all fractures and should be used only in selected cases. Its chief danger is still the fact that it converts the closed into an open fracture and invites disaster if complications ensue.

Indications.—

1. When manipulation will fail or has failed because of (a) interposed soft parts, (b) separation of fragments by muscular contraction—patella—olecranon, (c) delayed treatment with malunion, (d) certain intra-articular fractures
2. When reduction cannot be maintained, oblique fractures of femur, tibia, or femoral neck
3. In fractures complicated by injury to blood vessels, or nerves, necessitating their exposure

The guiding principle in the reduction of fractures is to employ the correct method, for the correct case, in the correct manner, at the correct time, and to select when possible the nonoperative procedures.

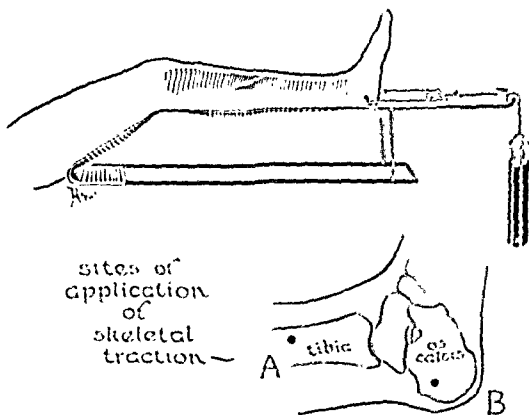


Fig. 551 — Skeletal traction on femur and tibia

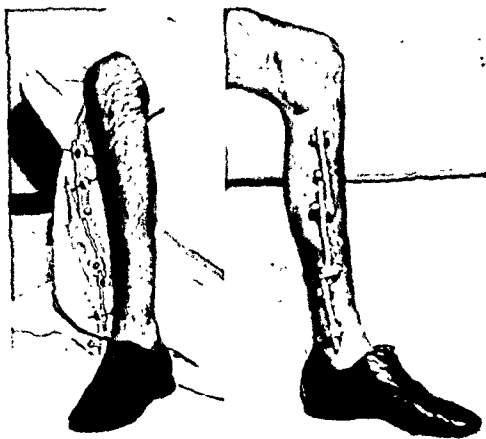


Fig. 552 — External skeletal fixation for fractured tibia

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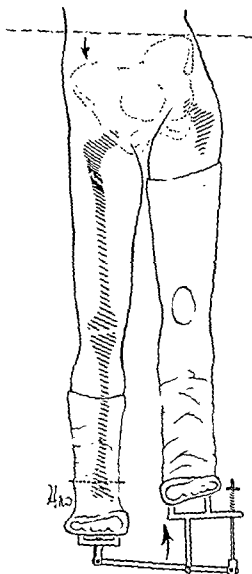


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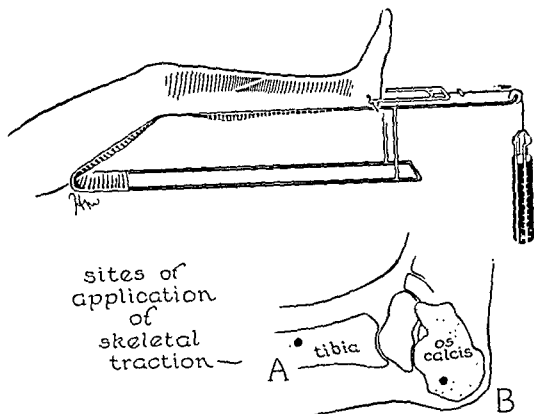


Fig 550—Skeletal traction on Braun splint.

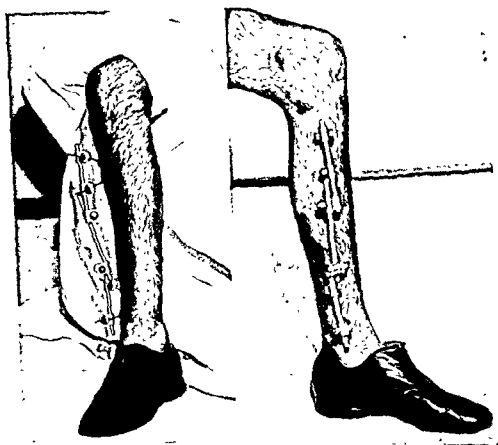


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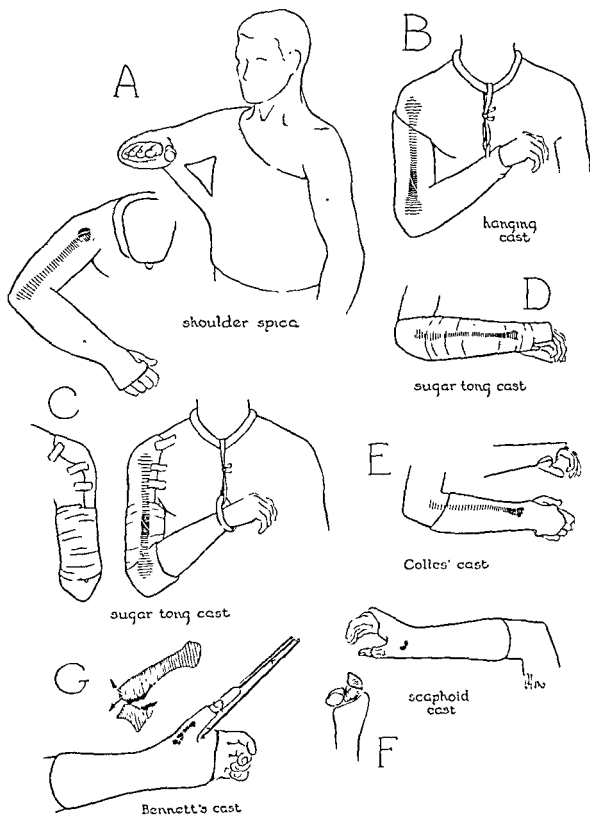


Fig 552—Type casts for fractures of the upper extremity.

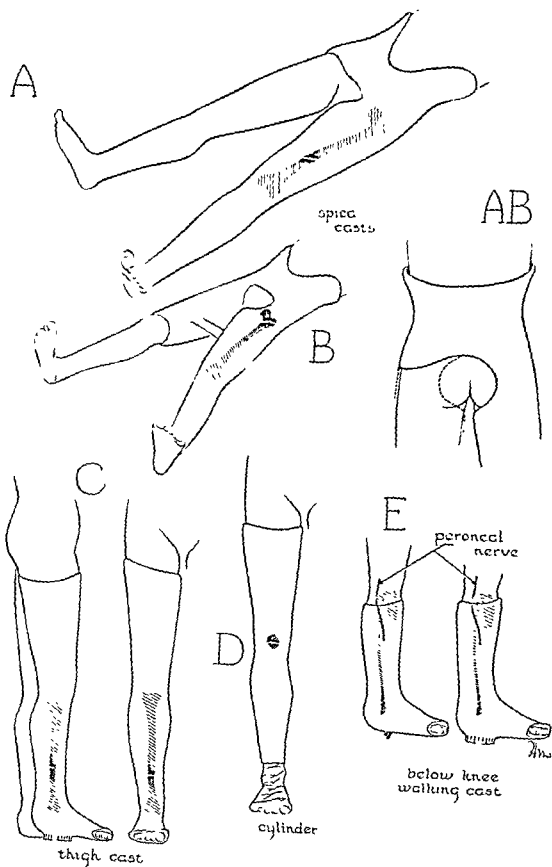


Fig 553—Type casts for fractures of the lower extremity.

IMMOBILIZATION

Plaster-of-Paris Technique

This constitutes the most useful of the methods for the immobilization of fractures. Since the introduction of the plaster bandage by Mathisjen in 1852, the technique has greatly improved, and today casts are applied in such a way as to preserve the maximum function of the part and to permit early ambulation. The most important principle is to apply the lightest possible splint with the parts held in the positions of optimum function. Excessive weight of cast is to be avoided since the requirement is a patient with an attached splint, not a splint with an attached patient.

Method.—The plaster bandage and slab are the building blocks of the finished cast, and in order to standardize the work it is necessary to have uniform materials.

Uniform bandages and slabs must possess:

- 1 A standard width and length
- 2 A uniform amount of plaster applied to crinoline or mesh
- 3 A standard time for hardening
- 4 A minimum of shrinkage during the process of drying

The plaster may be applied directly to the skin. This is called the *nonpadded* technique popularized by Böhler. Most workers cover the limb with stockinet and place a variable amount of sheet wadding over bony prominences before the plaster is applied. This is the more popular method and is called the *padded technique*. The danger of pressure effects on the skin, circulatory mechanism, or superficial nerves of the part is thereby lessened.

The bandage is immersed in water until the bubbling ceases and is then applied evenly without constriction, taking care to smooth one layer against the other and remove all air spaces. After the cast is applied, hardening may be accelerated by the use of an electric drier or baker. A cast that is lighter, harder, and more resistant to moisture and soiling may be obtained by the use of commercial preparations such as Melmac.

If the cast is applied to a fresh fracture and swelling is expected, the circular cast is split to enable this to occur without danger

to the limb. Elevation of the limb is essential. *The patient is taught to contract all muscles possible under the cast and to move all adjacent unsplinted joints.* In the upper limb active use of the fingers is most important. The splinted part is carefully observed for 36-48 hours following the application of the cast to see that no complications occur, such as interference with circulation.

Standard Casts.—

Upper Limb.—

Shoulder spica:

- For avulsion fractures of greater tuberosity
- For arthrodesis of the shoulder
- For ruptures of rotator cuff

Hanging cast:

- For fractures of the lower half of shaft of humerus

For fractures around elbow

Sugar tong cast: For fractures of shaft of humerus

Colles' cast: For fractures of lower end of radius

Sugar tong cast: For fractures of radius and ulna

Scaphoid cast: For fractures of scaphoid

Bennett's splint: Traction with cast for Bennett's fracture dislocation

Lower Limb.—

Plaster spica. For fractures of hip or femur

Thigh cast. For fractures of tibia and fibula including unstable ankle fracture-dislocations

Cylinder. For fractures of patella

Below-knee cast. For first and second degree ankle fractures

Spine.—

Cervical collar and Minerva jacket (see Fig. 722): For cervical injuries

Body jacket (see Fig. 723): For lower dorsal and lumbar fractures

Complications.—

1 *Plaster sores* result from continued pressure on the cutaneous tissues overlying bony prominences. The sacral area, anterior iliac crest, malleoli, heel, and bony points around the elbow are the sites most frequently involved. The thin, emaciated patient confined to bed is subject to these sequelae. The lower edge of the cast may cause ulceration of the skin if the limb distal to this point is swollen.

from dependency. Creases on the deep aspect of the cast from movements during the application may cause subsequent skin ulceration. This is especially frequent over the anterior aspect of the ankle.

2. *Circulatory disorders* are prone to occur if the cast is too tight. This may follow immobilization of the recently reduced fracture with subsequent swelling of the limb. Careful attention must be given to the peripheral pulse and cutaneous circulation. Pain and swelling necessitate relief of the constriction by splitting the cast and elevating the part. The cast may predispose to Volkmann's ischemic contracture in elbow injuries or even to gangrene of the distal part where the circulation is completely occluded for too long a period. Sympathetic block is an accessory therapeutic measure to be considered in such cases.

3. *Neurologic lesions* may occur from direct pressure on nerve trunks. The most commonly involved nerve is the peroneal on the neck of the fibula. This may be damaged by the upper edge of a below-knee cast or by pressure from an unpadded area of the hip spica or thigh cast.

4. *Loss of reduction* may result if the cast is applied with too much padding or when the limb is swollen. In both cases the cast loosens in time. This is obviated by avoidance of excessive padding and change of cast on subsidence of edema.

Traction

In cases in which traction has been used to secure reduction, immobilization may be obtained by the use of continued traction. This is especially useful in fractures of the shaft of the femur.

External Fixation

The use of two pin units and connecting bar can be employed for certain cases.

Internal Fixation

This may be accomplished by the externally applied plate, the Smith-Petersen pin, longitudinally placed Kirschner wires, or the intramedullary nail (Kuntscher). Immobiliza-

tion by internal fixation is usually supplemented by a plaster cast until clinical union is obtained.

PROTECTION UNTIL CONSOLIDATION

A considerable interval of time intervenes between clinical union and final consolidation as demonstrated by x-ray examination. In the upper limb, this protection can be secured by graduating the function of the part and avoiding sudden strain or injury. No special apparatus is required.

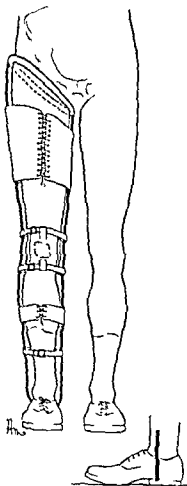


Fig. 554—Walking caliper (Thomas)

In the lower limb, the protection is secured by graduating weight-bearing through the use of crutches and cane. In fractures of the femur, the Thomas walking caliper is an additional support. In fractures of tibia and fibula the caliper or a plaster cylinder may be worn.

FUNCTIONAL USE OF THE SOFT PARTS

The earliest moment after the initial treatment the patient is informed of the extent of his problem and the injury assessed in relation to his possible time in hospital, absence from home and work, and the economic factors involved. His cooperation both mentally and physically is required for efficient treatment, and this can be secured only if he understands the manner in which he can assist the surgeon. The doctor's main thought must be directed toward the problem of the patient as a whole in relation to his environment, and he must try to fit the treatment of the fractured limb to this pattern.

The greatest benefit is usually rendered by assuring the patient that he will eventually make a good recovery and return to full work, that the period required will be considerably shortened if he will actively contract all muscles of the part even though covered by a plaster cast, that active movements of all unsplinted joints and the constant use of the unsplinted fingers are essential.

Encouragement is given by the physiotherapist who supervises these activities and employs heat, massage, contrast and whirlpool baths together with active exercises in his regime.

For patients requiring prolonged institutional care, occupational therapy will keep the mind occupied and stimulate progress.

At the earliest moment, the patient should be made ambulatory and functional use of the part encouraged. Return to light duties should precede heavy routine work. For patients with Colles' fractures properly splinted, typing, driving a car, and such activities are permitted.

In lower limb fractures, most sedentary occupations can be carried out in a walking cast. In severe injuries in the working class, a period in rehabilitation centers should precede return to full duty.

OPEN FRACTURES

In these cases, the principle is to treat the wound in the soft tissues so as to obviate infection and convert the open into a closed fracture.

This ideal may be possible if the soft tissue damage is not extensive and the patient is seen

within 6-12 hours. In civilian practice, with the use of chemotherapy and antibiotics the percentage of successful closures of such wounds is much higher than in the period preceding 1940. Extensive wounds in military practice are best left open and treated by secondary suture.

The initial treatment requires the general care of the patient and measures directed to combat hemorrhage and shock. Following this, a meticulous cleansing of the wound by excision of devitalized skin, necrotic tissue, fascia, and muscle is performed in the operating room under appropriate anesthesia.

Displaced fractures are reduced and immobilization arranged by internal fixation or by external splinting. The wound is closed, if this is possible without tension, relieving incisions may be necessary; otherwise it is lined and lightly packed with fine mesh gauze. Local antibiotics may be placed in the wound. After 5-7 days the pack is removed and the wound is closed by secondary suture or plastic procedure.

Antibiotics are given systemically for at least 6 days. Antitetanic serum is administered routinely.*

COMPLICATIONS OF FRACTURES

The complications of fractures include the immediate associated lesions together with those disorders that develop later as the result of the original accident. Complications may be classified as *immediate*, *delayed*, or *remote* and may be *general* (systemic) or *local* in character.

Examples

Immediate complications

- 1 Traumatic shock
- 2 Radial nerve paralysis with fractured shaft of humerus

Delayed complications

- 1 Fat embolism which is most common 18-72 hours after the accident
- 2 Volkmann's ischemic contracture

Remote complications

- 1 Recumbency lithiasis from prolonged confinement to bed
- 2 Secondary ulnar paralysis in a case which has developed cubitus valgus as a result of an old fractured lateral humeral condyle with premature synostosis

*Gas gangrene (See section on Surgical Bacteriology and Chemotherapy)

Classification.—

General:

1. Traumatic shock (see Chapter 4, Shock and Blood Transfusion)
2. Pyrexia
3. Crush syndrome
4. Fat embolism (see Chapter 5, Preoperative and Postoperative Care)
5. Pulmonary embolism
6. Recumbency lithiasis (see Chapter 31, Genito-urinary System)
7. Bedridden state, pressure sores, hypostatic pneumonia

Local

- 1 Injury to adjacent joints (see Chapter 37, Joints)
 - Joint adhesions and stiffness
 - Synovitis
 - Dislocations
 - Traumatic arthritis
- 2 Complications of union
 - Nonunion
 - Delayed union
 - Malunion
 - Aseptic necrosis
- 3 Injury to muscles, tendons, ligaments, and periosteum adjacent to fracture site, e.g., myositis ossificans, rupture of extensor pollicis longus tendon after a Colles' fracture
- 4 Injury to nerves (see Chapter 11, Neurosurgery), such injuries usually contusions and rarely ruptures
 - Upper limb
 - a Brachial plexus—Fractured clavicle Fracture dislocation of shoulder
 - b Axillary nerve—Dislocation of shoulder
 - c Radial nerve—Fracture of humerus
 - d Ulnar nerve—Fracture dislocation of elbow
 - e Median and ulnar nerves—Involved in Volkmann's contracture
 - f Median nerve—Irritated by anterior dislocation of the semilunar bone
 - g Digital nerves—In compound injuries of fingers
 - Lower limb
 - a Sciatic nerve—In posterior dislocation of hip
 - b Peroneal nerve—In fractures of neck of fibula, from pressure of plaster cast
- 5 Injury to blood vessels (see Chapter 33, Peripheral Vascular Diseases)
 - Arteries
 - a Traumatic arterial spasm leading to Volkmann's ischemic contracture or even gangrene
 - b Arteriovenous aneurysm
 - c Complete rupture
 - Veins Hematoma—thrombosis
 - Vasomotor imbalance—Sudeck's atrophy
- 6 Compound injuries (see Chapter 41, Infection of Bones)
 - Infection leading to osteomyelitis

Factors Predisposing to Joint Stiffness

1. Interference with circulation to the distal part of the limb caused by splinting in a faulty position. For example, splinting a case of Colles' fracture in forced volar flexion in the older patient results in swelling of the hand and fingers with subsequent stiffness.
- 2 Excessive or prolonged traction through joints such as the knee or fingers.
3. Reactionary edema after injury to a limb.
4. Effusions into joints often result from ligamentous damage
5. Infections near joints, e.g., skeletal traction pin sinuses
6. Passive stretchings
- 7 Disuse atrophy.
8. Reflex autonomic imbalance.

Treatment.—

Prophylactic.—

1. Maintenance of functional activity of all unsplinted joints and active contractions of all muscles under casts.
2. Avoidance of splinting in positions other than those of function
3. Use of traction through joints of minimal required force, and only for periods absolutely necessary.
4. Elastic support to prevent edema.
5. Use of sympathetic block to overcome autonomic imbalance.

Curative—When stiffness has developed, the appropriate use of heat, massage, and active exercises is the basis of treatment. The range of movement must be measured and the progress charted to ascertain whether the therapy is beneficial. Forced manipulations and passive stretchings are best avoided.

DELAYED UNION AND NONUNION OF FRACTURES

The time required for the union of a fracture depends on many factors. It is therefore most difficult to define accurately when such healing is too slow.

Delayed union may be said to occur when the average time for the particular fracture has been passed and the x-ray shows decalcification and widening of the fracture line.

Nonunion is present when there is movement at the fracture line when tested clinically and the x-ray shows sclerosis of the bone ends

Factors Which Tend to Delayed Union and Nonunion.—

1. **Inadequate reduction** In certain fractures such as those of the patella or olecranon the fragments may be widely separated. Interposition of soft tissues between the bone ends is frequently found. This may be avulsed periosteum, as found in fractures of the medial malleolus and neck of the femur, or muscle, as in the case of fractures of the shafts of long bones. Failure to oppose and fix the frag-

3. **Excessive traction.** This causes a gap wider than the osteoblasts can readily bridge.

4. **Poor circulation** to the fractured fragments, especially if aseptic necrosis of a fragment occurs. This is most commonly seen in fractures of the scaphoid and neck of the femur where the proximal fragment or femoral head is avascular.

5. **Infection.** This causes fibrosis and interference with the circulation and may also result in faulty immobilization.

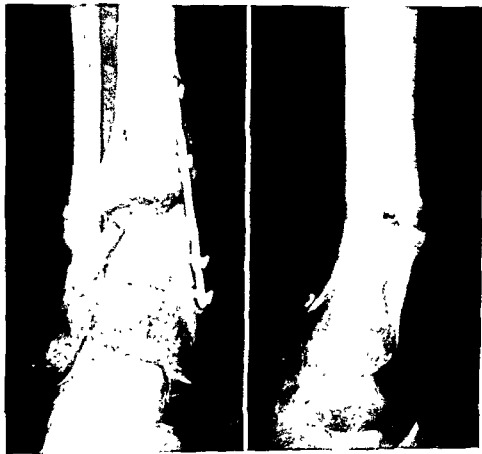


Fig. 555.—Nonunion and malposition of tibia

ments by operative means leads to nonunion. In fractures of long bones a part of the shaft may be missing (e.g., gunshot wounds). Such gap fractures fail to unite unless bone is grafted or unless the ends are opposed.

2. **Inadequate immobilization.** Constant movement at the fracture line creates the conditions required for a false joint or pseudarthrosis. Practically all fractures will unite if properly reduced and immobilized for a sufficiently long period.

6. **Absence of functional use of the part.** Stress and strain both stimulate union if achieved without shearing at the fracture line.

7. **Old age, invalidism, poor nutritional state.**

8. **Pathologic conditions of bone** when the fracture occurs through a malignant growth of primary or secondary nature.

Degrees of Nonunion.—

1. **Absolute nonunion**—a gap exists, e.g., separated fracture of patella.

2. Fibrous union—ends are sclerosed and dense fibrous tissue exists between the fragments.

3. Pseudarthrosis—a false joint has developed.

Treatment.—Delayed union and nonunion are best prevented by correctly applying the

a bone graft to open up fresh vascular channels in each fragment, and at the same time afford internal fixation, is the procedure of choice. Drilling of the sclerosed ends after exposure or freshening the bone ends and application of a metallic plate may be used in certain cases.



Fig 556—Pseudarthrosis of the humerus

proper treatment to the fresh fracture. When delayed union is diagnosed the first consideration is given to the probable causes which have been enumerated, and, if possible, such are corrected. Often proper immobilization of the fracture associated with graduated function will accelerate the healing process.

When sclerosis of the bone ends has occurred and nonunion is established, the use of

MALUNION

When fractured fragments have united in an overlapped, angulated, or displaced position, the fracture is said to be *malunited*.

Causes —

1. Failure of adequate reduction
2. Failure of adequate immobilization.
3. Displacing forces of muscular pull, faulty traction, or faulty splinting.

Treatment.—If sufficiently severe to interfere with adequate function or for cosmetic reasons, the area may be exposed, the bone refractured, and the corrected position obtained.

Immobilization is then secured by internal or external fixation

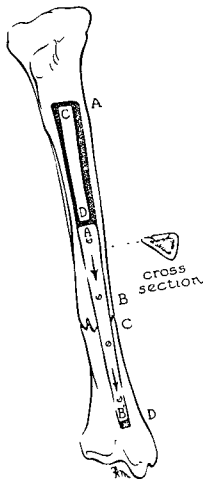


Fig 557.—Sliding bone graft of tibia. The bone graft is cut from the anteromedial surface of the tibia across the fracture line. The portion of the graft below the fracture line is removed, and the remaining bone graft is driven down across the fracture line and appropriately fixed with screws, two above and two below. The residual portion of the graft is placed in the defect in the upper tibia.

MYOSITIS OSSIFICANS

When muscle, ligament, or periosteum is avulsed from its bony attachment, a hematoma develops. In this matrix osteoblasts proliferate and produce bone. Irritation by stress or strain during the early stages increases this bone formation. The most common site for this

development is around the brachialis anticus insertion after dislocation of the elbow.

Treatment.—When the presence of the calcified shadow is seen on x-rays after a joint injury, attention should be directed to prevent any passive stretching of the area. No operative procedures should be considered until the range of movement has become stationary and the x-rays show a dense shadow. If such constitutes a bony block, excision should be considered.

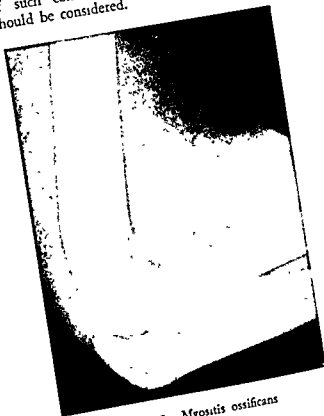


Fig 558.—Myositis ossificans

ASEPTIC NECROSIS

Following a fracture or dislocation, the blood supply to a fragment or area of bone may be less than the nutritional requirements. When such occurs the part undergoes aseptic necrosis. If in a position to be revascularized, the granulations grow into it and the bone is replaced by creeping substitution (Pemberton).

Union of fractures where one fragment is avascular is greatly delayed. This is seen in cases of fractures of the scaphoid with a small proximal fragment, in fractures of the femoral neck with death of the head, and in segmental fractures of long bones such as the tibia.

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procureable From</i>
<i>Splint 'em Where They Lie</i> (Application of fixed traction splints for fractures of the extremities, emergency splinting of spine injuries). (1911) (By New York and Brooklyn Regional Fracture Committee of the American College of Surgeons Boardman M. Bosworth, MD)	23 min	Silent Color	Edgar P. Fleischmann, MD 11 E. 68th St. New York 21, N. Y.
<i>Fractures: An Introduction</i> (1948) (Presented by Committee on Fractures and Other Traumas, Robert H. Kennedy, MD, Chairman. Directed by Harrison L. McLaughlin, MD, New York Advisory Committee. LeRoy C. Abbott, MD, Guy A. Caldwell, MD, R. Arnold Griswold, MD, Paul B. Magnuson MD, A. William Reggio, MD)	33 min	Sound Color	Johnson & Johnson Orthopedic Products Division New Brunswick, N. J.
<i>Principles of Fracture Reduction</i> (1953)	31 min	Sound Color	Central Office Film Library Veterans Administration Vermont Ave. and H. St., N.W. Washington 25, D. C.
<i>The Immediate Care and Transportation of the Injured</i> (1956) (By George J. Curry, MD, Michigan)	20 min	Sound Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.

Joints

H. Fred Moseley, D.M.

The function of joints is to permit movement of the bones that make up the articulation. This movement occurs between the smooth articular cartilages that cover the bone ends. Secretion of lubricating fluid by the synovial membrane is controlled by functional requirements. The range of motion is determined by the shape of the articulating surfaces, the capsular and ligamentous attachments, and the musculature related to the bones concerned. Injury and disease have as their chief complication interference with this gliding mechanism and consequent joint stiffness.

INJURIES

Penetrating Wounds

The superficial joints of the hand, foot, knee, or elbow may be contaminated by puncture wounds or opened by laceration. Foreign bodies such as parts of needles, pins, nails, glass, projectiles, clothes, wood, and dirt may all form part of the injuring force.

Treatment.—The probability of infection indicates the serious nature of these lesions. At the earliest moment, the wound tract should be excised under appropriate anesthesia. Foreign bodies are removed and the joint irrigated with saline. Penicillin in aqueous solution is instilled into the joint cavity. The synovial membrane is closed without drainage. If all contamination has been removed within 6-12 hours of the accident, the superficial

wound may be closed, otherwise it is arranged for secondary suture on the 5th day. A pressure bandage is applied. The limb is splinted in a bivalved cast. Systemic penicillin is given until the possibility of infection has passed.

If the patient is seen later and the joint presents an effusion with local and general signs of infection, aspiration of the fluid and daily instillation of 300,000 units of aqueous penicillin are performed. The bacteriologist determines the causative organisms and the chemotherapeutic or antibiotic agent required. For penicillin-sensitive organisms, penicillin can be injected locally into the joint together with intramuscular administration. Joints which in former years were destroyed by suppurative arthritis can thus be salvaged.

Ligaments

These are the capsular thickenings that prevent abnormal displacements of the bones. The ligament is blended with the periosteum and through this is attached to the bone by dense fibers penetrating along Volkman's canals. Muscular balance prevents excessive tension on ligaments, and rupture does not occur unless these muscles are caught off balance or the force involved is greater than the protecting power of the muscles concerned.

The usual injury is a sprain which may be defined as a rupture of the fibers near the bony attachment. Sprains may be incomplete,

involving only a few fibers, or complete, when all the fibers are ruptured. Sometimes the ligament avulses the piece of bone to which it is attached, giving a sprain fracture.

Treatment.—Incomplete sprains do not cause joint instability, and treatment is palliative. Adhesive strapping arranged to minimize tension on the affected fibers suffices. Cold applications for 6-12 hours, followed by heat, massage, or even Novocain infiltrations, can be used to diminish pain.

Complete sprains and sprain fractures, as they permit joint instability, deserve as serious consideration as fractures and often produce more prolonged disability.

Complete sprains of the external collateral ligament of the ankle should be treated in a walking cast for 6-8 weeks. Sprains of the internal collateral ligament of the knee are usually incomplete, and strapping support suffices. When the ligament is completely torn in association with dislocation or valgus fracture dislocations, operative suture is probably best. Sprain fractures, with displacement of the medial malleolus, require open operative suture. Severe sprains of the wrist must always be differentiated from the fractured navicular by careful radiologic studies.

Subluxations and Dislocations

When ligaments and capsule are sufficiently torn to permit joint instability, the articulating surfaces may displace partially or completely on each other. Partial displacements are usually self-reducing and are called subluxations. When complete separation of the surfaces occurs, the bones are locked in the displaced position by muscular spasm, and these complete persistent displacements are called dislocations.

If the initial treatment does not secure capsular and ligamentous healing, the instability persists and the joint is subject to recurrent subluxations or dislocations. The shoulder most frequently presents this clinical syndrome.

Occasionally dislocations are compound in nature or associated with a fracture. Damage to nerve or blood vessel may occur at the initial injury or result from forcible manipu-

lative procedures. Pathologic dislocations occur when tense effusion and inflammation destroy the ligaments and joint stability, thus permitting gravity and muscle imbalance to displace the component bones.

Treatment of Acute Dislocations.—Early reduction under anesthesia affording complete relaxation of muscular spasm is indicated. The part should be protected until healing of the ligaments is secured. Re-education of joint movements and musculature is then essential.

Internal Derangements

Injury and disease may result in the presence of tissue which intrudes between the joint ends. Such tissue may be unorganized, as is found in chronic infective synovitis with formation of fibrin concretions called melon or rice seed bodies, or may be organized tissues of fibrocartilage, articular cartilage, or bone.

When completely detached, these fragments are called loose bodies and give rise to recurrent effusions, joint locking, and instability. By far the most common internal derangements occur in the knee from rupture of the semilunar cartilages.

Traumatic Synovitis and Hemarthrosis

Joints which are subjected to a direct blow or a severe sprain become painful and filled with an excess of thin synovial fluid. This condition is called traumatic synovitis and the collection of fluid a joint effusion. If the ligaments are torn or a fracture into the joint is present, hemorrhage may fill the joint, giving an hemarthrosis. Unless severe tissue damage is present, the blood remains fluid. Both these conditions may follow joint operations.

Treatment consists in aspiration of tense effusions and application of pressure dressings. In the knee and ankle, protection from weight-bearing is advisable, but joint and muscle exercises assist absorption and accelerate recovery.

Traumatic Arthritis

When injury to the bones or articular cartilage leaves a residual mechanical derangement

JOINTS

of a joint or when capsular and ligamentous tears give permanent instability to the articulation, the wear and tear processes are accentuated and gradual destruction of the joint mechanism ensues. This destructive process is called *traumatic arthritis*.

Traumatic arthritis is most commonly seen in the weight-bearing joints but especially occurs in the closely fitting joints such as the ankle and elbow. Aseptic necrosis of articular fragments such as the femoral head and proximal half of the scaphoid leads to this deterioration if protection from stress and strain is not secured during the revascularization of the ischemic fragments.

Treatment.—Traumatic arthritis is minimized by accurate reduction of fractures involving the articular surfaces together with the removal of loose fragments of bone or cartilage from the joint cavity. Avoidance of function by splinting is required in simple injuries of joints for a period of 2-3 weeks, but in cases with aseptic necrosis the time factor will be governed by radiologic evidence of revascularization.

INFECTIONS

Bacteria may be carried to joints by the blood stream, introduced directly by wounds, or invade from adjacent bone or soft tissue infections.

Acute Pyogenic Arthritis

The bacteria most commonly found are streptococci, staphylococci, pneumococci, and occasionally *Salmonella typhi*. These reach the synovial membrane by the blood stream and initiate an inflammatory reaction. A seropurulent exudate develops which gradually increases in quantity, distending the joint cavity. The limb takes up the position of comfort, i.e., that of maximum joint capacity; e.g., knee flexed 20 degrees. The tension and inflammatory involvement of sensitive tissue result in great pain which is evoked by the slightest movement. Intensive muscle spasm is Nature's protective response.

Clinical Picture.—The patient presents the general reaction to infection with raised temperature, pulse rate, and leukocytosis. The degree varies with the severity of infection.

Locally the joint is warm, swollen, and tender. All movements are resisted by the patient.

Diagnosis.—History of infection elsewhere, such as otitis media, tonsillitis, skin infections, or pneumonia, suggests the site of origin and type of bacteria involved. Aspiration of the joint with microscopic examination and culture is essential.

Treatment.—Bed rest and general supportive measures are indicated. Aspiration of the joint fluid and instillation of 1 million units of penicillin should be performed daily until the correct antibiotic has been ascertained. Intramuscular penicillin is also given. Systemic and local antibiotic therapy should be continued until resolution occurs. The limb is splinted in the position of greatest comfort.

When the infection has been overcome, gradual restoration of function can be obtained with physiotherapy.

Gonorrheal Arthritis

Infection of joints and synovial sheaths of tendons and bursae occasionally occurs and then usually in the 3rd week after genital tract infection in the adult. Spread is by the blood stream. Such involvement is infrequent since the advent of penicillin.

The clinical picture differs from that caused by streptococci and staphylococci in the lesser degree of toxemia and systemic reaction. Locally the joint signs are similar. The process may involve several joints.

The arthritis is rapidly cleared by penicillin which has replaced previous therapies such as treatment by hyperpyrexia.

Chronic forms of neisserian involvement of joints are associated with chronic synovitis giving recurrent effusions and with a form of articular rheumatism.

Tuberculous Arthritis

Arthritis caused by the tubercle bacillus is one of the most serious and disabling types of joint disease. Before the pasteurization of milk and herd testing, the bovine bacillus was a frequent invader of joint tissues.

The bacilli reach the region by the blood stream from a primary focus in lymph gland

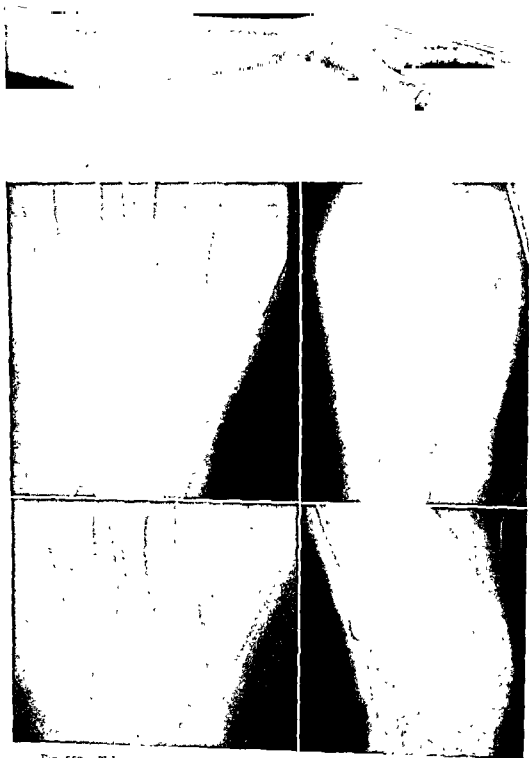


Fig 559 —Tuberculous wrist

Top, clinical appearance, center, x-ray appearance, bottom, after bone graft

or pulmonary tissue. The initial lesion may be in the juxta-articular bone with secondary involvement of the synovia or it may start in the lining membrane. An indolent, slowly progressing granulomatous thickening of the membrane occurs. The cartilage is eroded and sequestered by the carious process, and the ligaments are destroyed. Muscle spasm develops early and joint stiffness presents on clinical examination.

Diagnosis.—The patient may present the general picture of tuberculosis with loss of weight, afternoon elevation of temperature, and night sweats. If seen early, the joint signs,

shows marked osteoporosis and loss of joint spacing.

Treatment.—

General.—The joint process is only part of the systemic disease and the general treatment of tuberculosis is essential. Long periods of observation in tuberculosis centers are necessary, with supportive therapy and training in the way of life required. Streptomycin has been found useful in joint tuberculosis and is given in a dosage of 1 Gm twice weekly associated with a daily dosage of 12-20 Gm. of para-aminosalicylic acid or one of the isonicotinic acid derivatives such as isoniazid



Fig. 560—X-rays of tuberculous hip—early and late stages. Note the osteoporosis involving the acetabulum, head, and neck of the femur with loss of the joint space in the early stage. In the late stage the joint has been destroyed and bony ankylosis has occurred. The bone healing is indicated by the increased density of the bone as shown by x ray.

such as a limp when tired if a lower limb joint is involved, are minimal. X-rays at this stage may be negative. Tuberculin tests are positive. There is a relative lymphocytosis and raised sedimentation rate.

With progress of the disease, the joint becomes swollen, and its movements are restricted. Local heat is not obvious. In the knee the thickened synovial membrane and enlarged joint contrasted with the wasted thigh and calf give the classic appearance to which the term "white swelling" is applied.

Aspiration of the joint with guinea pig inoculation is necessary for diagnostic purposes. In doubtful cases biopsy is indicated. X-ray examination in the fully developed case

A course of treatment covers, on the average, a period of 60-90 days. When secondary sinuses are present, local application is utilized as well. Closure of sinuses can now be expected with this treatment.

Local.—The principle of treatment for joint tuberculosis is rest. In the young, conservative measures employing casts and braces are used. In the adult, resection of the joint and arthrodesis are indicated during a period when the bodily reaction to the disease is favorable as indicated by normal temperature, pulse, and gain in body weight.

Syphilitic Arthritis

The incidence of syphilitic disease of joints has been greatly diminished by active public

health programs and free medical treatment. Cases, therefore, are rarely seen, but clinical types must be kept in mind for differential diagnosis.

The *early stage* is sometimes associated with arthralgia involving several joints, especially the knee, and may be accompanied by a chronic form of joint effusion. General anti-syphilitic treatment affords relief.

bilateral and symmetrical and lacks the general systemic toxemia seen in tuberculosis. Biopsy of the synovial membrane may be required.

Gummatous synovitis is also seen in children 8-14 years of age who show the stigmas of congenital syphilis, such as interstitial keratitis, saddle nose, and Hutchinson's teeth. The process is usually bilateral and involves the knees in a painless effusion.



Fig 361—X rays of Charcot's knee. Note the irregular calcification of the soft tissues in the lateral view. A marked effusion must be present to displace the patella so markedly. In the antero-posterior view the joint laxity is indicated by the loss of accurate apposition of the femoral condyles and tibial plateau.

The *late stage* presents the gummatous process in the tissues and this may cause an infiltration of the synovial membrane, giving an indolent form of peri-arthritis.

Diagnosis.—This is made with recognition of the systemic disease. The Wassermann reaction is positive. The process must be differentiated from tuberculous synovitis. It will be noted that the syphilitic process tends to be

The name *Clutton's joints* is given to this condition.

Painless nodules occur quite frequently around joints. These juxta-articular nodes vary from pea to walnut size.

Treatment.—This consists of general anti-syphilitic measures. Relief from weight-bearing and splinting limits excessive function.

Charcot's Joints

Patients with tabes dorsalis and syringomyelia may present a rapid, painless destruction of one or more joints. In both of these diseases the spinothalamic tracts are destroyed, with loss of pain sensation. This is regarded as the causative factor in the joint changes since injuries are not associated with pain, and protective muscular action is absent. Syphilitic arteritis in the vessels supplying the joint probably also predisposes to the degenerative changes.

The joints most frequently involved are the knee, hip, ankle, shoulder, and spine.

Clinical Picture.—This is made on the above findings in addition to the presence of the neurologic signs of tabes or syringomyelia.

VARIOUS TYPES OF ARTHRITIS

Degenerative or Osteoarthritis

This is the most common form of joint disease and is the result of the wear and tear processes of daily life. The inherited constitution and vitality of the joint tissues are determining factors in its occurrence since the process runs in families. Injury, excessive weight, and chronic postural strain all play a part. It is characteristically seen in the patient over 50 years of age and most commonly involves the weight-bearing joints, i.e., knee, hip, and spine.

Pathology.—The soft tissues of the joint such as the ligaments and synovial membranes show the most marked changes. The ligamentous attachments are frayed and the synovial membrane thickened and hypertrophied into villous fringes. The cartilage over the weight-bearing or pressure points becomes fibrillated and gradually sequesters into the joint, giving rise to loose bodies and recurrent effusion. Gradual deterioration of the whole joint then ensues.

Diagnosis.—Clinically the joint presents the signs of an irritative process. The movements are restricted in all directions and pain is elicited on manipulation. The knee takes up a position of flexion, while the hip becomes flexed and adducted with some external rotation. X-rays show typical *lipping* and possibly

loss of joint spacing. Examination of the hands may show the characteristic Heberden's nodes.

Treatment.—In the early stages, protection from excessive function, with diet to cause loss of weight in the obese, will help. Salicylates and physiotherapy relieve. Intra-articular injections of 1-2 ml. hydrocortisone acetate have been found beneficial, especially for the knee. When loose bodies form, arthrotomy with removal of debris sometimes gives excellent results. In late stages, orthopedic procedures such as arthroplasty or arthrodesis may be required.

Gouty Arthritis

Uric acid deposition in patients with gout may occur in any articulation. The great toe joint is the site of election. The knee, hip, lumbar spine, and elbow (olecranon bursitis) all may present acute or chronic involvement.

Treatment.—Colchicine 0.5 mg. hourly till diarrhea occurs is most effective in the *acute attack*. Intravenous injection of 3 mg. colchicine gives dramatic relief. Butazolidin 800-1,200 mg. daily is also valuable. Cold applications and protection from pressure or tension are fundamental in the local therapy. The patient with a chronic case must be advised to moderate his diet which should be low in fat and red meat. Alcoholic beverages are prohibited. Salicylates and a maintenance dosage of colchicine, 5 mg. b.i.d., are beneficial in preventing further attacks. Benemid 1 Gm t.i.d. is also useful in increasing the excretion of uric acid. Moderation in food, drink, work, exercise, and sexual activities will be found the best way of life.

Rheumatoid Arthritis

These cases are seen on the surgical wards only in the late stages, presenting the deformities of chronic polyarticular disease.

The characteristic fusiform swelling of the proximal phalangeal joints, the flexed swollen knees, flexed and adducted hips, and plantar flexed feet from ankle involvement are usually present.

Treatment of a surgical nature is directed to orthopedic procedures such as correction of



Fig 564—X-rays of degenerative arthritis of knee with loose bodies, patella and loose bodies removed from knee

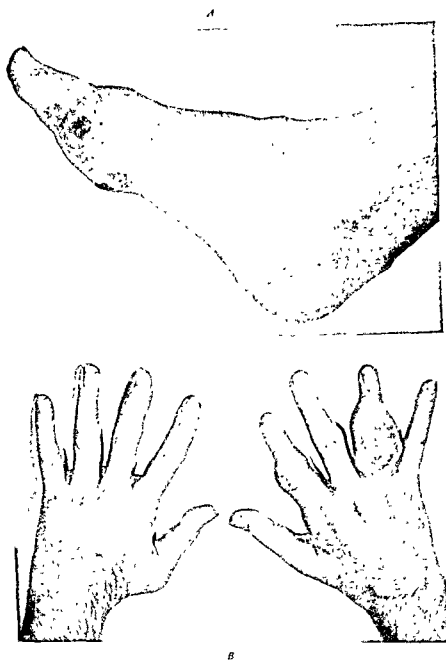


Fig. 565 —*A*, Advanced gouty arthritis of great toe joint with sinus formation
B, Similar changes involving the joints of the hands

deformity by manipulative measures and establishment of the capacity for ambulation by reconstructive and arthrodesing operation on the weight-bearing joints involved. The use of gold, cortisone, and ACTH, together with active physiotherapy, has greatly diminished the frequency of serious deformities.

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Film Reference

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Gout and Gouty Arthritis (Teaches current knowledge on history, etiology, diagnosis, and treatment of gout and gouty arthritis) (1954) (John H. Talbot, M.D., Buffalo, and Alexander B. Gutman, M.D., New York)	27 min	Sound Color	Sharp & Dohme 640 N. Broad St Philadelphia 1, Pa

Chapter 38

Bursae

H. Fred Moseley, D.M.

The word *bursa* is derived from the Latin *bursarius* and signifies a sac. Such bursae or sacs are formed of a fibrous outer membrane and are lined by a layer of mesothelial cells which secrete the lubricating fluid corresponding to the synovial fluid in joints.

Bursae are found in constant sites in the human body and function to diminish friction and obviate pressure where one plane of soft tissue, such as skin or tendon, glides over another plane which usually presents a prominence of bone or tendon.

The *constant bursae* are those shown in Fig. 566, but bursae may develop as the result of a deformity that produces a prominence over which the soft tissues must move or as the result of an occupation that subjects a certain area to repeated pressure and movement of soft tissue over the underlying skeletal parts. Such occasional sacs are called *adventitious bursae*. Some bursae communicate with joints, such as the semimembranosus bursa behind the knee. This has been described as a posterior herniation of the synovial membrane. Other bursae may communicate with joints as the result of attritional changes in the intervening tendon or capsule. The best examples of such lesions are found in the subacromial, iliopsoas, and prepatellar bursae.

Chronic bursal disease often necessitates surgical removal. This is followed by reformation from the residual bursal and adjacent areolar tissues in response to functional activity of the parts.

TRAUMATIC BURSTITIS

Trauma affects the bursal mechanism in the same variety of ways and with the reactions corresponding to those described for joints.

The superficial bursae such as the olecranon and prepatellar bursae may be injured by a penetrating wound. Such open or compound injuries will be diagnosed by the presence of the wound, which on examination leads to the lumen of the bursa and shows the discharge of mucinous fluid.

Treatment includes the careful excision of the wound edges and primary suture if seen within the period before infection has supervened on the contamination. The part should be locally splinted to secure rest and penicillin given to prevent the development of infection.

If the patient is seen after the infective process has developed in the wound, the bursal wall will be thickened and a seroputulent discharge will be present.

Treatment at first should be conservative with local rest by splinting of the part. The discharge should be studied as to its bacterial content and antibiotic sensitivity. Penicillin is best given immediately and changed to the appropriate antibiotic if the organism is not penicillin-sensitive. Some cases will require excision of the chronically infected bursa when the active infection has been controlled.

Trauma may also set up an acute traumatic bursitis as a result of a single contusion or from repeated unusual pressure and friction.

A direct fall on the point of the elbow or knee may initiate the process in the olecranon or prepatellar bursa. Working in the supine position under a car or aeroplane with pressure on the elbows and scrubbing floors on the knees are common causes.

The irritated bursa fills with serous fluid containing more or less blood, depending on the severity of the injury. In the contusion injuries, fissured fractures of the underlying bone or rupture of the tendon attachment or periosteum may be present

villous processes of the mesothelial lining develops. There is an excess of fluid present which is more serous in character than normal. The bursa tends to fill up and becomes painful on overuse. In time, loose bodies of a fibrinous or even calcified nature may form, and pressure on the area may be extremely painful. The bursa involved is subject to recurrent attacks of pain and the muscles in the area may exhibit painful protective spasm.

The treatment of the chronically thickened bursa presenting recurrent attacks of pain and

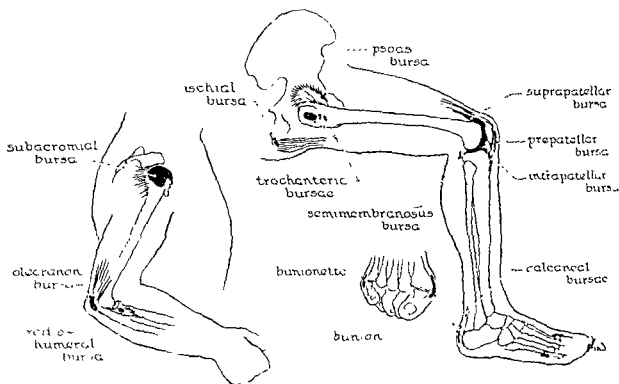


Fig 566—Bursae of the upper and lower limbs

Treatment consists of rest to the part with application of cooling lotions for the first 8-12 hours to diminish the traumatic inflammatory process, followed by heat and massage and gradual resumption of function to accelerate the absorption of the inflammatory products. If the bursa is tensely swollen, aspiration of the fluid followed by a pressure dressing to diminish reformation accelerates the recovery and affords relief of pain.

Although an acute trauma frequently produces bursitis, chronic repeated trauma of lesser degrees is the most common factor. The changes in the bursal wall are more gradual, and in time a thickened fibrous wall with

swelling is extensive. Conservative measures such as rest and protection from injury are justifiable in the early stages. Aspiration of the fluid is usually followed by recurrence. Instillation of 1 ml. (50 mg) hydrocortisone acetate is sometimes of value.

INFECTIVE BURITIS

Pyogenic infections of bursae may be primary and result from direct introduction by penetrating wounds or may be metastatic due to hematogenous spread in cases of septicemia. Antibiotic treatment is usually adequate, and drainage by aspiration is rarely required.

Gonococcal infection occurs secondarily to a genital tract infection and is controlled by penicillin.

Tuberculosis may develop in bursae, usually by spread from adjacent bone or joint lesions. The trochanteric bursa is a typical site for such a tuberculous lesion, and radiologic examination will reveal changes in the underlying bone once the disease is well established.

Syphilitic bursitis is much less commonly seen today because of the early efficient treatment of the systemic disease. It is characteristic of the tertiary gummatous stage, and the bursae most frequently involved are the prepatellar bursae. The process is typically bilateral and symmetric.

The diagnosis is suggested by the presence of bilateral thickened bursae which are typically painless and have developed without adequate traumatic cause. The serologic examination and clinical history will establish the presence of syphilis.

Treatment consists of the systemic treatment of syphilis.

METABOLIC BURSITIS

In this group of cases may be included, for descriptive purposes, bursitis due to deposits of uric acid and its derivatives and to deposits of the phosphate and carbonate of calcium.

Systemic gout with the recurrent precipitation of uric acid around joints leads to attacks of acute pain which are most frequent in relation to the great toe joint and related bursa. Sudden pain in relation to the olecranon bursa with diffuse swelling without apparent cause is sometimes due to gout.

Deposits of calcium phosphate and carbonate are most typically seen in the rotator cuff of the shoulder with secondary involvement of the subacromial (subdeltoid) bursa, although as in gout any joint or bursal mechanism may be involved.

In the cases of lesser severity of involvement by deposits of uric acid and of the phosphate and carbonate of calcium, we have the explanation for many syndromes loosely diagnosed as rheumatism, arthritis, neuralgia, and neuritis.

BURSAE OF THE UPPER LIMB

Three constant bursal mechanisms of the upper limb commonly present clinical syndromes. In order of frequency, these are subacromial (subdeltoid), olecranon, radiohumeral.

Subacromial (Subdeltoid) Bursitis

Involvement of this bursa is the most common cause of shoulder pain, and this is the "bursitis" so frequently discussed in the lay press.



Fig 367—A calcified deposit which has ruptured into the subacromial bursa.

Knowledge of this entity was disseminated by Codman, who demonstrated that the bursa is secondarily involved by inflammatory processes due to trauma, degeneration, and calcified deposits in the subjacent rotator cuff. The bursa functions as a secondary joint mechanism between the humeral head and the coracoacromial arch, and this gliding mechanism is necessary for the movements of rotation and elevation of the arm above the horizontal plane.

Contusions of the area from falls on the upper limb, degenerative changes, and calcified deposits in the underlying cuff produce irritation of the sensitive bursal lining. Depending on the severity of the inflammation

are the intensity of pain and limitation of function

In the typical case due to calcium deposition, the patient gives a history of recurrent rheumatic pain on change of weather or overuse. The acute attack develops suddenly with inability to raise the arm, and sleeping on the affected side is impossible due to the severe pain. X-ray examination will indicate the presence or absence of calcium deposits

Treatment.—Conservative measures include support in a sling and rest to the part. Cooling applications or an ice bag affords more relief in the acute phase than heat. Analgesics are

pulley exercises. X-ray therapy has many advocates

In the chronic cases with and without the x-ray presence of calcium deposits, the use of diathermy, exercises, and the protection by wool garments in cold weather are advocated

Olecranon Bursitis

Acute, recurrent, and chronic forms are seen and are related to varying occupations subjecting the elbow area to repeated friction, pressure, or contusion. Miners, students, and mechanics are especially afflicted. The acute

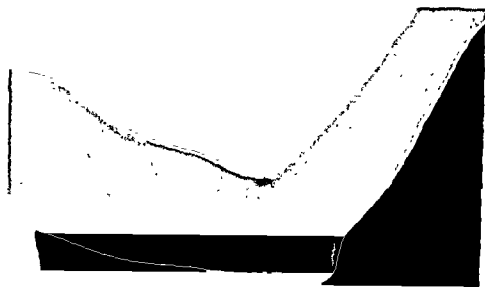


Fig. 568 —Chronic olecranon bursitis

required for pain. If not contraindicated by a history of peptic ulcer, ulcerative colitis, severe diabetes, tuberculosis, or psychotic states, Meticorten 10 mg. q.i.d. for 4 days, followed by 5 mg. q.i.d. for several days, has been found most efficacious when combined with local therapy. Needling with local anesthetic infiltration and injection of hydrocortisone (compound F) afford benefit in some cases.

If x-rays show a large deposit of calcium, surgical excision during the acute phase is best. Proper treatment for small deposits is a course of physiotherapy, including heat and

forms subside with rest, but the recurrent and chronic types frequently necessitate excision of the bursa

Radiohumeral Bursitis

This bursa is situated between the common extensor origin and the supinator brevis. Irritation of this mechanism by excessive pronation and supination is one cause of "tennis elbow." Rest, heat, and salicylates usually afford relief. Infiltration with local anesthetic followed by injection of 1 ml. of hydrocortisone (compound V) sometimes produces dramatic results.

BURSAE OF THE LOWER LIMB

The constant bursae of the lower limb are more frequently subjected to trauma than those of the upper limb. The bursae commonly involved are ischial, trochanteric, semimembranosus, metatarsophalangeal, psoas, prepatellar, calcaneal.

Ischial Bursitis (Weaver's Bottom)

A large bursa exists between the gluteus maximus and the ischial tuberosity and hamstrings origin. Constant friction and pressure from occupations involving the seated position for prolonged periods may initiate a bursitis. The chronically distended bursa may produce a swelling of considerable size. Treatment is excision.

Psoas Bursitis

A bursa exists in relation to the anterior capsule of the hip joint and communicates with the joint in certain cases of degenerative and tuberculous arthritis. Chronic enlargement of this bursa is thus a lesion for differential diagnosis in swellings of the groin.

Trochanteric Bursitis

Several bursae exist in relation to the great trochanter of the femur. The large bursa deep to the tendon of the gluteus maximus may be acutely involved by calcified deposits in this area. Tuberculosis of the adjacent bone may cause a chronic process in this bursa.

Prepatellar Bursitis

This type was frequently seen in the domestics and cleaners of a previous generation. The acute form was presented by the uninitiated worker who attempted to scrub a large area from a fixed position. Today, with modern apparatus, "housemaid's knee" is seldom seen. Bilateral disease of chronic nature with grossly thickened bursae should suggest syphilitic involvement. Serologic examination, if positive, will confirm the diagnosis, and systemic treatment is curative.

The treatment of the chronically inflamed bursa is excision. The acutely inflamed bursa will subside with rest.

Semimembranosus Bursitis

This form of bursitis may be the cause of leg pain with stiffness in the movements of the knee. Examination of the popliteal space will reveal an oval tense swelling which disappears on flexing and becomes prominent on extension of the knee. It is typically seen in young boys 10-15 years of age, and if causing sufficient symptoms, it is best excised.



Fig. 569—Arthrogram, using Diodrast, showing communication of popliteal cyst with knee joint.

In older patients with arthritis or an internal derangement of the knee such as a posterior meniscus tear or chondromalacia of the patella, the bursa, if in communication with the joint cavity, will act as a blow valve and distend when the joint presents an effusion. Such distended bursae should not be excised. Treatment should be directed to the joint disease.

Excision of the grossly enlarged bursa causing pressure in the calf is indicated in

cases of extension of the disease process from rheumatoid arthritis involving the knee.

Calcaneal Bursitis

Bursitis may involve the bursa between the Achilles tendon and the bone or the subcutaneous bursa over the posterior prominence. Both types are due to ill-fitting shoes, and correction of this problem and protection from further injury suffice.

Metatarsophalangeal Bursitis

Inflammation of the bursa over the great (bunion) or small toe joint (bunionette) is a common complaint from ill-fitting shoes. Because of style and fashion, female patients predominate. Most disorders are chronic. Treatment must be devoted to correction of the chronically deformed structures by orthopedic procedures. Properly fitted shoes must be prescribed.

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Title	Running Time	Sound or Silent	Procureable From
Calcified Deposits in the Rotator Cuff (1948) (By H. F. Moseley, D.M., Montreal)	30 min	Silent Color	American Cyanamid Co. Surgical Products Division Danbury, Conn.

Fractures and Other Disorders of the Upper Extremity

Fractures and Other Disorders

H Fred Moseley, DM

CLAVICLE

The clavicle is so called from its resemblance to the clavica of the musical score. It is highly developed in man but may be congenitally absent, i e, cleidocranial dysostosis. This bone is absent in certain running and jumping animals, e g, the horse.

Functional Anatomy.—The clavicle functions as:

- 1 A strut to keep the glenohumeral joint clear of the trunk, thus increasing the range of the hand as the effector organ of grasp
- 2 A rigid attachment for powerful muscles
- 3 The only bony connection of the upper limb and trunk.
- 4 A protection to the vital structures in the cervicobrachial and thoracobrachial outlets
- 5 A cosmetic factor at the base of the neck

Incidence.—Fractures of this bone are four times more common in the male than in the female. Sites of fracture. body—outer end—inner end

Mechanism—The usual cause is the transmission of force indirectly from a fall on the outstretched hand, elbow, or point of the shoulder. Fractures occasionally result from direct violence.

Fractures of the Body

The body is the area most frequently fractured. This portion marks the junction of the curvatures and has little protection from muscular or ligamentous attachments. Greenstick fractures are common in young children.

In complete fractures, the sternomastoid draws the inner fragment upward, whereas the weight of the limb causes the outer fragment to be displaced downward, forward, and medially.

Diagnosis.—This is made on the history, the point of local tenderness or deformity, the localization of pain and crepitus to the clavicle on movements of the arm, and the positive x-ray findings.

Principles of Treatment.—These are as follows:

1. To brace the whole shoulder girdle so as to carry the outer fragment upward, outward, and backward
- 2 To depress the inner fragment
3. To maintain reduction
4. To continue active function of the hand, elbow, and shoulder during immobilization of the fracture
5. To check progress by x-ray

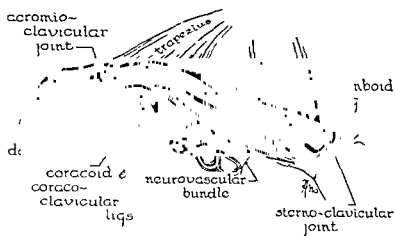


Fig 570—Anatomy of the clavicle

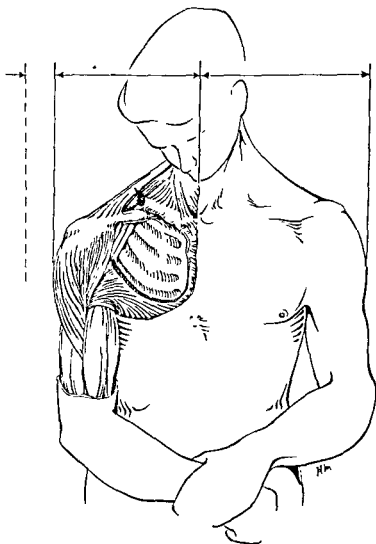


Fig 571—Fracture of clavicle showing typical deformity. The shoulder girdle falls inward and downward, diminishing the prominence of the shoulder. Note how easily injury to the neurovascular bundle could occur

Discussion of Present-Day Methods.—Unless some contraindication exists, most surgeons are content to use conservative methods and try to achieve good results by understanding the principles of reduction and using the following for support.

1. The three-handkerchief or figure-of-eight bandage
2. The figure-of-eight plaster cast

3. Adhesive strapping
4. Bandage to shoulder cross
5. Various commercial splints of light metal

The three-handkerchief method is the simplest to use, but it is necessary to explain to the parents of children or to the adult patient the principles involved: the necessity to hold the shoulder braced upward and back.



Fig. 572 —Fracture of the body of clavicle with typical displacement

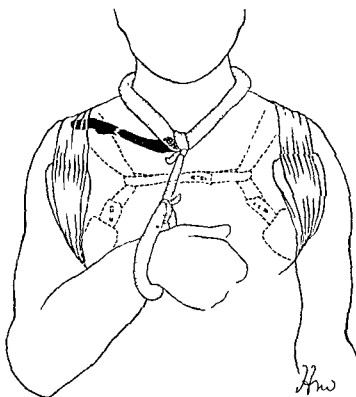


Fig. 573 —Three-handkerchief bandage applied for fractured clavicle.

ward and the daily adjusting and padding of the bandages. Meticulous reduction and supervision are required in the young lady where the cosmetic result is important. Three to four weeks' immobilization is usually adequate.

Open operation is rarely required. If malunion must be prevented, the introduction of an intramedullary wire (Murray) is best. This must not be lightly undertaken. Exposure of the fracture site with insertion of the wire under direct vision will prevent penetration into important structures in this area.

Fractures of the Outer End

Such fractures are protected from marked displacement by muscular and ligamentous attachments. Splinting is often unnecessary and may be minimal. When the fracture involves the acromioclavicular joint, resection of this portion eventually may be indicated.

Fractures of the Inner End

Fractures of the inner end are protected from marked displacement by the muscular and ligamentous attachments. The routine methods of treating fractures of the body are therefore not usually necessary for fractures of the inner or outer end. A sling is sufficient.

Complications of Clavicular Fractures

Nonunion is uncommon except after infection or unsuccessful operative intervention. *Treatment* consists of freshening the bone ends and employing intramedullary fixation supplemented by onlay or cancellous bone grafts.

Stiffness of the shoulder is the most common complication in patients over 40 years. It is sometimes due to a concomitant injury to the shoulder joint but more commonly results from immobilization of this joint during the therapy and is best prevented by keeping the shoulder in use throughout. *Injury to the subclavian vessels, dome of the pleura, or brachial plexus* occasionally occurs.

CLAVICULAR ARTICULATIONS

Acromioclavicular Joint

The possibility of injuries of this joint is often overlooked.

Functional Anatomy.—The joint depends for its strength on the coracoclavicular ligaments as well as the capsular ligaments strengthened superiorly by the aponeurotic attachments of the deltoid and trapezius muscles. An intra-articular meniscus is present in 30-40% of cases. The clavicle may have an overriding articular facet with a loose joint arrangement, or the clavicular facet may be vertical with a closely knit mechanism. The joint permits a small amount of movement in all directions, functioning in the range above 120 degrees and especially in circumduction above 90 degrees abduction.

Mechanism.—Minor sprains and subluxation may be produced by forces transmitted directly or indirectly. Complete dislocations occur when the patient falls on the point of the shoulder. The whole shoulder girdle is forcibly depressed and the clavicle is arrested by the first rib. Continuation of the force causes rupture of the capsule and aponeurosis on the superior aspect followed successively by rupture of the trapezoid and conoid ligaments.

Diagnosis.—There is history of injury followed by pain over the superior aspect of the joint. In sprains and subluxations, forcible adduction of the arm across the chest will accentuate the pain, which can be localized by palpation to the posterosuperior aspect of the joint. In doubtful cases, repeated circumduction of the arm above the horizontal plane, as in throwing a ball, will assist in localizing the pain and tenderness to this joint.

In dislocations the prominence of the outer end of the clavicle is obvious. X-ray examination confirms the displacement and is best done with the patient standing with a 25 pound weight in each hand. Rupture of the coracoclavicular ligaments can thus be readily diagnosed by increase in the coracoclavicular spacing.

Treatment.—For patients without rupture of the coracoclavicular ligaments, no special treatment is required. It is probable that, if symptoms persist for any length of time, the lesion is a derangement of the intra articular meniscus.

In those with complete dislocation, conservative treatment with the shoulder girdle elevated

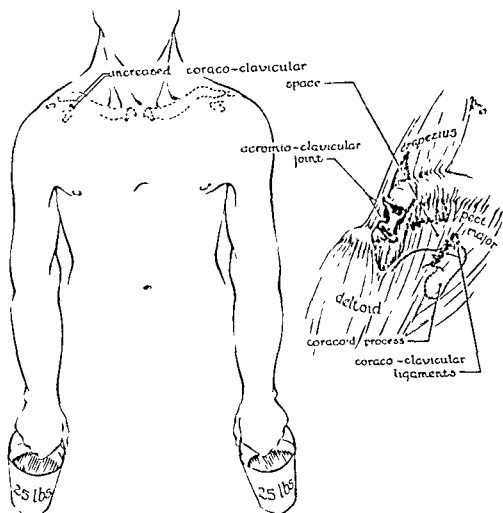


Fig 574—Acromioclavicular dislocation



Fig 575—Complete dislocation of acromioclavicular joint with increased coracoclavicular spacing indicating rupture of conoid and trapezoid ligaments.

FRACTURES OF THE UPPER EXTREMITY

and downward pressure applied over the outer end of the clavicle for 6 weeks will result in good function in the majority of cases. This position can be secured and maintained by adhesive strapping and sling or in a plaster spica. Some deformity usually persists.

In patients who are left with a painful joint after minor injuries or with dislocation after conservative treatment and in some acute dislocations, resection of the outer inch of the clavicle with repair of the conoid and trapezoid ligaments by suture of the clavicle to the coracoid process with fascia or silk gives rapid and excellent results. This is recommended in preference to fascial or metallic repair of the joint.

Sternoclavicular Joint

The sternoclavicular is an exceptionally strong joint and is the pivot for movements of the shoulder girdle, i.e., elevation, depression, protrusion, retraction, and circumduction. It is less frequently injured than the acromioclavicular joint. It constitutes the articulation between the upper limb and the trunk and is involved in sprains, acute and recurrent subluxations, and dislocations. Anteroinferior displacement usually occurs. Posterior dislocations are less common but more serious because of possible pressure on, or damage to, the trachea, esophagus, and great vessels.

Mechanism.—Injury to this joint results from forcible pressure of the clavicle forward and inward. The capsule is torn and forward subluxation or dislocation occurs.

Diagnosis.—The prominence of the inner end of the clavicle is readily seen. Tomograms afford useful x-ray visualization of this joint.

Treatment.—In sprains and subluxations no special treatment is required. In complete dislocation, reduction is readily effected by bracing the shoulder backward and manually pressing the sternal end into place. This reduction is difficult to maintain and requires a plaster spica for 3-4 weeks with the arm at 90 degree abduction and 45 degree forward flexion with direct pressure over the joint in a backward and downward direction. Sometimes the figure-of-eight plaster bandage suffices as for a fractured clavicle.

In some acute and some recurrent dislocations, a fascial repair (Bankart) may be carried

out or the sternal end of the clavicle may be resected, if the patient's symptoms of pain and functional derangement warrant operative treatment.

FRACTURES OF SCAPULA

Fractures of the scapula are not common but occur in the following parts:

1. Glenoid fossa
2. Neck
3. Body
4. Acromion and spine
5. Coracoid process

Glenoid Fossa

Fragmentation of the bony rim may occur in anterior or posterior dislocations, especially if recurrent.

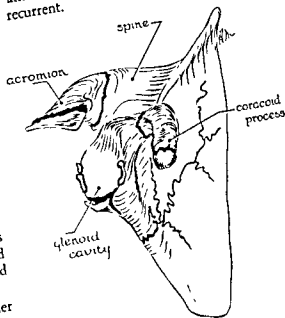


Fig. 376—Composite diagram of type fractures of the scapula

Fractures of the fossa are sustained when the humeral head is forced strongly against it by direct violence. Lesser injuries are contusions of the articular cartilage grading into multiple fissured fractures corresponding to the central dislocation found in the hip.

Treatment.—The treatment of fractures of the rim is part of that of the dislocation. Fractures of the fossa present a variable picture and are frequently associated with injury

to the humeral head. In severe cases the problem is how to minimize the inevitable traumatic arthritis.

In the majority of cases, treatment by early movement is indicated. Where loose fragments of bone or cartilage exist in the joint or where associated ruptures of the rotator cuff are diagnosed, exploration of the joint should be carried out.

ture appears similar to that of anterior dislocation of the shoulder. The deformity readily disappears on pressing upward on the elbow, and reappears on release of pressure.

Treatment.—Cases impacted in fairly satisfactory position may be treated by early movement in the relaxed muscle position. During ambulation, the weight of the limb is taken by a sling arranged so as to elevate the arm.

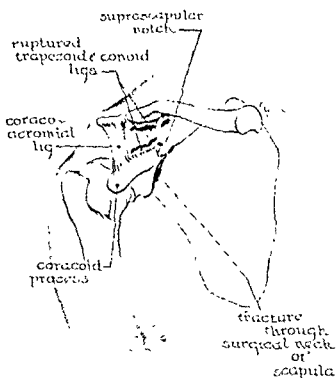


Fig. 577.—Fracture of the surgical neck of the scapula

Neck

The fracture line may pass to the inner or outer side of the base of the coracoid. The majority are impacted without serious displacement. When the fracture line passes to the inner side and the coracoclavicular ligaments are ruptured, the outer fragment is depressed by the weight of the arm, and the clinical pic-

An axillary pad and encircling flannel bandage afford comfortable fixation.

In those patients with instability of the fragments or where other injuries necessitate bed rest, traction in the abducted position, together with elevation of the outer fragment by a band around the upper arm and arranged with gentle traction to the head of the bed for 3-4 weeks, is best.

Body

Fractures of this part are caused by direct violence or crushing injury which also frequently injures the underlying ribs. Because of the buffering and splinting action of the muscles arising from the three fossae, displacement is uncommon.

Treatment.—Aspiration of the hematoma, which often develops, may be indicated. Otherwise treatment is by early function.

Acromion and Spine

Fractures of the acromion must be differentiated from epiphyseal separation in the young or from an ununited epiphysis by contralateral x-ray in later years

The usual cause is direct violence, but sudden action of the condensed middle segment of the deltoid against resistance may produce the fracture. The superior periosteum and aponeurosis first tears, allowing the fragment to hinge, and, if the force carries on, this is followed by tearing of the inferior periosteum with separation of the fragment by deltoid traction. If the fragment is large, some deformity of the deltoid convexity is apparent

Treatment.—These fractures usually heal by fibrous union. Where the fragment is depressed or separated, operative treatment with resuture is indicated to give a solid origin to the deltoid

The same principles apply for fractures of the spine of the scapula.

Coracoid Process

Fractures of the tip are most commonly associated with anterior dislocations of the shoulder. Avulsion through the base may occur in place of rupture of the coracoclavicular ligaments in complete dislocation of the acromioclavicular joint

Treatment in both types is part of that of the associated dislocation

FRACTURES OF THE UPPER END OF THE HUMERUS

Fractures of the various portions of the upper end of the humerus occur as isolated

injuries or may be associated with dislocations of the shoulder.

Surgical Neck

Incidence.—Fracture of the surgical neck is a common fracture and occurs chiefly in the older age groups.

Mechanism.—Dehne classifies fractures of the surgical neck of the humerus on a mechanistic basis. Three groups are defined:

1. *Lateral Mechanism.*—In this group the patient falls with the arm abducted until the surgical neck abuts on the acromion. Either the anteroinferior capsule gives way, permitting dislocation, or fracture occurs at the surgical neck from compression and the greater tuberosity is sheared off by the superior rim of the glenoid fossa. This gives a three-fragment fracture which is most stable in adduction and is displaced by abduction.

2. *Dorsal Mechanism.*—A second manner of falling is backward, and the arm is dorsi-

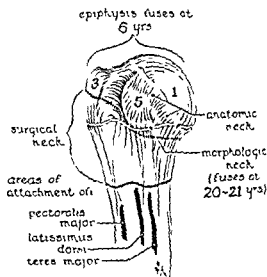


Fig. 378.—Nomenclature of upper end of humerus

Centers of ossification appear in the head, and greater and lesser tuberosities at 1, 3, and 5 years, respectively. These join together to form the superior humeral epiphysis at 6 years which fuses with the diaphysis at 20-21 years

The *anatomic neck* is indicated by the sulcus at the periphery of articular cartilage of the humeral head. The *morphologic neck* marks the junction of the superior humeral epiphysis and the diaphysis, while the *surgical neck*, so-called because it is most commonly involved in fractures, marks the area below the tuberosities and above the insertion of the climbing muscles indicated on the shaft.

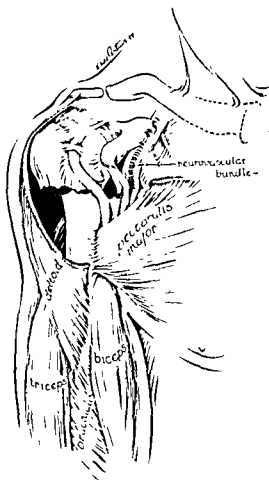


Fig 579—Fracture of surgical neck



Fig 580—Impacted adduction fracture of surgical neck of humerus due to dorsal mechanism
A, Anteroposterior view B, Axillary view

flexed to break the fall. The force of the impact is taken on the elbow and transferred in an anterior and cephalad direction along the shaft of the humerus to the head restrained by the coraco-acromial arch. A fracture of the surgical neck occurs with the axis of the shaft in posterior flexion and adduction. Such fractures are often impacted and stable and consist of two main fragments.

3. *Central Mechanism.*—The fall may be directly to the affected side with the arm in varying degrees of abduction. The impact is taken by the articular head of the humerus on the glenoid fossa. Either surface may be fractured.

Besides this classification, others classify the fractures into those with the shaft in (a) *adduction* or in (b) *abduction* in relation to the head.



Fig. 381.—Displaced surgical neck fracture before and after internal fixation. Lateral views showed anterior displacement of humeral shaft which was corrected at operation.

A and B, Before operation.

C and D, After internal fixation, using two Vitallium nails.

(From Moseley, H. F.: *Shoulder Lesions*, ed. 2, New York, 1953, Paul B. Hoeber, Inc.)

Many are *impacted*, and it must be remembered that the fractures of the surgical neck are extracapsular and joint stiffness is largely the result of extra-articular adhesions following immobilization.

Diagnosis.—This is made on the history of a fall together with symptoms of pain and limitation of shoulder function. Palpation demonstrates tenderness at the surgical neck, and radiologic examination gives the final proof. Detailed views in anteroposterior and superoinferior planes are required. Stereoscopic views may be indicated.

Treatment.—In all cases impacted in satisfactory position and in most cases in patients over 45 years, the treatment advocated is that of immediate movement in the relaxed muscle position. A sling or collar and cuff is used for support.

In the younger patient (20-45 years), with marked displacement, the proximal fragment being abducted and externally rotated, operative treatment with internal fixation by two nails gives excellent results. Early movement is encouraged postoperatively.

Treatment in abduction splints or in traction is required in some cases

Greater Tuberosity

Contusion Fractures

In cases of anterior dislocation of the shoulder, the greater tuberosity may suffer contusion from impingement on the anterior glenoid rim (see Fig. 585). This develops into the posterolateral notch seen in recurrent dislocations. The diagnosis is made by x-ray. Treatment is that for the dislocation.

Avulsion Fractures

These occur by contraction of the supraspinatus, infraspinatus, and teres minor, often in conjunction with anterior dislocation. When the dislocation is reduced, the fragments usually fall into position as indicated by x-ray. If this does not occur, the arm may be splinted in abduction and external rotation, or the displaced tuberosity may be sutured in its normal position.

Avulsion of Supraspinatus Facet

Occasionally, only the supraspinatus facet is avulsed. This is diagnosed by x-ray and the patient's inability to abduct the arm. If it is displaced under the acromion, reduction is necessary to remove the bony block.

Two forms of treatment are possible:

1. Resuture of the torn cuff with or without removal of the bony fragment
2. Splintage in abduction for 4 weeks

Separation of Upper Humeral Epiphysis

Incidence.—These occur in children between the ages 5-15 years.

Diagnosis.—This is made on a typical x-ray picture associated with the presence clinically of a severely injured shoulder.

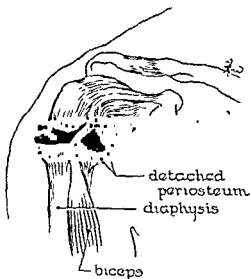


Fig. 582—Diagram showing separation of upper humeral epiphysis

Treatment.—In the displaced fracture, open operation is advocated with the use of Kirschner wire fixation for 3-4 weeks, followed by its removal.

When specialist facilities are lacking, fluoroscopic reduction and immobilization in adduction and internal rotation may be employed.

Lesser Tuberosity

Fractures of this tuberosity occur in rare cases of anterior dislocations of the shoulder.

and represent an avulsion fracture caused by the subscapularis

Treatment.—After reduction of the dislocation the limb is rested in a sling for 3-4 weeks, and this is followed by gradual re-education of shoulder function.

DISLOCATIONS OF THE SHOULDER

The shoulder is the most common joint to suffer dislocation.

Functional Anatomy.—The shoulder joint is part of the shoulder girdle complex and does not function as an isolated unit. The glenohumeral joint is a ball-and-socket joint possessing great mobility. The middle segment of the deltoid and the supraspinatus act as a

team, abducting the humerus on the scapula with simultaneous descent of the head in the fossa. This is the last shoulder function evolved and is the first lost after injury or disease.

The head of the humerus, covered by the rotator cuff, revolves in relation to the coracoacromial arch with interposed subacromial bursa. This mechanism is of great clinical significance and merits the name superior humeral joint. As the humerus must externally rotate during abduction, lesions in this area restrict abduction.

In abduction and forward flexion, movements of the humerus, scapula, and clavicle occur simultaneously and rhythmically, i.e., scapulohumeral rhythm. Clinical examination should center on alterations in this rhythm rather than on localized restriction of movements of the arm.



Fig 583. Anterior dislocation of right shoulder of 7 days duration. Note vascular changes in the whole limb. There is characteristic flattening of the deltoid, and the axis of the humerus is directed toward the middle of the clavicle. (From Moseley, H. F., *Shoulder Lesions*, ed. 2, New York, 1933, Paul B. Hoeber, Inc.)

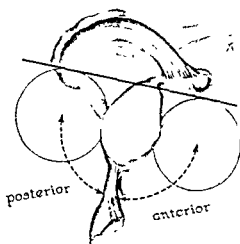


Fig 584.—Possible sites of exit of head from glenoid fossa. (From Moseley, H. F., *Shoulder Lesions*, ed. 2, New York, 1933, Paul B. Hoeber, Inc.)

Classification.—Anatomic considerations determine the classification of dislocations of this joint. Superiorly, the coracoacromial arch acts as a barrier to dislocation except in the rare cases of its disruption when a superior dislocation occurs. For practical purposes all dislocations occur below a line joining its anterior and posterior pillars. Inferiorly, the long tendon of the triceps acts as a strong supporting structure to the inferior capsule in the position

of abduction in which most dislocations occur. Therefore dislocations are divided into those which occur *anterior* or *posterior* to this tendon of the triceps.

The distance to which the head will be displaced anteriorly will depend on the forces concerned, and it may be mentioned that the head has even been driven between the ribs into the chest. The earlier writers classified this distance in relation to the coracoid process as *extracoracoid*, *subcoracoid*, and *intracoracoid* or *subclavicular*.

The exact position along the anterior glenoid rim at which the head dislocates will depend on the degree of abduction at the moment. The extremes are the *directly anterior* displacement caused by a fall backward on the elbow (*dorsal mechanism*) and the *directly inferior* displacement caused by a fall laterally with the arm in full abduction (*lateral mechanism*) producing the *axillary* or *subglenoid* dislocation. In this latter group is included the *luxatio erecta* in which the patient reports with the head subluxated and the arm held in full elevation.

A Simple	{ Anterior Posterior
B Recurrent	{ Anterior Posterior
C Complicated by	{ Fracture Rupture of rotator cuff Nerve injury Vessel injury

Mechanism of Injury.—*Anteroinferior* dislocation results from a fall on the upper limb in such a way that the arm is abducted and externally rotated, levering the humerus against the acromion as a fulcrum out of the glenoid fossa. Sometimes dislocation occurs from a direct force from behind. These two types of fall correspond to the lateral and dorsal mechanisms discussed as the basis for surgical neck fractures.

Posterior dislocation occurs with the arm in adduction and inward rotation with a force transmitted through the humerus in an upward and backward direction.

Post-Traumatic Anatomy.—Operative and autopsy findings tend to show that the usual lesions are as follows:

1. Separation of the anteroinferior or posterior glenoid labrum in the respective anterior or posterior dislocations.

2. Separation of the capsule from the inferior aspect of the neck of the humerus.

3. Separation of the capsule and subscapularis tendon with or without the lesser tuberosity.

4. Posterolateral and anteromedial contusion fractures of the humeral head in anterior and posterior dislocations, respectively.

5. Increased joint capacity indicative of capsular stretching.

Diagnosis.—*Anterior* dislocations are diagnosed by the history of the accident, the location of pain, loss of function of the shoulder, and loss of the rounded contour of the deltoid. Palpation reveals the absence of the head under the acromion and its forward position. The axis of the humerus runs toward the middle of the clavicle.

Radiologic examination confirms the clinical opinion and should be carried out routinely to ascertain whether an associated fracture is present.

When the shoulder has been dislocated longer than 3-4 weeks, the possibility of reduction by closed manipulation becomes doubtful and the condition may be diagnosed as a *chronic* dislocation.

Posterior dislocations are not suspected and are most frequently overlooked, especially in fat patients.

Diagnosis is made on the inability to abduct or externally rotate the arm which is held in internal rotation. The head may be palpated in the subspinous position where maximum tenderness is present.

Routine x-rays are often inconclusive and stereoscopic and superoinferior views are a necessity.

Manipulation under an anesthetic may be the final diagnostic point.

When the patient gives a history of several dislocations, the diagnosis, *recurrent* dislocation, is made.

Treatment

Acute Anterior Dislocation

Reduction is best carried out under the relaxation provided by general anesthesia but can be effected without this assistance.

FRACTURES OF THE UPPER EXTREMITY

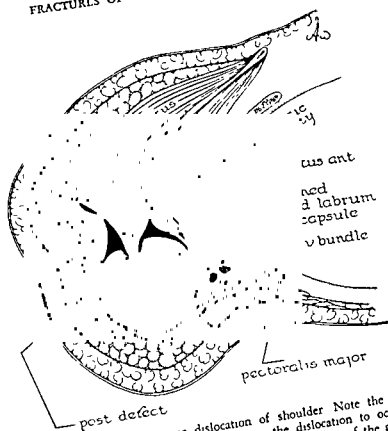
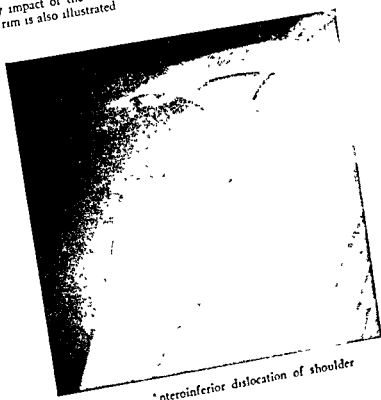


Fig 585—Cross section of anterior dislocation of shoulder. Note the detachment of the glenoid labrum and subscapularis anteriorly, permitting the dislocation to occur. The contusion fracture produced by impact of the articular head in the posterior area of the greater tuberosity on the anterior glenoid rim is also illustrated.



It should be done at the earliest possible moment.

The principle of reduction is to make the head retrace its passage with the least possible further injury to the soft tissues

Three methods are available.

1. Gentle traction in abduction to 140 degrees and external rotation, i.e., the position of dislocation, associated with direct backward pressure on the head as the arm is adducted and internally rotated

2. Hippocratic method: Traction and adduction of the arm with the unbooted heel in the axilla as a fulcrum

3. Kocher's method: Long axis traction with the arm first externally rotated, then, while in forward flexion, adducted across chest and finally internally rotated

Postoperative Therapy.—The most certain way to prevent recurrence is by bandaging the arm to the side for 4 weeks and following this by re-education of the limb musculature and movements.

An alternative is to permit function of the joint but prevent for 4 weeks abduction above 90 degrees or external rotation. The power of the intrinsic muscles is maintained by resistance exercises in this range.

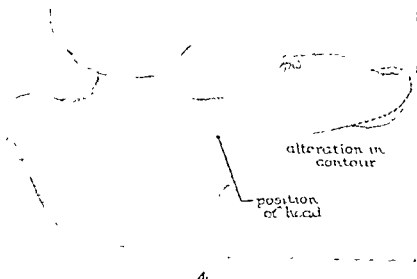


Fig 587—A, Posterior dislocation of right shoulder showing alteration in contour

B, X-ray showing anterointernal notch and posterior dislocation of shoulder (From Moseley, H F: An Atlas of Shoulder Dislocations, courtesy Abbott Laboratories)



Chronic Anterior Dislocation

Reduction has been accomplished up to 6 weeks following injury but after 3-4 weeks may be dangerous because of adherence of the axillary artery and brachial plexus to the productive scar tissue in front of the joint. If the patient is young enough to justify operative treatment, this would be carried out through the anterior approach to the joint. Should the dislocation be of long standing and function adequate, operation should be avoided.

Posterior Dislocation

Reduction can be effected by traction and external rotation in adduction associated with outward and forward pressure on the head secured by a fist or the unbooted heel in the axilla. There is a tendency to instability of reduction, and operative suture of the capsular defect may be indicated.

Recurrent Dislocation

Surgical treatment for this condition is becoming standardized. Repair of the capsule torn from the glenoid rim (Bankart) is per-

formed. Silk sutures through drill holes, staples, fascia (Gallie), bone graft, and metallic rim are different materials employed.

Complicated Dislocations

Dislocations may be complicated by fractures of the greater tuberosity, surgical or anatomic neck, lesser tuberosity, or glenoid rim. Diagnosis is made by x-ray examination.

The principle of treatment is the reduction of the dislocation and fracture as well. In the adult patient open operation is frequently needed.

Ruptures of the rotator cuff should be suspected in patients over 50 years who do not regain the power of abduction and in cases where the axillary nerve is intact as indicated by deltoid contraction against resistance. Operative suture is required.

Dislocations of the shoulder may be accompanied by nerve injuries:

1. Axillary nerve
2. Posterior cord
3. Complete brachial plexus

Sometimes nerve injury results from the excessive force used in reducing the dislocation. These nerve injuries usually recover on conservative therapy, as the injury is one of contusion or stretching.

Rupture of the axillary vessels has been noted, especially in manipulation of the chronic dislocation. Loss of limb and death have resulted.

LESIONS OF THE SOFT TISSUES

Ruptures of the Rotator Cuff

The term *rotator cuff* is applied to the musculotendinous envelope formed by the supraspinatus, infraspinatus, and teres minor attached to the greater tuberosity and the subscapularis attached to the lesser tuberosity. The former are external rotators, the last an internal rotator of the humerus. The tendons of these muscles fuse with the capsule before insertion on the humerus. They function as a fine adjustment in the shoulder mechanism.

Incidence.—Ruptures of the rotator cuff are frequent injuries in patients over 50 years but usually fail to receive adequate medical attention.

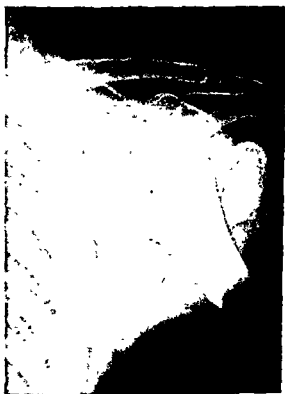
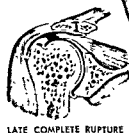
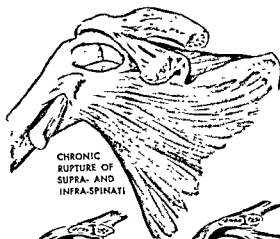
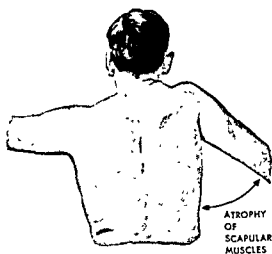
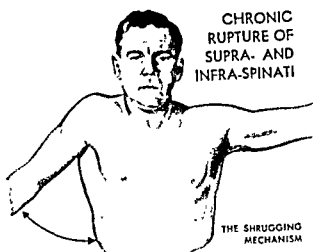
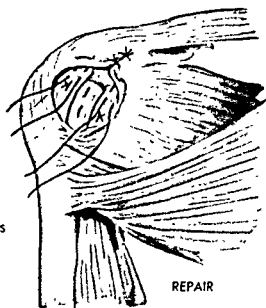
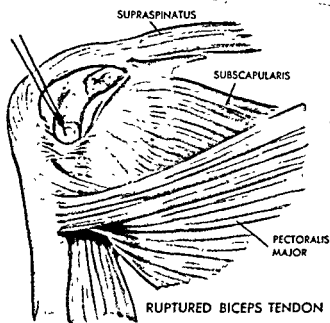
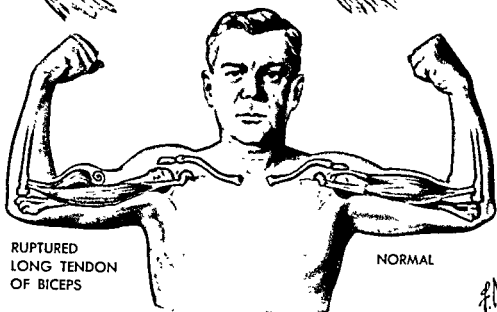
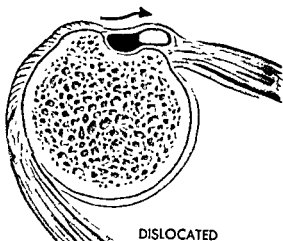
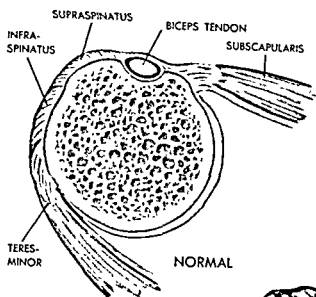


Fig. 588—X-ray of fracture dislocation



BICIPITAL LESIONS



ture or dislocation. The axillary nerve is not paralyzed. Procaine injection demonstrates marked weakness in attaining or maintaining abduction. The drop arm sign indicates massive rupture.

Treatment.—Unless contraindicated by age or general health, the ideal treatment is operative suture at the earliest moment. The tendon is sutured to a trough cut near its original insertion. Postoperative care consists of rest in an abduction splint or in a sling. It is best to repair in such a way that the limb can be in a sling without tension on the suture line. Early movements in the relaxed muscle position can be carried out, but active abduction, lifting the weight of the arm, should not be permitted for at least 4-6 weeks. The whole period of re-education requires 3-6 months, but the benefits of operation are most clearly seen after 1 year. When patients are seen at a late period with weak and painful shoulders and with restricted movement, a trial of the benefit to be obtained by re-education of the shoulder with pulley exercises should be carried out before reconstructive surgery on the torn tendons is considered.

Rupture of the Long Tendon of the Biceps

Rupture of the tendon of the biceps may occur in the intra-articular portion, at the junction with the muscle belly or at its insertion. The first is by far the commonest type.

Incidence.—As for ruptures of the rotator cuff, the biceps tendon is ruptured in the laboring class and in the age group over 50 years.

Mechanism.—The rupture may occur spontaneously from years of gradual attrition involving also the supraspinatus tendon. In other cases it occurs simultaneously with rupture of the supraspinatus tendon when a heavy weight is suddenly lifted above the level of the shoulder.

Clinical Picture.—The patient feels a sudden pain in the shoulder with a snap and notes a lump in the arm. The pain may be minimal in the attrition rupture in senile patients.

Examination reveals tenderness in the bicipital groove and restriction of shoulder movements. Crepitus is usually present on rotation

of the humeral head. Signs of rupture of the rotator cuff are noted.

On supination of the forearm against resistance, the belly of the long head will harden, showing loss of its elongated shape. When relaxed, the loss of the tension of the tendon permits increased mobility to the examining fingers.

Diagnosis.—Diagnosis is readily made on the history of injury and presence of the swelling in the arm. The possibility of concomitant cuff rupture must be considered.

Treatment.—In old patients, reparative treatment is elective. In the active laborer, suture of the tendon in the bicipital groove with plastic repair of the cuff is indicated. The patient should not be permitted heavy use of the limb for 8-12 weeks.

Recurrent Dislocation of the Biceps Tendon

This is a rare condition in which the tendon slips forward over the lesser tuberosity and occurs where repeated stretchings or one severe injury has weakened the bicipital sheath at the top of the groove.

Diagnosis.—This is made by feeling the tendon slipping forward as the humerus is externally rotated.

Treatment.—Therapy is directed to transection of the tendon and fixation of the distal end in its groove.

FRACTURES OF THE SHAFT OF THE HUMERUS

The shaft consists of that portion between the surgical neck and the condyles.

Mechanism.—These fractures may be caused by direct or indirect violence. The resultant injury is a transverse, spiral, or comminuted fracture.

The deforming forces are the fracturing violence and muscle pull.

Fractures occurring above the deltoid insertion have the upper fragment drawn inward by the pectoralis major and latissimus dorsi, whereas the lower fragment is displaced outward by the deltoid. Fractures below this level have the upper fragment drawn outward by the deltoid in relation to the lower fragment. The sling position most commonly used

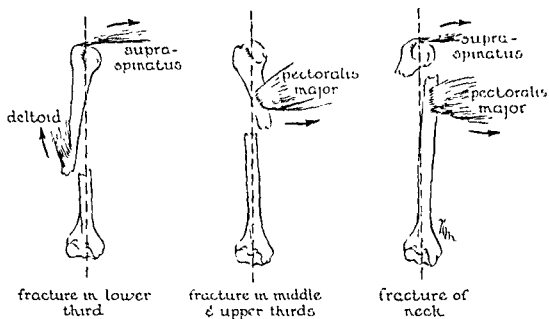


Fig 590 - -Influence of muscular pull on fractures of the humerus at different levels



Fig 591 —A, X-ray of fractured humerus treated in hanging cast.
B, Clinical union with improved alignment.

Diagnosis.—The arm is useless in the complete fractures, with local tenderness, swelling, and deformity marking the site. Radiologic examination gives the exact details of the bony injury and should include three dimensional studies.

manipulative reduction is unsatisfactory when injury to the radial nerve is present. The best approach is that of Henry extending along the outer aspect of the biceps. An intramedullary nail is an alternative form of internal fixation but is less satisfactory.

The time for healing in fractures of the humerus is about 6-12 weeks, being less in the spiral type of fracture and in the young patient.

Complications.—Nonunion may occur in the transverse fractures in the upper and lower thirds as a result of inadequate treatment. Injury to the radial nerve is the most common complication. Stiffness of shoulder and elbow with rasmotor disturbance of the hand may occur. These are best prevented by functional use of the limb and avoidance of passive splinting.

Supracondylar Fracture

Supracondylar Fracture
This is the most common fracture of the lower end of the humerus and occurs most frequently in children and adolescents. The greatest incidence is from 5-8 years. This type accounts for over 50% of elbow fractures.

Mechanism.—The force is usually indirect through the hand and forearm to elbow with posterior displacement of the lower fragment of the humerus. The elbow is almost completely extended.

The reverse fracture may be produced by a direct blow from behind on the flexed joint.

Post-Traumatic Anatomy.—The line of fracture runs upward and backward. The sharp lower end of the humeral shaft may injure the brachial vessels or median nerve. The lower fragment frequently presents a lateral or medial in addition to the posterior displacement.

Clinical Picture.—The patient holds his elbow at 140 degrees. The differential diagnosis must exclude a posterior dislocation. Gross swelling may obscure the bony landmarks of the epicondyles and olecranon which are in their normal relations to each other in contradistinction to the findings in posterior dislocation. X-ray examination is conclusive.

Treatment.—Conservative methods usually suffice. In spiral fractures and in the transverse fractures after manipulative reduction, a spica or sugar-tong cast is applied. In fractures in the lower half, a hanging cast can be used. Amputation has been unsuccessful, trac-

When reduction is employed

Traction methods are advised in small hospitals in preference to open operation with internal fixation. The latter is the method of choice in the hands of specialists when

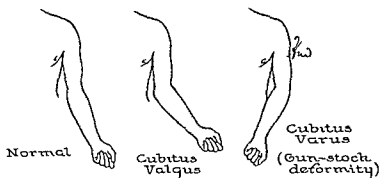
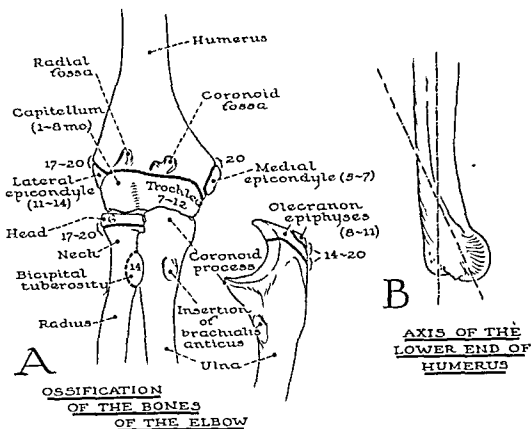


Fig 593.—Surgically applied anatomy of the elbow. The epiphyses of the lower end of the humerus, except that for the medial epicondyle, fuse together at 14-15 years of age and finally unite with the shaft at 17-20 years of age. In each site the earlier dates indicate the time of appearance (in years) of the ossific center, whereas the later dates indicate fusion time.

FRACTURES OF THE UPPER EXTREMITY

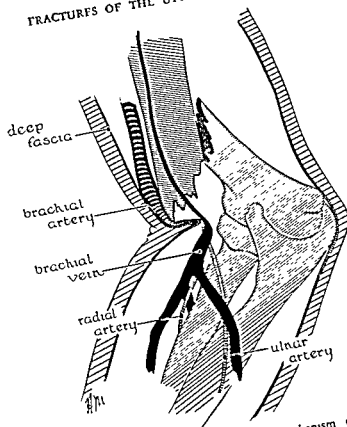


Fig. 594.—Diagram of supracondylar fracture with mechanism of Volkmann's contracture (After Bunnell.)

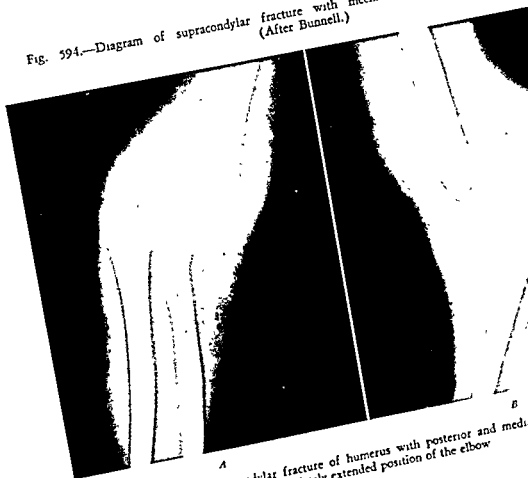


Fig. 595.—Supracondylar fracture of humerus with posterior and medial soft tissue shadow and almost completely extended position of the elbow.
A, Anterior view. B, Lateral view.

Treatment.—The surgeon's approach is determined by the following factors

- 1 Extent of displacement of the fragments
 - 2 Degree of swelling around the elbow
- This factor is a composite of the severity of the injury and the time interval since the accident.
- 3 Circulatory status of the peripheral part of the limb The radial pulse should always be

studied, as well as any signs of circulatory embarrassment such as pallor or cyanosis of the hand and fingers. Because of the possibility of Volkmann's ischemic contracture, all supracondylar fractures should be supervised in hospital

If the patient is seen early and displacement is minimal, a posterior plaster slab or a sling alone, arranged to maintain the elbow in

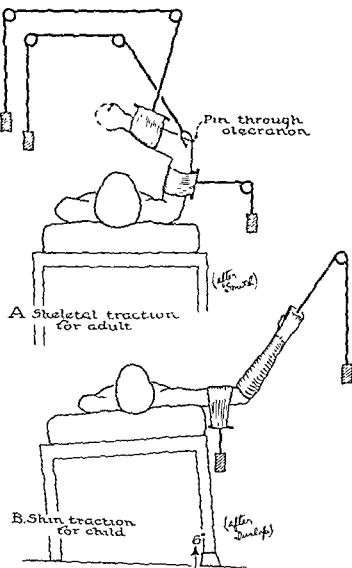


Fig 596—A, Skeletal traction through olecranon, used in the adult for fractures of the lower end of the humerus

B, Danlop's method for supracondylar fractures in the child, occurring especially in the age period 5-12 years. The traction is maintained after x-rays show good reduction and is continued until clinical union of the fragments is present and control x-rays show callus forming.

Countertraction in each case is applied by fixing the trunk to the mattress or opposite side of the bed.

FRACTURES OF THE UPPER EXTREMITY

flexion at less than a right angle, suffices. Gentle active movements may be initiated after 10-14 days. This group rarely gives complications.

If seen early but the displacement is marked, manipulative reduction at the earliest moment under general anesthesia, with radiologic control, is essential. The lateral or medial displacement is first corrected, and then gentle traction with direct pressure downward and forward on the olecranon is used to correct the posterior displacement. The axis of the forearm should be in the direction of the shoulder of the affected side, not in the direction of the opposite shoulder as often suggested. The latter will result in a cubitus valgus (gunstock) deformity. A posterior plaster slab is applied in the maximal flexion permitted by the circulatory condition. This is usually 10 degrees of flexion less than the point where weakening of the radial pulse is noted. The pulse is supervised for 24-48 hours following the manipulative reduction, and danger signals such as pain, pulse changes, pallor, or cyanosis are watched for. The patient is instructed to move the fingers at all times. Immobilization is maintained for 3 weeks. The maintenance of the reduction is checked by x-ray. Re-education of function follows.

If seen late and there is marked displacement associated with brawny swelling around the elbow, the circulatory status should be carefully noted. Most cases in this group should be treated either by skin traction, with the limb elevated and abducted from the side, or by skeletal traction through the olecranon, with the arm in the position of 90 degrees forward flexion. These methods are preferred to manipulation which may not secure or maintain adequate position and often accentuates the traumatic inflammatory reaction. Traction is maintained for 2-3 weeks, and the position is checked periodically by x-ray. Manual manipulations may be required to supplement the treatment by traction. When the traction is removed a collar and cuff or sling is applied and active exercise therapy begun. Traction should be the method employed in any case where the possibility of Volkmann's contracture developing is anticipated.

Fracture-Separation of the Lower Humeral Epiphysis

These epiphyseal injuries may involve.
The entire epiphysis
The lateral condyle or
The medial epicondyle

Lower Humeral Epiphysis

The lower humeral epiphysis consists of two ossific units for the capitellum and trochlea which unite around puberty. There is a further center for the medial epicondyle and an inconstant one for the lateral epicondyle. Fracture-separation of the lower humeral

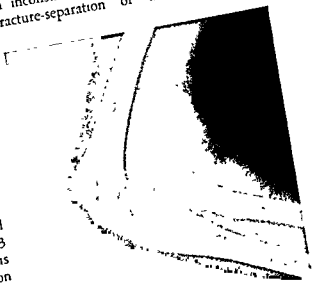


Fig. 597.—X-ray of fracture-separation of lower humeral epiphysis with forward displacement

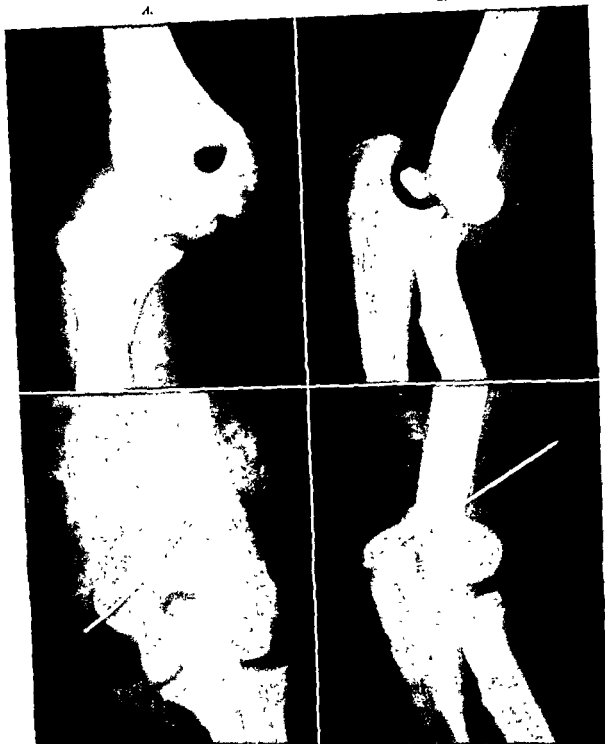
epiphysis with or without the medial epicondyle occasionally occurs before puberty, and the displacement is usually forward. This type of case is generally included with the supracondylar fractures, and the principles of treatment are identical. Cases which have presented the forward displacement require immobilization in extension to preserve the reduction.

Lateral Condyle

This is a serious epiphyseal injury occurring in children and adolescents. The fracture line separates the capitellum and lateral portion of the trochlea together with the lateral epicondyle and common extensor origin.

A.

B.



C.

D.

Fig 598.—Posterolateral dislocation of the left elbow with inclusion of the medial epicondyle within the joint

A and B, Anteroposterior and lateral views before reduction. Note position of the fragment of the medial epicondyle.

C and D, Similar views after reduction and fixation of the medial epicondyle by Kirschner wire

Displacement may be minimal or there may be lateral displacement alone or a rotatory element may be produced by the pull of the extensor muscles

In patients with minimal or lateral displacement, manipulative reduction may be adequate. Plaster immobilization for 3 weeks can be utilized

When the lateral displacement cannot be corrected by manipulation, in those patients presenting displacement but seen several days after the accident, and in most patients presenting a gross deformity due to rotation, open operative reduction and internal fixation, using one or more fine Kirschner wires, are necessary. The wires are removed after 3-4 weeks.



Fig 599 ---Intercondylar T fracture of humerus

Even after operative reduction, growth disturbances tend to occur. Premature closure of the lateral condylar epiphyseal line with overgrowth of the medial condylar element produces a cubitus valgus deformity. A late complication after 10-20 years is secondary ulnar palsy

Medial Epicondyle

Avulsion of the medial epicondylar epiphysis occurs as a result of valgus strain of the elbow and usually complicates postero-

lateral dislocation of this joint. When minimally displaced, no special treatment is necessary. However, when grossly displaced by pull of the common flexor muscles, or when retained within the joint after reduction of the dislocation, or when associated with injury to the adjacent ulnar nerve, open operative reduction and internal fixation are required. Transposition of the ulnar nerve may be necessary.

Condylar Fractures

Fracture of the medial or lateral condyle or both occurs in the adult as a result of direct or indirect violence. The fracture most frequently involves the lateral condyle.

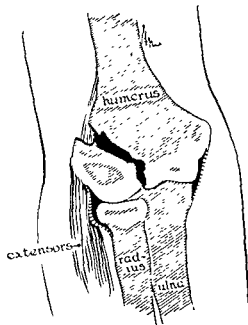


Fig 600 ---Lateral condylar fracture

Patients with minimal displacement require rest for 3 weeks. In the displaced fracture, accurate anatomic reduction is indicated at the earliest moment, and this is best carried out by open operation and internal fixation

Two types of lateral condylar fractures are described. The *unstable type* presents a lateral subluxation since the fractured fragment includes the lateral lip of the trochlea, and the greater sigmoid notch is not stabilized on the remainder of the trochlea. In this type the ulnar collateral ligament is ruptured.

The *stable type* does not show the lateral subluxation, as the lateral lip of the trochlea is not separated.

In both types operative reduction is indicated when displacement is present. Internal fixation should secure anatomic reduction of the articular surface, and in the unstable type suture of the collateral ligament is also required.

Operative procedures on these fractures often lead to aseptic necrosis of the fractured fragment, and secondary procedures may be necessary. For this reason, in the stable type the fragment, if completely separated from its blood supply, should be removed primarily.

In supracondylar, dicondylar, and comminuted fractures of the lower end of the humerus in adults, skeletal traction, excision of devascularized fragments, or a combination of both procedures may be indicated.

Kocher's Fracture

This rare but important fracture consists of the separation of a semilunar fragment of articular cartilage and bone from the capitellum. This may be associated with other fractures of the elbow such as that of the radial head. The fragment is usually displaced into the front of the joint.

Diagnosis is by x-ray, and the treatment is by excision of the fragment.

Fractures of the Epicondyles

Fractures of the epicondyles occur in the young, as epiphyseal separations, or in the adult, from direct violence or by avulsion. The medial epicondyle is the more frequently separated. Conservative therapy suffices except in gross displacement of the medial epicondyle with injury of the ulnar nerve when operative intervention is indicated. This consists of anterior transposition of the nerve with removal or internal fixation of the fragment. When the epicondyle is retained in the joint after reduction of a dislocation, faradic stimulation of the flexors usually secures reduction.

Open operative reduction and suture or removal of the bony fragment often constitute the best treatment.

FRACTURES OF THE HEAD AND NECK OF THE RADIUS

Fractures of the head of the radius are disabling because of the subsequent limitation of pronation and supination of the forearm and hand. They are often associated with dislocation of the elbow.

Mechanism.—Fractures result from indirect violence through the hand transmitted up the radius to the head which is impacted on the capitellar surface of the humerus. Direct violence may also cause fractures to the head alone or to all bones forming the elbow joint.

Post-Traumatic Anatomy.—Radiologic examination indicates the fissuring of the bone, but the cartilaginous damage of the radius and capitellum must be surmised. The types of fracture are indicated in the diagram.

Diagnosis.—This is made by noting the swelling over the head of the radius together with local tenderness and pain accentuated by rotation. X-rays are conclusive and indicate the extent of the injuries.

Treatment

Separation of the Upper Radial Epiphysis.—The typical radial and anterior angulation is shown in Fig. 601. When the angulation is less than 60 degrees, manipulative reduction under general anesthesia should be performed. This consists of placing the elbow in extension, forcing the joint into varus to open up the lateral joint space, and applying direct pressure on the head in a posterior and medial direction, correcting the displacement. Radiologic examination in the operating room will indicate the result. In cases where adequate correction has not been attained or where the original displacement was more severe, open operation and gentle replacement should be performed. *On no account should the radial epiphysis be removed before the age of 15 years, as this leads inevitably to growth disturbances at the elbow and wrist. Restriction of pronation and supination is the invariable sequel. Immobilization in full flexion for 10-14 days is required following operation.*

Fracture of the Neck of the Radius.—In the presence of marked displacement, operation is indicated. If the head can be anatomically

replaced and sutured, this should be done. Usually in the adult the whole head and the loose fragments must be removed and the neck covered with soft tissue to give a smooth surface.

Fractures of the Head.—Fissured fractures without displacement require immobilization in flexion for 3 weeks to allow healing of the fractured joint surface and fixation of loose fragments of cartilage.

Fissured fractures with displacement and comminuted fractures are best treated by resection of the head and removal of all loose fragments at the earliest moment. Postoperative therapy consists of gentle movements, but

traumatic arthritis is minimized by avoiding heavy work for 6-12 weeks.

FRACTURES OF THE OLECRANON

Mechanism.—Fractures of the olecranon may be caused by indirect violence through the triceps or by a direct blow to this prominence.

Post-Traumatic Anatomy.—This injury should be compared with that of the patella. Both involve the extensor mechanism of a hinge joint. The indirect type of fracture is transverse, and when separation is present the aponeurotic attachment of the triceps on each

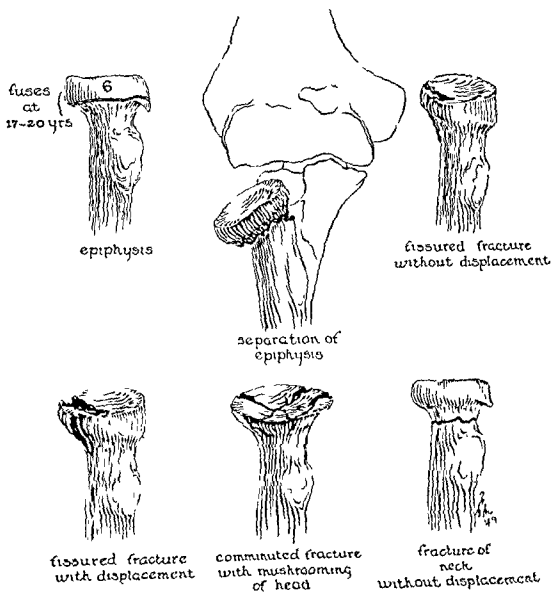


Fig 601.—Types of fracture of head and neck of radius

side is torn. Direct violence results in a comminuted type of fracture.

Diagnosis.—Clinically the area is swollen and tender. The gap between the fragments may be seen or palpated

Treatment.—Stable transverse or comminuted fractures without displacement may be treated by early function associated with local hot moist applications to diminish the swelling

Fractures with separation require exploration and suture. Suture should include the aponeurotic tear as well as the osseous fragments. In the older patient excision of the fragments is often the method of choice. Excision is definitely contraindicated when the



Fig 602—Separated fracture of olecranon

fracture is sufficiently distal to permit anterior subluxation of the ulna. Internal fixation with an intramedullary nail is the treatment of choice in such cases

Immobilization may be in a sling or light plaster cast at 90 degrees. Movements may be encouraged at an early stage, 7-10 days, and excellent results are obtained in 6-12 weeks

COMPLICATED INJURIES OF THE ELBOW

All varieties of injury are found in this region. One of the most difficult is that in which the projecting elbow is struck by a passing vehicle—so-called "sideswipe" fracture. This may be compound and usually consists of a comminuted fracture of all three bones.

LESIONS OF THE SOFT TISSUES

Myositis Ossificans

This is one of the most common complications of injuries of the elbow. Operations required for such trauma should be performed at the earliest moment. When undertaken after the 4th day the probability of myositis ossificans ensuing is greatly increased. The anterior capsule, brachialis anticus insertion, and annular ligament are most frequently involved (See general section on Fractures.) The condition is aggravated by passive movements, especially by forcible extension in the early stages after injury.

Treatment.—Function may be encouraged while the range of motion is increasing. When suspected, the course of the ossification should be followed by repeated x-rays, and gentle active movements only must be permitted. If the patient is seen late with a dense area of calcified tissue limiting motion, operative removal must be considered.

Volkman's Ischemic Contracture

This occurs most frequently after elbow injuries and is due to an interference with the circulation of the forearm muscles. The cause may be a spasm of the brachial artery or an obstruction to the venous return

Treatment.—Prevention by early reduction of dislocations and displaced fractures and by elevation of the limb to encourage venous return is best. When the onset is suspected, because of pain in the forearm, swelling of fingers, and absent radial pulse, hot moist packs and sympathetic block may help. If this fails, exposure of the vessels is required. Excision and replacement of the damaged brachial artery may be necessary.

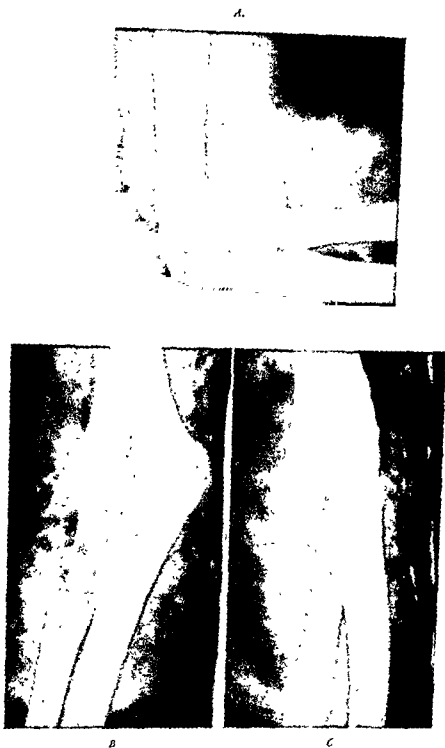


Fig. 603—*A*, End result of fracture-dislocation of elbow which developed traumatic arthritis with myositis ossificans.

B and *C*, Postoperative x-rays after resection of the deformed radial head and calcified soft tissues.

Seddon's idea of resection of the ischemic muscle infarct, involving chiefly the flexor digitorum profundus and flexor pollicis longus, after an interval of 3-4 months to observe recovery affords considerable promise.

After the chronic contracture is established, even extensive reconstructive surgery leaves considerable impairment of function.

Secondary Ulnar Palsy

This follows fractures of the lateral condyle in the young with premature synostosis and development of cubitus valgus. The ulnar

Tennis Elbow

Tennis elbow is a clinical syndrome with several underlying causes.

It is characterized by tenderness over the lateral epicondyle and pain radiating over the extensor aspect of the forearm. There is weakness and pain on extending the wrist against resistance, and any use of the limb accentuates the discomfort.

The lesion is a sprain of the common extensor origin and may be associated with deposition of the phosphate and carbonate of calcium. Rest together with heat and salicylates



Fig 604—Syndrome of tennis elbow caused by calcified deposits in the common extensor origin.

A, Supination

B, Pronation. Note pressure of orbicular ligament on deposit.

nerve is gradually involved in a traumatic neuritis which develops years after the injury. Clawing of the 4th and 5th fingers, with typical sensory changes, is present.

Treatment consists of anterior transposition of the ulnar nerve.

is effective usually. Local needling of the point of maximum tenderness under local anesthesia, followed by infiltration of the area with 1 ml hydrocortisone, frequently produces a cure. If the condition becomes chronic, subperiosteal resection of the common extensor origin is the

best therapy. Calcified material should be removed

Stiff Elbow and Traumatic Arthritis

This is an all too frequent complication of elbow injuries. The elbow is a closely fitting joint and does not react favorably to intra-articular trauma. All operative procedures should remove any mechanical irregularity or loose fragment. This joint does not permit passive movements of a forcible character which usually result in increasing stiffness. It should, if possible, be splinted in flexion above the right angle, and in re-education the progress should be accurately charted after measurement of range by the goniometer. Unless the range is progressively increasing without loss of flexion, the therapy is either being instituted too early or is excessive. There may be an intra-articular derangement causing delay in progress, and the condition should be reviewed.

DISLOCATIONS OF THE ELBOW

Dislocations occur in this joint more commonly in the young and as a result of indirect forces transmitted through the forearm and hand. The majority (80%) are posterior dislocations and may be complicated by fractures of the medial epicondyle, head of the radius, or the coronoid process.

Posterior Dislocation

Mechanism.—Posterior dislocation is caused by a fall on the outstretched hand, the force being transmitted upward with the elbow dislocating by sudden hyperextension and contraction of the triceps.

Post-Traumatic Anatomy.—The displacement of the ulna is upward and backward. The coronoid process may be fractured. The capsule is torn anteriorly and on each side from the humerus.

Clinical Picture.—The patient holds the painful and swollen elbow at 140-160 degrees. Flexion is impossible. The relation of the epicondyles and olecranon is altered, and the olecranon process is very prominent. X-ray examination indicates the displacement and associated fractures.

Reduction is best effected under general anesthesia. Gentle traction on the hand with the elbow at 160 degrees, with direct pressure on the displaced olecranon in a downward and forward direction, usually reduces the displacement with ease. The limb should be splinted in as much flexion as the circulatory state will permit. Dislocations of more than 2 weeks' standing usually require open operative reduction.



FIG. 605.—Patient with posterior dislocation

Postoperative Therapy.—Gentle active movements can be carried out almost immediately as the reduction is stable. Movements should consist of active flexion. If the range of motion progressively increases, function may be augmented. A full range may be secured in 1-6 weeks. Alternatively, plaster immobilization in the flexed position for 3-4 weeks may be employed.

Posterolateral Dislocation

This differs from the above in the lateral displacement, and x-rays may show avulsion of the medial epicondyle. After reduction, the

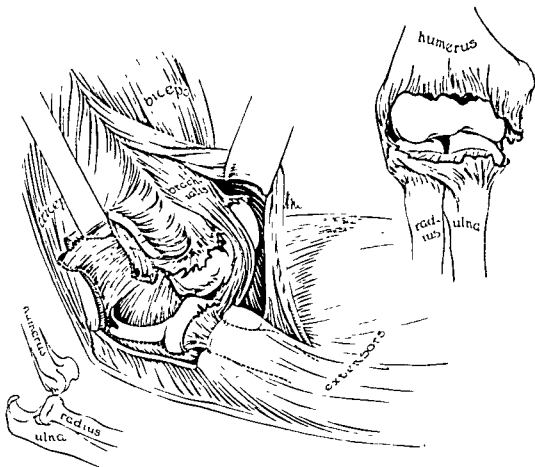


Fig 606.—Posterior dislocation of elbow

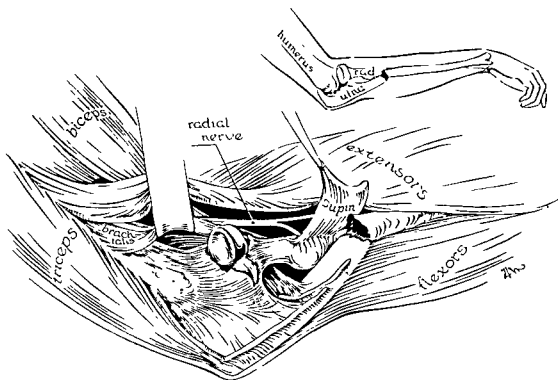


Fig 607.—Monteggia fracture dislocation

medial epicondyle sometimes remains within the joint. Operation with suture or removal of this fragment and with anterior transposition of the ulnar nerve is then indicated. The ulnar nerve may be injured in this dislocation. These patients are best treated in plaster casts as the reduction may be unstable.

Subluxation of the Radial Head (Pulled Elbow)

The radial head and neck are of approximately the same size up to the age of 8 years. Before this time a sudden pull by the parent or nurse on the child's limb subluxates the radial head distally out of the orbicular ligament, with resultant pain and limited elbow function.

On examination the forearm is held in mid-rotation. Any attempt at movement of the elbow, especially with supination of the forearm, is resisted. Radiologic examination is negative for demonstrable displacement.

The subluxation is corrected with a definite click when the forearm is quickly supinated with or without the assistance of general anesthesia.

Monteggia Fracture-Dislocation

It is one of the injuries around the elbow most frequently overlooked and at the same time most difficult to treat.

Mechanism.—The injury results from a fall against an object which acts as a wedge, fracturing the upper third of the ulna, and as the force carries on, the pronated radius is levered forward, rupturing the annular ligament.

Post-Traumatic Anatomy.—This consists of a fracture of the upper third of the ulna with the fragments angulated forward associated with an anterior dislocation of the head of the radius with ruptured annular and radio-humeral ligaments. The biceps tendon tends to maintain this displacement.

Diagnosis.—Such a complicated injury should be suspected in all fractures of the upper third of the ulnar shaft. X-ray examination should be carried out to confirm the diagnosis.

The opposite displacements occur rarely when the violence is in reverse direction.

Treatment.—Early cases should be treated by operative reduction, internal fixation of the fractured ulna, and suture of the annular ligament. Boyd's posterolateral incision is used. The elbow is splinted in flexion and supination for at least 3-4 months.

Late cases with nonunion require open reduction with bone grafting of the ulna and excision of the radial head. If malunion is present, resection of the head of the radius may be necessary to improve pronation and supination; reconstruction of the ulna is not usually indicated.

FRACTURES OF THE FOREARM

Fractures of the forearm may involve one or both bones and are caused by direct or indirect violence. The site of fracture may be in the upper, middle, or lower third, the frequency increasing distally.

Mechanism.—A fall on the outstretched hand is the usual cause. When both bones are broken, the distal fragments are dorsally displaced. Direct violence generally gives a transverse fracture, and if both bones are broken, the fractures are usually at the same level.

Post-Traumatic Anatomy.—In the young, the greenstick type of fracture is common, and considerable angulation is present. In the adult, wide separation is possible.

In fracture of one bone, it must always be remembered that overriding of the fractured ends can occur only with subluxation or dislocation of the other bone at the superior or inferior radioulnar joint. In fracture of the radius, muscular forces must be considered in analyzing the displacements. *Fractures above the pronator teres* have the upper fragment flexed and supinated by the biceps and supinator, whereas the lower fragment is pronated and displaced toward the ulna by the pronator teres and pronator quadratus. *In fractures below the insertion of the pronator teres*, the upper fragment is flexed and supinated to a minor degree, and the lower fragment is displaced toward the ulna. *In fractures in the lower third*, the distal fragment of the radius is pronated and drawn toward the ulna by the pronator quadratus.

In injuries caused by direct violence, the direction of the force is the determining factor in the displacement.

The mechanics of the post-traumatic anatomy of forearm fractures is of the greatest importance. The greenstick fractures, if carefully reduced, are usually stable. Fractures of one or both bones in the adult are prone to re-displacement after reduction unless the fractured surfaces solidly interlock. This tendency to instability after reduction is the reason why fractures in this area are especially suitable for open operation and internal fixation.

Diagnosis.—Diagnosis is readily made in cases of angulation or gross displacement. X-rays in two planes are essential.

Treatment.—From the point of view of therapy the fractures may be divided into (1) greenstick fractures, (2) fractures without displacement, and (3) fractures with displacement.

Greenstick fractures are manipulated under general anesthesia by traction and direct pressure over the point of angulation. The angulation is just overcorrected. Great care must be

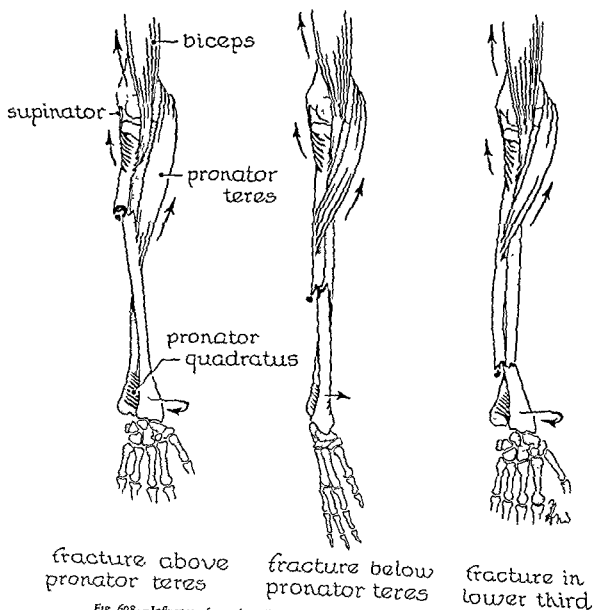


Fig. 608—Influence of muscle pull on fractures of the radius, at different levels.

taken to avoid complete fracture and displacement.

A cast is applied from axilla to metacarpal heads with elbow at 90 degrees, forearm in mid-position, and wrist slightly dorsiflexed. *Fractures of one or both bones without displacement* are placed in the same type of cast. The time of immobilization varies from 6-12 weeks.

tained and a cast applied. The possibility of redisplacement must be checked by repeated x-ray. The cast is changed when the swelling subsides. Thumb traction associated with the cast may prevent some cases of recurrent displacement.

Open operation and fixation of the one bone in single fractures and both bones in dual fractures is the safest and best method



Fig 609—Typical x-rays of fractured radius before and after plating

Fractures with displacement are treated by (1) manipulation and plaster cast, (2) open operation and internal fixation, and (3) external skeletal fixation

Manipulation under a general anesthetic with fluoroscopic or x-ray plate control is the safest method. Stable reduction must be ob-

in the hands of the specialist. Postoperative fixation in a cast is required for 2-3 months. Plates may be removed when union has occurred

Intramedullary fixation has been used in fractures of these bones and is best for fractures of the ulna. External fixation by two pin

units of the Roger Anderson or Stader type can be used by those working routinely with this method. Open operation with plating is advised in preference to this method.

Complications.—*Malunion* is common in forearm fractures because of factors mentioned above, together with the longer period required for consolidation in these bones.

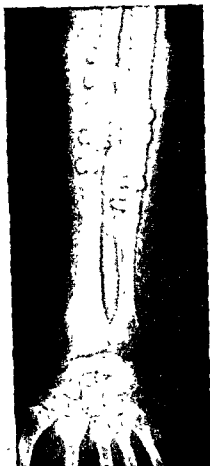


Fig 610—X-ray of cross union

Cross union may occur because of the tendency of the bones to approximate each other after fracture. This tendency is diminished by splinting in mid-pronation, at which point the bones are farthest apart. Both *malunion* and *cross union* have their chief disability in limiting the range of pronation and supination. *Delayed* and *nonunion* are fairly frequent.

Dislocation of superior radioulnar joint may persist after fracture of the upper third of the ulna in Monteggia fracture-dislocation.

Dislocation of inferior radioulnar joint may result from fracture of the radius with overriding or as a concurrent injury. If disabling, resection of the lower inch of the ulna gives considerable improvement in function.

RACTURES OF THE WRIST

Fractures of the lower end of the radius, with or without injury to the articulating bones, constitute the largest group of cases attending traumatic clinics. These injuries occur at all age periods but are most frequent during the active period of life. In northern climates they increase during the winter months.

Mechanism.—The usual cause is a fall on the outstretched hand.

Post-Traumatic Anatomy.—The typical deformity described by *Colles* consists of three parts. The lower fragment is displaced (1) posteriorly, (2) radially, (3) with its articular surface rotated to face more dorsally.

If the radial displacement is sufficient, the ulnar styloid is avulsed with the ulnar collateral ligament. This triple displacement constitutes the dinner-fork deformity. The reverse deformity with forward displacement is called *Smith's fracture*.

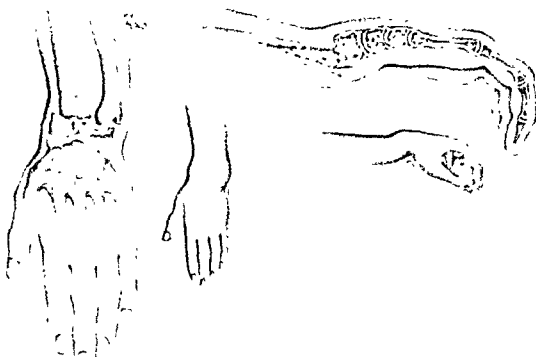
All cases, however, do not fall into this type and the variations are legion. It probably leads to poor work to classify all these cases as *Colles' fractures*, and each must be analyzed and treated as an entity.

In the young adolescent, separation of the lower radial epiphysis, usually through the metaphysis, is the common injury. In older patients, a comminuted impacted fracture with foreshortening of the radius in relation to the ulna is common.

The most important factor in functional restoration is the mechanical alignment of the inferior radioulnar joint.

Derangement of this mechanism results in a limitation of pronation and supination, the latter being that more commonly limited.

Diagnosis.—This is made on the typical deformity when present, the local tenderness, and radiologic findings.



A

Fig. 613—A, Diagram shows the bony displacement and clinical appearance in a typical Colles' fracture with posterior and radial displacement. Note the so called dinner fork deformity as shown in the lateral view.

B, Malunion of a Colles' fracture. Note the radial displacement of the whole hand and the prominence of the lower end of the ulna.



Treatment.—Fractures without displacement should be treated in a functional cast permitting all movements of the digits. Time factor 3-6 weeks.

Fractures and epiphyseal separations with displacement are best reduced by the Helferich or handshake method. The essentials are traction and countertraction for 3-5 minutes under

general or local anesthesia. Following this displacement, the lower fragment is compressed volarward and toward the ulnar side. It is almost impossible to overcorrect a Colles fracture, and adequate reduction of the triple displacement is essential. Both uncorrected displacements and foreshortening will give a derangement of the inferior radioulnar joint with interference to perfect rotation.

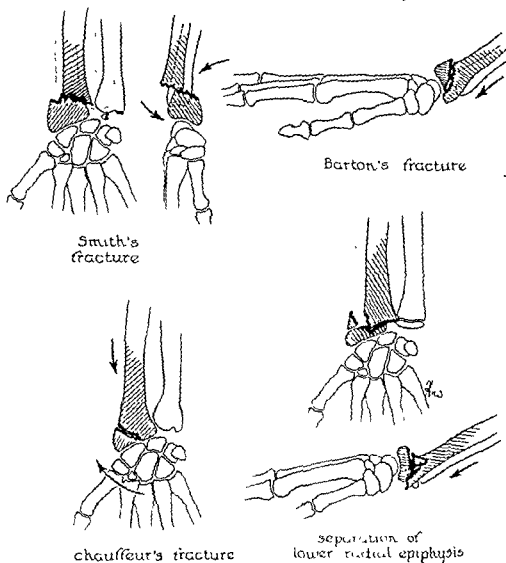


Fig. 614—Type fractures of lower end of radius

Smith's fracture is the reverse of Colles fracture. The lower fragment of the radius with the hand is displaced volarward. The mechanism is a hyperflexion injury or direct blow in a forward direction on the dorsum of the hand.

Barton's fracture is a posterior fracture subluxation of the carpus with separation of a large fragment of the posterior articular surface of the radius. This lesion is typically unstable.

Chauffeur's fracture, so-called from the days before the push button starter in the motor car, was produced by the backfire and sudden reverse of the crank handle. It consists of a fracture separation of a large lateral fragment of the radius with the styloid process.

Separation of the lower radial epiphysis, usually with a triangular fragment of the diaphysis, occurs in the young before closure of the epiphysis at 18-21 years. The displacement occurs most commonly posteriorly but may be anterior.

The essential point to ascertain after reduction is whether the reduction is stable. The vast majority of correctly reduced fractures are stable and can be immobilized in a neutral position. (See general section on Fractures.) Probably not more than 15% are unstable as indicated by the tendency to immediate displacement after manipulation

Reduction is successful up to 10-14 days. After this period refracture by closed or open procedures may be necessary in the younger patients.

After removal of the cast, exercises to restore pronation and supination as well as the function of the whole limb are indicated.

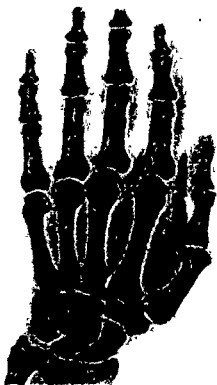


Fig 615—X ray showing the osteoporosis of Sudeck's atrophy with normal hand for comparison

These latter should be immobilized in volar flexion of 10-20 degrees and ulnar deviation. The Cotton-Loder position of full volar flexion is not recommended, because of the tendency to circulatory interference. The typical functional cast should be applied. In the comminuted fractures and in unstable reductions, limitation of elbow movements by inclusion in the cast for 2 weeks is desirable. The average period for plaster splintage is 4 weeks, and during this period, active movements of the fingers are necessary. Elevation of the limb postoperatively to diminish swelling is required.

Complications

Malunion.—Many cases are seen where failure to secure adequate correction of the radial deviation has occurred. Most symptoms relate to the inferior radioulnar joint, although many cases have function which is surprising in view of the deformity. Resection of the lower inch of the ulna will improve some of these cases.

Sudeck's Atrophy.—Post-traumatic bone atrophy occurs most frequently after injuries at the wrist. With the earliest sign of painful swelling and stiffness of the fingers, the cast must be split, the limb elevated, and active

movements of all fingers encouraged. This usually succeeds in arresting the vasomotor disturbances. Should these measures fail, procaine injection of the inferior cervical ganglion should be considered.

Rupture of the Extensor Pollicis Longus Tendon.—This may occur some weeks after the Colles' fracture from injury at the time of the fracture and subsequent attrition.

FRACTURES OF THE SCAPHOID

Fractures of the carpal bones are not common. Injuries of the scaphoid are the most frequent and most important. Neglect of early recognition and immobilization leads to painful nonunion and traumatic arthritis.



Fig. 616.—Type fractures of the scaphoid in relation to the vascular supply

The other carpal bones may be involved in sprain fractures or comminuted by direct or indirect violence. Such cases are rare and are discovered on radiologic examination.

Mechanism.—Fractures of the scaphoid are caused by falls on the outstretched hand with dorsiflexion and deviation to the radial or ulnar side. Fracture through the waist occurs when the two poles are locked and compressed. Forced ulnar deviation may cause a sprain fracture of the tuberosity.

Post-Traumatic Anatomy.—The common injury is a fracture through the waist. Sometimes the line of fracture passes more proximally, giving a small ulnar fragment. The importance of this rests on the fact that the vascular supply to the scaphoid enters on a narrow area on the dorsal aspect of the bone and, in the cases with a small ulnar fragment, this portion of the bone may be devoid of circulation and undergo avascular necrosis. These are prone to nonunion. In certain cases of waist fractures, the radial fragment is displaced and this is often overlooked.

Diagnosis.—All sprains of the wrist with tenderness localized to the radial side of the joint and pain on dorsal or radial movements must be regarded as fractures of this bone until proved otherwise by radiologic examination in anteroposterior, lateral, and especially oblique planes. If the x-ray is negative and the clinical symptoms and signs persist, x-rays must be repeated in 10-21 days. Often a narrow fracture line may be overlooked, with inevitable nonunion.

Differential diagnosis from bipartite scaphoid is made by the even line between the fragments and the bilateral presence of this congenital anomaly.

The fracture line in the fresh case is usually narrow; after 10-21 days it appears broader because of the hyperemic decalcification. Cystic areas appear after 6-21 weeks, and sclerosis may be present after 4-6 months. Ischemic necrosis of the small ulnar fragment may be noted after 3-4 weeks by the relative density of this fragment in comparison to the radial fragment which is undergoing the decalcification of disuse.

Treatment.—Fractures of the tuberosity require only palliative therapy and give little disability.

All fresh fractures of the body require immobilization in a cast which places the wrist and thumb in the position of grasp. The thumb is best immobilized up to the interphalangeal joint (See general section on Fractures). Cases with displacement of the radial fragment need forcible manipulation through the full range of movement. The cast may be removed after 6 weeks, and progress of union should be determined by x-rays. Some cases in young adults may join in 8 weeks, but 16 weeks is more nearly the average. Union is determined when complete obliteration of the fracture line occurs.

If x-rays show ischemic necrosis of the ulnar fragment, two courses are open:

- 1 Continued plaster immobilization which may require 12-24 months
- 2 Removal of the ulnar fragment

There is no unanimity of opinion on this point and the case must be judged on economic

and other bases. The conservative approach is the best in most cases, and in clerical and professional personnel nonunion may be accepted and is often asymptomatic.

Cases of delayed union or nonunion, with pain interfering with work, may be treated by drilling, bone graft, replacement by spherical prosthesis, or arthrodesis of the wrist.

Post-Traumatic Anatomy.—The forced hyperextension of the hand on the forearm causes the hand with the carpus to displace dorsally, leaving the lunate bone in its normal position. This is called a *perilunar dislocation of the carpus*. Sometimes the navicular bone remains beside the lunate, giving a *perilunar-navicular dislocation*. If a concomitant fracture of the navicular occurs, the ulnar fragment usually



A

FIG. 617—A, Fracture of the scaphoid tuberosity. B, X-ray showing the faint fracture line of fractured scaphoid associated with Colles' fracture.

C, Aseptic necrosis with increased density of ulnar fragment.

D, Nonunion of fractured scaphoid. Note sclerosis of bone fragments, also osteophytic changes on proximal fragment and styloid process of radius, indicating traumatic arthritis.



B

C

D.

DISLOCATIONS OF THE CARPUS

Dislocations of the carpus are uncommon, and most occur in relation to the lunate and scaphoid bones, although carpometacarpal and other dislocations occur from unusual direct or indirect violence.

Mechanism.—The cause is usually a fall on the outstretched hand or forced hyperextension of the wrist. Complicated fracture-dislocations result when the hand is caught in machinery.

remains with the lunate, and the radial fragment displaces posteriorly with the carpus, resulting in a *perilunar transnavicular dislocation*.

This is the first stage in the mechanism, since if forward recoil of the hand ensues, the proximal part of the os magnum impinges on the posterior cornu of the lunate and ruptures the posterior radiolunate ligament. The lunate bone is then displaced forward, rotating around

the anterior radiolunate ligament. The carpus has now returned to its normal position and produced an *anterior dislocation of the lunate*. If the navicular remained in whole or in its ulnar part with the lunate, this bone would be displaced forward as well, giving an *anterior dislocation of the lunate and navicular* or *anterior dislocation of the lunate and ulnar half of the navicular*.

the hand in the first stage or the anterior position of the semilunar on palpation in the second stage. Radiologic examination is essential, and in the lateral view attention is best concentrated on the proximal articular surface of the os magnum which normally fits into the concave surface of the lunate. In such dislocations these articular surfaces are completely displaced from each other.

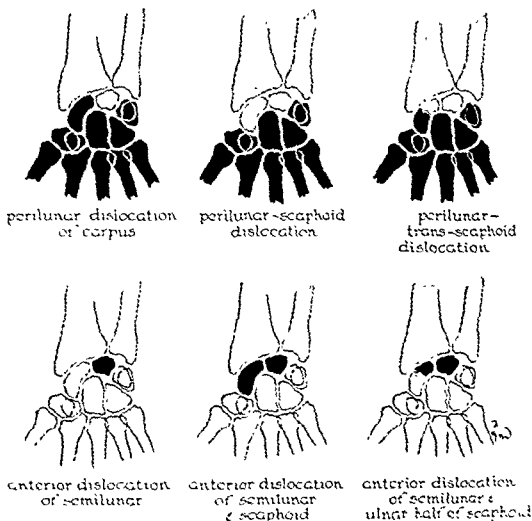


Fig. 618.—Dislocations of the carpal bones. In the upper series of drawings, the bones indicated in black are those displaced, and the displacement is dorsally. In the lower series the carpal bones indicated in black are those displaced, and the displacement is volarward.

A chip fracture of the posterior cornu of the lunate may be present or a marginal fracture of the radius most commonly on its posterior rim. When the semilunar bone is dislocated anteriorly, pressure on the flexor tendons and median nerve is present.

Diagnosis.—The diagnosis may be suspected because of the dorsal displacement of

Treatment.—Reduction is achieved at the earliest moment under the full relaxation of general anesthesia. In the perilunar dislocations, reduction is easier than in the second stage. Long axis traction followed by direct pressure forward on the carpus while the finger firmly presses the *semilunar backward from the volar aspect of the wrist* results in

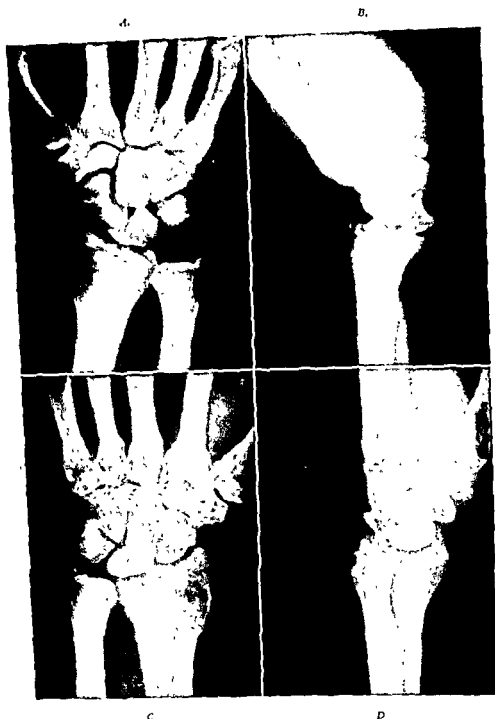


Fig. 619.—X-rays of perilunar dislocation of the carpus

A and *B*, Perilunar dislocation of the carpus in anteroposterior and lateral views. Note the comminuted intra-articular fracture involving the radial and dorsal aspects of the lower end of the radius

C and *D*, Corresponding views 5 months later. Note the aseptic necrosis of the semilunar bone. In the lateral view, the student should learn to recognize the articulation of the os magnum with the semilunar bone



Fig. 620—Dislocation of lunate and proximal half of scaphoid
A and B, Preoperative views

C and D, Several months following reduction performed 4 days after the accident. Note the aseptic necrosis of the lunate and proximal half of scaphoid. Also the failure of complete reduction of the fractured scaphoid and sclerosis of the fracture line.

reduction and prevents this stage from being converted into the more difficult second stage.

In the anterior dislocation of the lunate, long axis traction with the hand gradually dorsiflexed is required to open up the space for the lunate. Direct pressure on the posterior pole in a distal and posterior direction will make this bone retrace its steps.

This dislocation may be reduced by closed methods up to 10-14 days; after this time open

quired, and the time factor will be determined by x-ray evidence of union.

Most of these cases require a firm wrist strap protection when work is begun.

METACARPAIS

Fractures

Mechanism.—Fractures of the metacarpals are common and important injuries. They frequently result from a blow delivered by the fist or from a fall on the outstretched hand.



Fig 621—Kienbock's disease. Idiopathic aseptic necrosis or osteochondritis of the semilunar bone.



Fig 622—X-ray of Bennett's fracture dislocation.

operation is usually required. If this is done within 3-4 weeks, the bones may be replaced, later than this the fragments may be devitalized and are probably best removed.

In dislocations without fracture of the scaphoid, immobilization in a functional cast for 3 weeks is sufficient. When a fractured navicular is present, 8-12 weeks may be re-



Fig 623—X ray of fractured metacarpals.

Direct violence may be the responsible factor, and crushing violence resulting in compound injuries are all too common among industrial accidents.

Post-Traumatic Anatomy.—When the force is directed along the adducted thumb as in boxing, the inner limb of the saddle-shaped base of the first metacarpal is sheared

off against the greater multangular bone. This permits the carpometacarpal joint to dislocate outward and backward and results in the most common metacarpal injury, i.e., *Bennett's fracture-dislocation*. (See Fig. 552, G.)

Should the force be received directly on the flexed 5th finger and the knuckle, an impacted fracture of the neck of this metacarpal results. The head in this case is displaced forward on the neck, and there is usually a noticeable deformity. This injury is second in frequency among metacarpal injuries.

The shafts of the metacarpals may be fractured in spiral or transverse manner, and an important complication is the hematoma which frequently develops on the palmar and dorsal aspects of the hand.

Treatment.—Bennett's fracture-dislocation requires careful treatment to secure good results, and the instability of reduction must be overcome by two methods:

1. Long axis traction in moderate abduction
2. Direct pressure medially on the base of the metacarpal

Traction and splintage are required for 4 weeks. Failure to carry out this treatment results in enlargement of the joint, with pain and development of traumatic arthritis.

Follow-up studies of this particular fracture-dislocation have shown that the frequent result is an enlarged, painful joint due to malunion. Moberg has therefore recommended open operative reduction by reflection of the

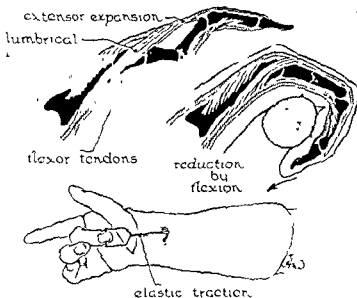


Fig. 621—Splinting of fracture of proximal phalanx

Diagnosis.—Diagnosis of Bennett's fracture-dislocation is made by the painful swelling with deformity and crepitus in this area. It is to be differentiated from a transverse fracture of the base of the metacarpal which is not an intra-articular fracture and which is stable on reduction. X-rays will show the triangular medial fragment in the Bennett fracture-dislocation.

Local tenderness, posterior angulation in the shaft fractures, and loss of the oleprominence in neck fractures are the clinical findings in other injuries.

thenar muscles and fixation by a Kirschner wire removed after 3-4 weeks.

Fracture of the base of first metacarpal is usually impacted with the distal fragments in the adducted position. Direct pressure over the external angulated prominence associated with long axis traction produces a stable reduction. The thumb should be splinted in a well-molded plaster cast for 3 weeks.

In the case with considerable forward displacement of the head, reduction is obtained by direct pressure on the base of the metacarpal and the phalangeal joint.

to a right angle and pushing forward on the shaft fragment while pushing back on the flexed finger. The fracture should be splinted to maintain position with these two opposing pressures or by longitudinal wire fixation.

Spiral fractures of the shafts are not usually seriously displaced. The hematoma on the palmar and dorsal aspects is a frequent complication, and the discomfort is best relieved by elevation, repeated hot baths, and gentle active finger movements. Unless the fracture is very painful and requires protection from blows, no splint is required. When indicated a closely molded plaster cast can be applied which extends to the knuckles and leaves the fingers free.

Transverse fractures of the shafts without displacement can often be treated by early gentle movements and hot baths to assist absorption of the hematoma.

ment is hyperextended and the proximal fragment flexed. Treatment consists of manipulation of the fragments into the correct alignment and maintenance with the finger flexed and long axis traction arranged to line up with the scaphoid tuberosity. Three weeks of elastic traction are required, during which period the other digits are constantly and actively exercised.

Crush injury of distal phalanx is one of the most common hand injuries. Most cases are compound fractures with severe damage to the soft parts.

In the average case, careful cleansing in saline solution followed by local application of a penicillin dressing and splinting will suffice. The minimal use of sutures is advocated. If possible, the nail is left as a splint.



Fig. 625—Mallet finger

Transverse fractures with displacement require manipulative correction and plaster splintage. Incorporation of traction on the corresponding flexed fingers may be required to maintain position. When closed manipulation fails, open operative correction without internal fixation can be safely advised. Only a small dorsal incision is required to permit replacement and interlocking of the fragments.

PHALANGES

Fractures and Other Injuries

Fracture of the proximal phalanx is that which gives most difficulty. The interossei and lumbricals attached to the dorsal aponeurosis buckle the fragments so that the distal frag-

ment is hyperextended and the proximal fragment flexed. Treatment consists of manipulation of the fragments into the correct alignment and maintenance with the finger flexed and long axis traction arranged to line up with the scaphoid tuberosity. Three weeks of elastic traction are required, during which period the other digits are constantly and actively exercised.

Mallet or Dropped Finger.—Mallet finger results when the terminal phalanx is suddenly flexed while the extensor mechanism is taut. It frequently occurs in baseball and cricket players, but the housewife may present the deformity after stubbing the digit. The extensor tendon may be torn off the base of the distal phalanx with or without its bony insertion.

Treatment to be effective must be carefully followed and may be one of two types.

1 Splinting in a plaster cast without interruption for 6 weeks with the distal joint hyperextended and middle joint flexed

2. *Suture with removable suture* placed through the phalanx. The hyperextension of the joint is best maintained by a longitudinally placed Kirschner wire

If either of these methods from the outset appears impractical, the hand may be permitted full movements with protection from strain or injury and will heal with some flexion deformity and stiffness of this joint. This is

placed posteriorly. Such dislocations most frequently occur in the thumb, and the head of the metacarpal is buttonholed on each side by the two heads of flexor pollicis brevis

Reduction.—Reduction is usually possible by hyperextending the thumb and maintaining traction, drawing the digit from side to side while exerting lateral and posterior pressure on the metacarpal head.

If closed reduction fails, dorsolateral incision is made and the buttonhole opened to permit reduction.

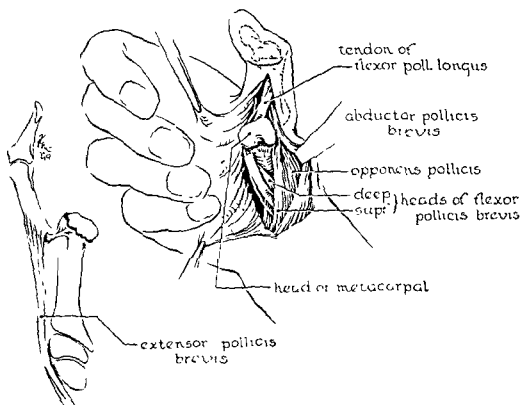


Fig. 626—Backward dislocation of thumb

all too frequently the end result from failure to splint in the correct position for the time indicated.

The joint should be splinted for 7-10 days in a flexed position. This is followed by gentle active movement.

Dislocations

Metacarpophalangeal Dislocations

These most frequently are caused by violent hyperextension. The head of the metacarpal is forced through the front of the capsule, while the base of the phalanx is dis-

Phalangeal Dislocations

These may occur in any direction, but usually the distal phalanx is displaced posteriorly. Reduction is achieved by traction and opposing pressure on the two phalanges. The joint should be splinted in flexion for 7-10 days.

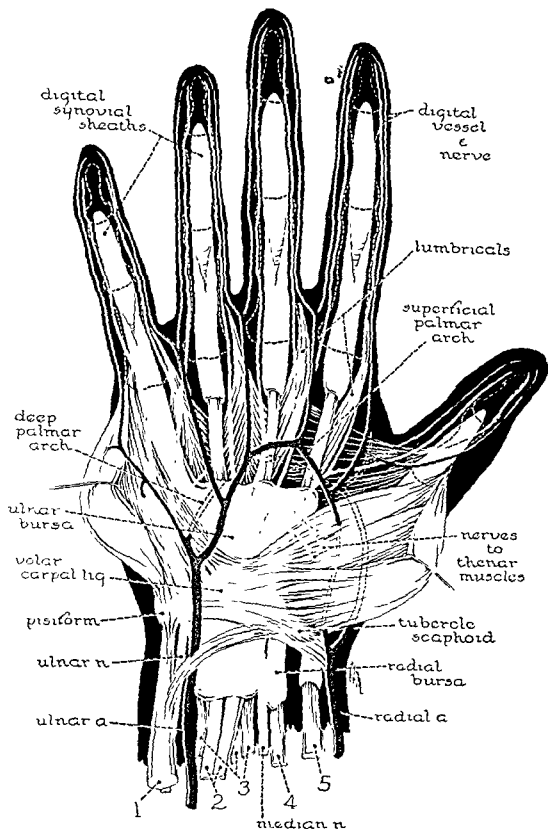


Fig. 627A—Anatomy of the palmar aspect of the hand

The arterial circulation, in red, shows the communication between the radial and ulnar arteries to form the superficial palmar arch and its digital branches, and, in dotted lines, the formation of the deep palmar arch and its anastomoses.

The ulnar and median nerves, in yellow, supply the digital sensory branches to the ulnar $1\frac{1}{2}$ and radial $3\frac{1}{2}$ fingers, respectively.

The digital, ulnar, and radial synovial sheaths are also shown.

1, Flexor carpi ulnaris; 2, Flexor digitorum sublimis; 3, Flexor digitorum profundus; 4, Flexor pollicis longus; 5, Flexor carpi radialis.

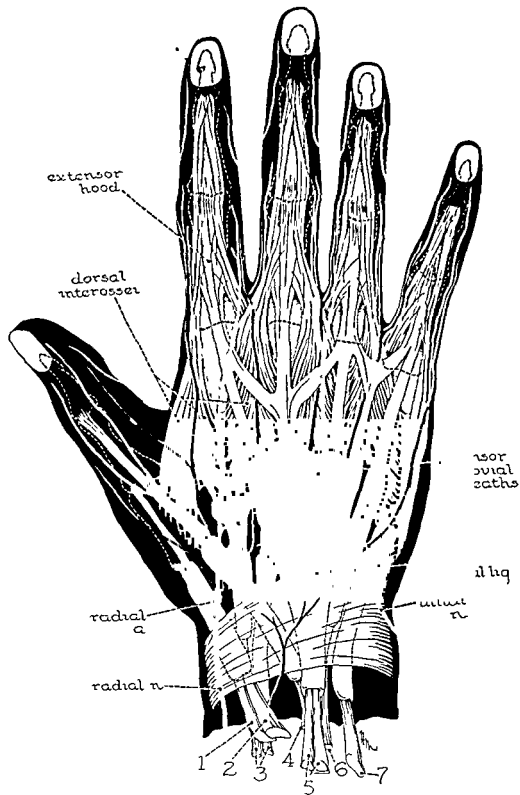


Fig 627B.—Anatomy of the dorsal aspect of the hand

The dorsal arterial circulation, in red, is shown with the radial artery terminating by passing between the two heads of the first dorsal interosseous muscle

The digital sensory supply, in yellow, from the radial and ulnar nerves is shown Also the various extensor synovial sheaths in their relation to the dorsal carpal ligament

1, Abductor pollicis longus 2, Extensor pollicis brevis 3, Extensors carpi radialis longus and brevis 4, Extensor pollicis longus 5, Extensor digitorum communis. 6, Extensor minimi digiti 7, Extensor carpi ulnaris

After tendon severance, a jellylike substance extends from the cut ends into which the connective tissue grows for about $\frac{1}{2}$ " along each, and there is increased vascularity and swelling. *During the first week* a fusiform enlargement develops, which increases throughout the second week. If the tendon is confined within a thecal tunnel during this period, the swelling may result in ischemic necrosis and eventual dense adhesions.

The tendon cells begin proliferation in the second week. There is little strength in the union until the third week, when the tendon fibers begin to bridge the gap.

Between the third and fourth week the healing mass begins to separate from the surrounding tissues, and the fusiform swelling contracts and increases in tensile strength. This is the stage and time when graduated function increases the speed of union as well as the re-establishment of the gliding mechanism.

Mechanism of Injury.—The tendons may be individually severed by the puncture wound of a sharp spicule of glass or metal. Several may be sectioned with associated injury to nerves, blood vessels, bones, and joints in machinery or by lacerations on knives, broken bottles, china, or through glass windows.

Diagnosis.—A most careful history and examination of the movements of the hand and individual tendon functions must be made. Concomitant injuries to digital or major nerves must be determined by attention to sensory and motor loss. X-rays should be taken to ascertain the extent of skeletal damage.

Any defect in the movements of the fingers should suggest a cut tendon, and this must be excluded when the wound is cleansed and sutured.

Principles of Treatment.—The treatment of cut tendons is determined by the environment in which the injury occurs. If near a center where adequate facilities exist, the case should be immediately referred. Tendon suture is not a procedure for the general practitioner. It is best, if specialist facilities are unavailable, to carry out a careful cleansing of the wound and skin suture and then refer the patient later for secondary tendon repair. This will produce much better results than if primary suture is attempted with inadequate technique.

The time factor is of the greatest importance. After 2-3 hours the development of complications is greatly increased. Antibiotics should supplement the most careful wound toilet, and sepsis should be avoided at all costs. *Tendon injuries are acute emergencies and should be so regarded from the moment when first seen by the medical profession.*

Antitetanic serum should be given as a routine. If previously immunized, a booster dose of toxoid can be prescribed.

Technique of Suture.—There are two methods of tendon suture to be considered:

1. The silk technique

2. The removable suture of stainless steel (Bunnell)

Both have their place and both require experience to obtain good results.

Principles Underlying Operative Treatment.—

1. The most delicate handling of the tissues is required

2. If both flexor tendons are sectioned in "No Man's Land," i.e., between the distal palmar and distal digital creases, the sheath should be exposed in its length and removed except for retaining bands over the joints. The flexor sublimis should be routinely removed if cut proximal to its insertion and only the profundus sutured.

3. In secondary suture of the flexor tendons, removal of the sublimis and profundus and replacement with a tendon graft from the palmaris longus sutured in the palm to the profundus tendon and in the theca at the base of the distal phalanx give the best results.

4. Incisions for the fingers should be in the midlateral plane, never longitudinally over the volar surface.

5. In the palm, incisions should follow the flexion creases.

Principles Underlying Postoperative Therapy.—

1. Immobilization with minimal tension is required for 3 weeks for the flexor and 4 weeks for the extensor tendons.

2. Every effort must be made to avoid postoperative swelling of the hand by elevation and by evenly applied compression dressings, with avoidance of constricting bandages.

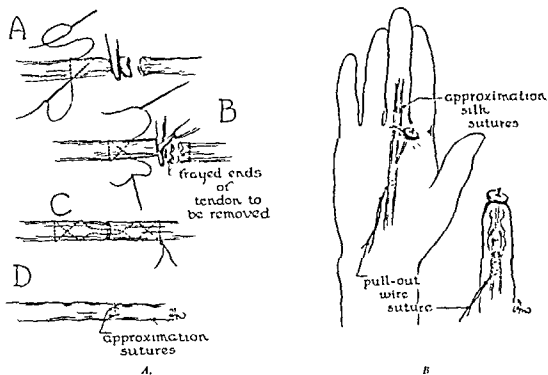


Fig 629—A, The silk technique B, Bunnell technique.

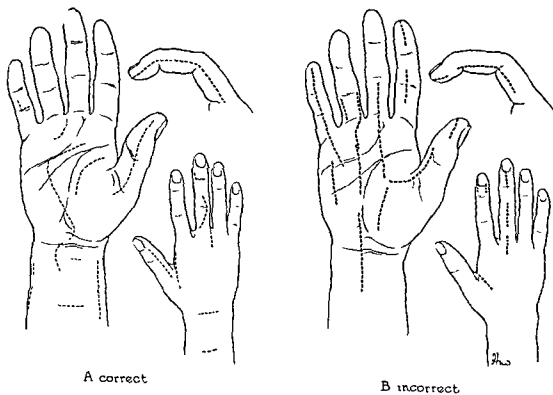


Fig 630—Lines of incision (After Bunnell)

3. The best physiotherapy is the voluntary movement of the fingers in warm soapy water. Lanoline is useful to soften hardened skin. Sponge rubber can be used for grasp exercises.

Results.—The results of tendon suture are greatly improved by attention to the above principles. When cut flexor tendons in the fingers have been properly repaired, the tip of the finger can be flexed to the distal palmar crease. This is also the result to be expected after tendon graft has been employed in place of secondary suture. Repair of the cut extensor gives, on the average, a much better functional result than that of the flexor tendon

space. The cause is usually a prick of the volar surface. Because of the fascial septa limiting swelling, tension rapidly develops with acute throbbing pain. This pain is

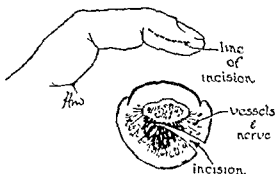


Fig 631—Incision of felon

INFECTION OF THE HAND

The hand is subject to the same infective processes as are the corresponding tissues in other regions of the body. The hand, however, is especially prone to involvement because of its functional activities

Historical.—The work of Kanavel and his followers did much to improve our understanding of this subject. The knowledge of the tremendous morbidity resulting from hand infections, together with the better understanding of the value of prevention and accurate therapy, has done much to lessen the ravages heretofore so frequently seen. The rise of industrial medicine with doctors and nurses present in the large factories enables prophylactic treatment of minor injuries and rapid transfer of the major accidents to specialist facilities. Finally, the development of the antibiotics has given the profession powerful agents to minimize hand infections. The future control rests in prophylactic care at the time of injury and in the use of the chemotherapeutic agents at the earliest moment associated with the most accurate and conservative surgery of this most important part of the body. Already the influence of these principles is obvious to those who compare this problem in large hospitals today with the period prior to World War II.

Infection of the Fingers

Felon

This type of infection is peculiar to the hand and connotes a cellulitis of the pulp



Fig 632—X-ray of felon with sequestration of terminal phalanx

especially severe if the hand is dependent and is relieved by elevation. If the tension is allowed to develop beyond a certain point, the circulation to the distal phalanx is occluded, with necrosis and subsequent osteomyelitis

Diagnosis.—The diagnosis is made on the history of rapidly increasing pain, localized to the pulp space. The tense swelling of the area is noted on palpation. The point of injury may or may not be visible.

Therapy.—Elevation and immediate antibiotic therapy will clear the early cases. If seen late, an incision on the side sectioning the septa should be performed.

Paronychia

This is defined as an infection around the nail. The bacteria enter through a crack on the thickened cuticle of the manual worker or through a hangnail in the office employee. In hospitals, this infection was frequent in nurses when the nail brushes were not sterilized. One of the common causes today is the injury during manicure with nail file or clipper.

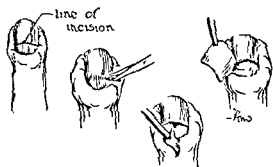


FIG 633.—Drainage of paronychia and subungual abscess.

The process begins superficially as a subcuticular infection at the side of the nail. This forms a localized head of pus, which evacuates itself or is easily evacuated. The infection may spread under the nail at the side and develop into a *subungual abscess*, lifting the nail from its bed. This is especially severe if the spread is to the base of the nail which is raised from its matrix. The skin overlying the nail base is raised, swollen, and reddish or bluish in color. The infection may spread completely around the nail, the so-called "run-around."

Diagnosis.—The clinical appearance at the different stages makes the diagnosis. Transillumination will show the pus beneath the nail as an opaque area.

Treatment.—Bathing the finger in hot water will soften the skin and accelerate localization and evacuation. When pus is present a small incision without anesthetic frequently suffices.

When the spread has occurred under the base of the nail, the nail should be transected at the lanula and the proximal portion avulsed under general or regional anesthesia. Local and systemic antibiotics will rapidly clear the infection.

Web Infection

One of the three triangular web spaces of the fingers may be infected by a prick, cut, or crack in the overlying dermis. The infection usually localizes, forming a pocket of pus, which tends to point dorsally. The process sometimes extends into the hand and involves the middle or thenar space.

Diagnosis.—The reddened swollen area, causing the adjoining fingers to spread, together with the spreading of the swelling onto the dorsum of the fingers is characteristic.

Treatment.—The hand should be elevated, and when the patient is ambulatory the hand can be secured in a sling. Hot bathing gives relief of pain. Systemic antibiotics are administered routinely. Incision will be required when the pus is localized.

Fascial Space Infections

Surgical Anatomy.—Three palmar and two dorsal fascial spaces should be considered.

- Palmar: Middle palmar space
- Thenar space
- Hypothenar space
- Dorsal: Subcutaneous
- Subaponeurotic

Palmar Spaces

The palmar fascia forms a dense roof overlying the deep structures of the palm consisting of the flexor tendons, the branches of the median and ulnar nerves, and the vessels. The floor is formed by the metacarpals, with the fascia overlying the interossei. From the ulnar border of the palmar fascia, a septum passes to be attached to the 5th metacarpal and separates the *hypothenar space* muscles

from the middle palmar space. On the radial side, a membranous septum passes to the 3rd metacarpal, forming the lateral boundary of the middle palmar space, which is thus separated from the *thenar space*. This latter consists of the area on the volar surface of the adductor pollicis and contains the tendons to the index and pollex together with the *thenar musculature*.

The importance of these fascial planes rests in the fact that they determine the passage taken by pus in its extension.

Dorsal Spaces

The dorsal subcutaneous space is found superficial to the dense layer of fascia and aponeurosis joining the extensor tendons. Deep to this plane is the subaponeurotic space. Pus contained in the deep space usually points at the periphery of the aponeurotic layer but may point directly dorsally in a collar-stud type of abscess.

Paths of Infection.—The middle and *thenar spaces* are those of the greatest surgical importance. Infection in the middle palmar space arises from direct puncture or lacerated wounds or from extension of deep infection of the long and ring fingers. Suppurative tenosynovitis of these two fingers or web infection may afford the source of infection by extension proximally.

The *thenar space* is involved by extension from deep infections of the index and volar aspects of the thumb.

The dorsal subaponeurotic space may be involved directly by puncture or lacerated wounds or secondarily by extension from the volar infections. Lymphatic drainage of the fingers extends to the dorsum and may carry the bacteria to the dorsal subcutaneous or subaponeurotic spaces.

Clinical Picture.—In severe infections the general reaction to infection will be present with raised temperature, pulse rate, and malaise.

Local signs give evidence of the infected space and vary with the extent and intensity of the inflammation.

Infection of the middle palmar space results in a swollen, brawny induration of the palm; the fingers are flexed and often the wrist also. Attempts to extend the fingers are resisted because of pain. Tenderness is localized over the middle of the palm. The site of the entrance of the bacteria may be seen. Because of the lymphatic drainage to the dorsum there is considerable swelling of this area as well.

Infection of the thenar space presents greater swelling due to the absence of the resistance of the palmar fascia. The tension

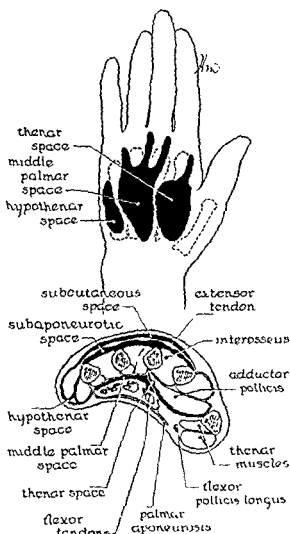


Fig 634.—Fascial spaces

The middle palmar space thus exists deep to the flexor tendons and extends distally along the areolar spaces around the lumbrical muscles to the three web spaces. Proximally it extends through the carpal tunnel into the forearm deep to the flexor tendons.

The *thenar space* extends distally along the lumbrical canal to the radial side of the index finger.

is minimized by the abducted position of the thumb. The swelling is marked on both the palmar and dorsal surfaces

Infections of the dorsal spaces present a reddened and convex surface. The appearance is similar in the swollen dorsum seen in extension of edema from the palmar infections. The differential diagnosis is important and is made by a study of the path of infection and by the presence of fluctuation.

Treatment.—

Prophylactic.—Fascial space infections are best prevented by appropriate care of initial injuries and by the earliest possible use of antibiotics when infection has developed.

Suppurative Tenosynovitis

Infection of the synovial sheaths of the flexor tendons prior to the use of antibiotics was a most crippling disease. Few patients indeed preserved the mobility of the fingers and hand. Today the cases are less frequently seen and when the patients are treated by prompt incision and local and systemic antibiotics, functional restoration can frequently be expected.

Surgical Anatomy.—The flexor tendons of the thumb and fingers are surrounded by a synovial sheath. In the case of the thumb and little finger, this sheath extends from its in-

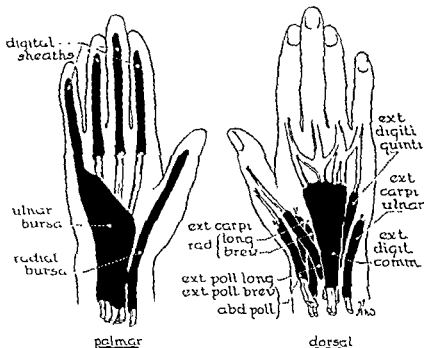


Fig 635—Synovial sheaths

Curative.—The hand should be carefully wrapped in bulky dressings which also afford splinting in the functional position of the hand and wrist. The part should be elevated. If the infection is severe, with general manifestation, the patient is best treated in bed. Massive antibiotic therapy should be given. When the process is localized and pus is present, surgical drainage is carried out. Local antibiotics may be instilled. A culture of the pus and a study of its antibiotic sensitivity are important.

section proximally into the radial and ulnar bursae surrounding the flexor pollicis longus and the flexor sublimis and profundus tendons, respectively. The synovial covering on the tendons of the index, middle, and ring fingers extends from the insertions to the distal palmar crease.

The radial and ulnar bursae lie close together in the carpal tunnel deep to the annular ligament. Communication exists here in the majority of cases. The sheaths extend proxi-

mally for a short distance above the annular ligament, projecting into the forearm fascial space.

The course of extension of the infective process can thus be readily seen. If the infection occurs on the thumb or little finger, involving the synovial sheath, extension will occur to the radial or ulnar bursa or finally both, with possible rupture into the forearm fascial space.

On the index finger, extension proximally will be into the thenar space, and on the long and ring fingers, into the middle palmar space.

Paths of Infection.—The synovial tendon sheath is usually involved from a puncture wound or laceration in the flexion creases on the digits. This may be a microscopic or macroscopic wound.

Clinical Picture.—The symptoms and signs of suppurative tenosynovitis are more marked than those of fascial space infections.

Toxic absorption is rapid, and the patient looks and feels ill with raised temperature (102° - 104° F.) and rapid pulse.

Locally there is exquisite tenderness localized to the digital sheath or to the ulnar and radial bursae, where extension has occurred. The finger is flexed and swollen when a single sheath is involved, or the whole hand is flexed and swollen when the ulnar bursa is involved. Attempts to extend the fingers give rise to intense pain.

Treatment.—

Prophylactic.—Immediate care should be given to puncture wounds. In medical personnel this is important in needle or scalpel pricks on septic cases. Careful sucking out of the blood and washing to encourage bleeding, followed by soaking in alcohol, is desirable.

Antibiotics should be given at the earliest sign of infection.

Curative.—Bed rest is indicated when infection has developed, and the part should be splinted and elevated. Systemic antibiotics should be forced.

The tendon sheath is best opened under tourniquet and general anesthesia, when the diagnosis is established. Local antibiotics should be instilled through a fine catheter.

When one digital sheath alone is involved and is seen late, amputation must be consid-

ered, with the exception of cases involving the thumb. This is especially true if involvement of bone or joint is present.

Lymphangitis

Lymphangitis is the most fulminating infection found in the hand. From a small prick a streptococcus or staphylococcus strain may enter the lymphatic system and rapidly spread proximally along the lymphatic channels.

The red streaks up the inflamed lymphatics are readily seen, and this renders diagnosis easy.

Treatment.—When first suspected, the patient should be treated in bed. In severe infections, general therapy directed to maintain the fluid balance is important. Today, with antibiotics, this dread process can rapidly be brought under control. Systemic antibiotics are forced. The limb is elevated and splinted. Cooling lead and opium or hypertonic saline dressings may be applied to diminish pain.

Human Tooth Wounds

Human tooth wounds of the hand are serious injuries and result from bites or a blow by the fist on the teeth. The wounds usually involve the knuckle area and penetrate through the extensor tendons into the adjacent joints.

Bacteriology.—*Streptococcus*, *staphylococcus*, *Bacillus fusiformis*, and spirochetes of Vincent's angina, *E. coli*, proteus, and anaerobic streptococci.

Clinical Course.—Unless treated early, the process runs a rapidly progressive course, with the development of a foul-smelling wound from involvement of tendons, joint, and subaponeurotic planes.

Treatment.—These wounds should be most seriously considered as the organisms are acclimated to human tissues. Surgical débridement is required with careful cleansing with saline, peroxide, and finally antibiotic instillation. The wounds are best left open and subjected later to secondary suture.

When seen later the path of infection should be opened and the wound treated with zinc peroxide. Cultures should be taken to indicate which antibiotic would be most efficacious.

DUPUYTREN'S CONTRACTURE

This consists of a contracture of the palmar fascia together with its digital extensions. It is named after Dupuytren, who described the pathology and clinical findings after dissecting a hand involved with this disease.

Clinical Picture.—The process is more common in the male sex and, contrary to common opinion, in the sedentary than in the manual worker. There is a hereditary tendency, and it is prone to occur in families subject to gout and cardiovascular degeneration. It has appeared acutely after coronary thrombosis. The patients are usually those with close-knit joints with a tendency to stiffness thereof even after minor trauma. The contracture develops in later years but has been seen in adolescence.

side of the hand. In the early stages there are signs of an active inflammatory process with round cell infiltration. The process runs a variable course, varying in its extent and intensity. Gradually, however, the fibrosis involves the skin, with loss of the fat lobules and skin glands by pressure atrophy. The extension to the fingers causes flexion of the proximal joints and increasing stiffness. The ring and other fingers are curled into the palm so that it is awkward for the patient when working and embarrassing when shaking hands.

Treatment.—Each case must be judged according to the stage of the disease and the actual disability to the individual patient.

When seen early the patient should be advised to avoid chronic trauma such as pressure

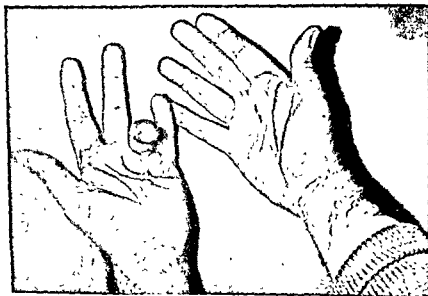


Fig. 636.—Dupuytren's contracture

The thickening first appears deep to the distal palmar crease and extends into the ring finger. The fingers involved in order of frequency are the ring finger, little finger, middle finger, index and thumb. The condition is frequently bilateral. There is an associated tendency to contracture in the plantar fascia and also in the fascia between the corpus spongiosum and corpora cavernosa of the penis.

Pathology.—The process begins as a fibroblastic proliferation in the palmar fascia deep to the distal palmar crease and on the ulnar

or strain to the involved tissues, for although trauma probably does not initiate the process, it appears to aggravate it. The use of a chamois glove for work or sports is helpful.

Operative therapy should be considered before the skin becomes too involved and the finger joints stiffened. If seen early, complete excision of the palmar fascia and its extensions through incisions in the palmar creases is best. If the skin is grossly involved, it must be removed and replaced by a split-thickness skin graft. When marked flexion and stiffness are present in the ring or 5th finger, interfering

with work, amputation may be indicated. The use of multiple subcutaneous fasciotomies does not give good results but may be employed as a preliminary to radical excision in severe cases.

GANGLIA

Ganglia are cystic swellings which are found in many parts of the body arising from joint capsules, ligaments, and tendon sheaths. They are especially common on the hands and feet.

Pathology.—Various theories exist as to their nature. Some believe them to be of neoplastic origin with the formation of the cystic contents by secretion of the connective tissue cells. Others hold that they develop because of connective tissue irritation in an area of mechanical derangement where ligament or tendon sheath plays over a bony prominence. Ganglia frequently communicate with joints, but few are due to herniation of the synovial lining.

Treatment.—Many ganglia around the wrist disappear spontaneously if a wrist support is employed. This is especially true of those over the dorsal aspect of the lunate bone.

The ganglia can be evacuated by direct blow or needling. The best method to secure a cure is by careful excision.

EPIDERMMOID CYSTS

Cystic swellings are found on the hands of manual workers and follow wounds with implantation of epithelial cells. Hence they are frequently called implantation dermoids. The cysts contain epithelial debris rich in cholesterol.

Treatment.—Excision.

TRIGGER FINGER

This is an interesting anomaly which results from a thickening in the proximal end of the

tendon sheath near the distal palmar crease or a swelling of the tendon mass where the sublimis divides to cross the flexor profundus. This results in a locking of the tendons at this point in the sheath, whereupon if the patient forcibly extends, the finger suddenly moves with a jerk. A similar derangement occurs in the thumb, and the site of disparity of tendon and theca is over the metacarpophalangeal joint.

Treatment.—Under local or general anesthesia the area is exposed through a transverse incision. The sheath is sectioned at the side for approximately 1", whereupon movements of the finger demonstrate that the mechanical derangement has been relieved.

TENDOVAGINITIS OF THE RADIAL STYLOID (DEQUERVAIN)

This condition should be correlated with trigger finger. It consists of a thickening of the tendon sheaths over the abductor pollicis longus and extensor pollicis brevis near the radial styloid. Mechanical irritation is probably the cause.

The patient complains of pain radiating from this area over the extensor aspect of the thumb and up the forearm. The lateral aspect of the lower end of the radius may show a tender fusiform swelling.

Treatment.—Under local anesthesia a transverse skin incision and longitudinal section of the tendon sheath are made. Immediate function is permitted. The results are excellent from this opening of the sheath.

CARPAL TUNNEL SYNDROME

In recent years a neuritis of the median nerve due to compression in the carpal tunnel has been described. The onset may be acute, subacute, or chronic and be caused by acute hemorrhage, acute or chronic tenosynovitis,

Fig 637.—Tuberculous tenosynovitis (compound palmar ganglion). This case began as a tuberculous osteomyelitis of the 5th finger, extending into the ulnar bursa, then involving the radial bursa, and late arthritis of the carpal joints. Fluctuation could be obtained from above to the dorsal aspect of the carpus from the volar aspect of the 5th finger, with excision of the diseased synovia, followed by a section of the radiocarpal, carpal, and carpometacarpal joints II and III. Antituberculous therapy was used in the preoperative and postoperative periods.

A, Anterior view. B, Lateral view. C, Melon seed bodies.



Fig 637.—For legend, see opposite page

ganglion formation, the presence of a displaced semilunar or scaphoid bone, or rheumatic changes in the soft tissue boundaries or contents.

Neuralgic pain and hypesthesia in the median distribution and motor weakness and atrophy of the thenar muscles will be noted on examination. The syndrome is immediately relieved by section of the transverse carpal ligament

TUBERCULOUS TENOSYNOVITIS

The synovial sheaths of the flexor and extensor tendons may be involved by tuberculosis. This is frequently secondary to disease elsewhere, such as in the lungs or lymphatic nodes. Characteristically the ulnar bursa is involved with the formation of a cystic swelling extending under the carpal ligaments in dumbbell-shaped arrangement. Fluctuation can be obtained from the forearm to the palmar portions, and the condition is referred to as a *compound palmar ganglion*. The cyst may contain fibrinous bodies of the melon seed or rice body variety.

Treatment.—The treatment is both general and local. The patient must be regarded as one with systemic tuberculosis, the treatment of the hand is incidental and should be timed for a period when the systemic reaction is minimal. Complete excision of the involved tissues is the ideal surgical therapy. Streptomycin, para-aminosalicylic acid, and isoniazid should be the associated chemotherapy.

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Open Wounds and Soft Tissue Injuries of the Hand

Martin A. Entin, M.D

Soft tissues of the hand include all structures, with the exception of the bony skeleton, and thus represent most of the vital components. The most important of these are nerves, tendons, blood vessels, ligaments, and skin. Because the hand has evolved as an organ of perception and prehension, bulk and padding have been sacrificed to permit facile motion and ready perception. Moreover, a high degree of accuracy is required in the fitting of the various structures in order to permit agile and accurate synergistic movement of the various digits. These features make the hand vulnerable to injury from outside. Since great precision is required, even a small amount of distortion produces serious interference with function. Furthermore, open wounds of the hand pose one of the most frequent challenges in surgery and require immediate repair in order to avoid infection and scarring, which inevitably follow if such defects are not covered.

CLASSIFICATION OF INJURIES

Two classifications are available: one is based on the type of the mechanism of injury, the other is based on the potentiality of possible reconstruction. Both are valuable, because they complement each other and aid in quick assessment of a given injury regarding the most efficacious treatment and the probability of the ultimate function.

Types of Injury Based on the Mechanism of Trauma

Mechanical Injuries—These types of injuries are differentiated on the basis of the predominating character of the wound and consist of the following:

- | | |
|-------------------------|------------|
| 1 Incised (lacerations) | 4 Crushing |
| 2 Slicing | 5 Avulsing |
| 3 Amputating | 6 Mixed |

Thermal Injuries—

- 1 Burns (See Chapter 6)
 - 2 Frostbite (See Chapter 6)
- Electric Burns*—(See Chapter 6)

Only the mechanical injuries will be detailed here.

Incised Injuries (Lacerations).—These injuries are produced by either sharp or blunt objects and can be clean-cut (linear) or irregular (ragged).

Clean-cut Wounds.—These are usually closed directly. However, a linear wound that traverses the creases of a digit in the middle of the palmar aspect inevitably leads to contraction; consequently, such a wound should be primarily broken up by a series of Z-plastics in order to minimize the contractile force of the straight-line scar.

Irregular or Ragged Wounds.—These wounds may be converted into tidy ones by excision, provided closure without tension is possible.

Because of the proximity of the important structures to the surface, a linear wound is likely to sever tendons or nerves. On the volar aspect of the fingers, one or both flexor tendons may be involved, and usually a secondary tendon graft is carried out for tendon injuries within the tendon sheath. However, severed tendons in the palm, on the volar aspect of the wrist, and on the extensor surface of the hand or forearm are usually repaired immediately.

Slicing Injuries.—These injuries are produced by sharp objects and can be clean-cut (by a knife) or irregular (power saw) and may be accompanied by loss of skin cover.

No Loss of Skin—All severed structures should be repaired in a fresh wound, except for tendons severed in the area of the fingers between the distal crease of the palm and distal pulley of the profundus tendon (no man's land). Because primary sutures of tendons in this area are notoriously unsuccessful, secondary tendon graft is usually recommended.

Loss of Skin—If no vital structures are exposed, a free split-thickness skin graft is applied to the defect. If important structures such as tendons, nerves, vessels and bare bones are exposed, the area is covered either by a local rotation flap or by a distant pedicle flap.



Fig 638—Incised wound of the wrist, complicated by severance of 7 extensor tendons.

A, Appearance of the incised wound on the dorsum of the right hand of a 30 year old male 2 hours after injury.

B, Immediate definitive repair was carried out and all tendons were sutured. Photograph shows the range of extension 4 months after injury.

C, Same as B, showing the range of flexion.



Fig 639—Incised injury, complicated by severance of flexor tendons.

A, Both flexor tendons were severed through a linear wound at the base of the right middle finger. Initial treatment consisted of suturing of laceration.

B, Definitive reconstruction of severed tendons was carried out 3 weeks after injury, using extensor tendon of the 2nd toe as a graft to the middle finger.

C, Flexion of middle finger 4 months after tendon graft.

Amputating Injuries.—This type of injury is produced either by sharp objects (e.g., knife, saw) or by blunt ones (e.g., press). There may be a loss of a portion of one or several digits, and the amputation may be transverse or oblique.

Finger-Tip Injuries and Partial Loss of Digits.—If the thumb and index finger are involved, every attempt should be made to preserve the remaining length of the digits by application of a skin graft if the bed is suitable, or by a flap from neighboring fingers, or by a flap from infraclavicular or pectoral areas. For the remaining fingers, direct closure with the aid of shortening of the bony stump is most practical. (See Fig. 641.)

Loss of the Whole Digit (Near Metacarpophalangeal joints).—If the entire thumb is lost, the disability of the hand is great, and some form of reconstruction of the thumb should be employed at a later date. Pollicization of the index finger is the best method of thumb reconstruction because it provides a movable digit with sensation that can oppose the remaining fingers.

If the index finger is lost, oblique amputation of the 2nd metacarpal is usually

recommended. This procedure is done primarily for cosmetic reasons and should be carried out in women.

Total loss of *other digits* does not require special procedures and is well handled by direct closure of the wound, although shifting of the little finger onto the stump of the amputated 4th metacarpal avoids scissoring and provides a better appearance.

Loss of All Fingers (Metacarpophalangeal Level).—This is a very disabling injury; some function can be salvaged by deepening the cleft between the remaining portion of the thumb and the 2nd metacarpal.

Total Loss of Hand.—This is not a salvageable injury, and reconstruction is not practical.

Crushing Injuries.—This type of injury is produced by a force of compression or rotation and occurs in rollers, wringers, presses, and falling heavy objects. Either of two types of crushing injuries may occur, *closed* or *open*. The force of compression may cause a tearing or shearing of various strata of tissues upon each other, so that although there may not be any open wound, the skin and the underlying tissue may, in fact, be detached from their blood supply. When seen early, such *closed*

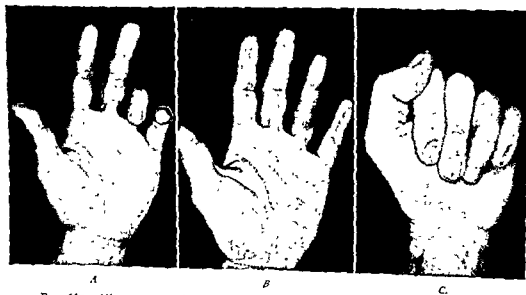


Fig. 640.—Slicing injury of the volar aspect of the ring finger, complicated by flexion contracture.

A, Flexion contracture of the left ring finger in a 17-year-old boy photographed 4 months after slicing injury.

B, Flexion contracture was relieved by excision of scar tissue and application of a pedicle flap, restoring complete extension.

C, Same as B, showing normal flexion of all fingers.

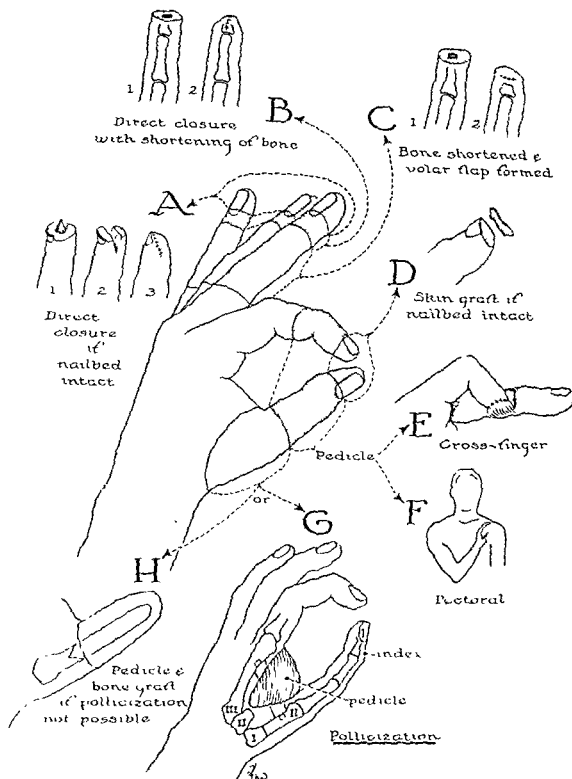


Fig 611—Amputating injuries. Diagrammatic representation of the management of the various amputating injuries of the digits of the hand.

A, Amputation of finger tip with nail bed intact can be closed directly by removing a wedge of bone and soft tissue of the pulp until the edges of the wound approximate.

B, Fingers amputated proximal to the nail bed, with the exception of index and thumb, are closed directly after shortening of the bone.

(Continued on opposite page.)

injuries may show little evidence of the gravity of the damage; however, later, edema and hemorrhage develop throughout the tissues and along the fascial planes. These pools of blood and fluid, if left untreated, impose additional insult to already injured tissues, causing further ischemia and even necrosis.

In the *open type* of crushing injury, the force of compression produces actual bursting of skin and fascia, through which deeper tissues can herniate.

Winger injury is a common example of the crushing type. It is easy to be misled into complacency in seeing a patient with this injury immediately after the accident, because occasionally it takes some hours for the edema and hemorrhage to become evident.

A good policy in management of all closed crushing injuries is to admit the patient to hospital. The involved area is x-rayed to rule out fractures, cleansed with antiseptic solution, wrapped in compression dressing, and mounted on a splint. Subcutaneous or subfascial hematoma should be evacuated immediately and again in 12 hours if accumulation recurs. Injection of hyaluronidase subcutaneously into the most distal part, along with compression and elevation, tends to reduce tissue edema. Such management will often prevent needless loss of skin in cases where damage is not irrevocable. (See also Chapter 30.)

Avulsing Injuries.—This type of injury has some element of crushing but is complicated by a partial or complete loss of tissue. Roller

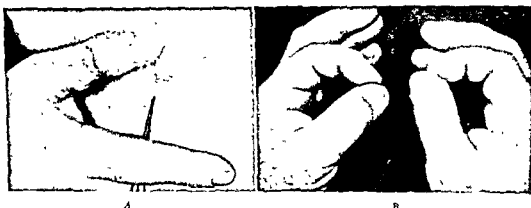


Fig. 642—Amputating injury of the right thumb through the distal joint

A, Definitive repair was carried out immediately by covering the bony stump with a pectoral pedicle flap

B, Appearance of the right thumb 3 months after injury, showing preserved length and good pinching action compared with the normal, uninjured hand

(Legend continued from opposite page)

C, Finger injuries resulting in loss of parts proximal to the distal joint are closed by shortening of the bone and formation of a volar flap of skin, so that the suture line is on the dorsum of the finger

D, Every attempt should be made to preserve the length of index finger and thumb. Amputations of index finger or thumb through the distal phalanx with the nail bed intact are treated by application of free-skin graft

E and F, For injuries proximal to the distal phalanx, if the bed is not suitable for a skin graft, cross finger pedicle or pedicle from pectoral area provides suitable skin cover with the least discomfort to the patient

G, When the thumb is lost, reconstruction of the thumb is possible by transfer of a new web cre

H, When poultization is not possible, the length of thumb can be extended by transfer of skin flap and bone graft. A long period of time is required to attain sensation in such covers

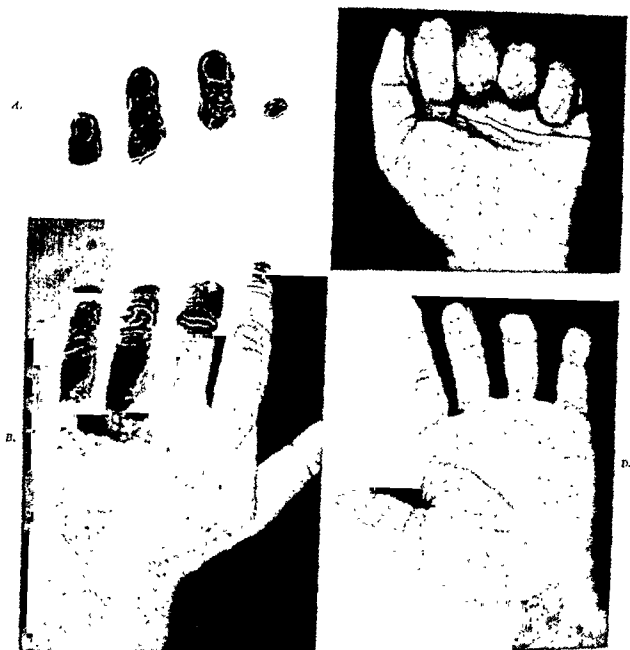


Fig. 643—Amputating injury involving all 4 fingers of the left hand
A. Portions of the fingers of the left hand amputated by power saw
B. Left hand showing level of amputation
C. Immediate repair was carried out with the aid of skin graft to index finger and direct closure of the remaining fingers.
D. Appearance of the hand 3 months after repair

and wringer injuries are common examples of this type, and in complete avulsions the separated skin could be replaced as a full-thickness graft by trimming away fat and other debris. If the avulsed skin is not suitable for reposition, a free skin graft from elsewhere is obtained.

Incompletely avulsed skin that has good blood supply should be replaced in its original position. However, if the skin has been crushed

If the bed from which the skin has been avulsed is not suitable to sustain even a thin graft (bare or fractured bone, open joints, bare tendons, and major nerves), a distant pedicle flap must be used. In a degloving injury involving a finger or the hand, the exposed part is buried into a pectoral or abdominal pocket.

The avulsing injury can be further subdivided into subgroups on the basis of whether

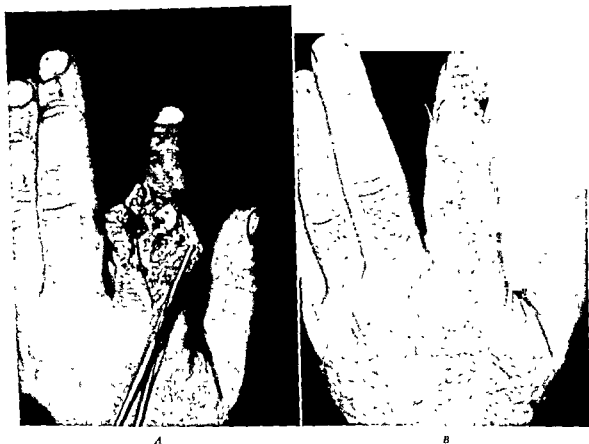


Fig 644—Open type of crushing injury

A, Photograph of the dorsal aspect of the left index finger showing the skin defect and comminuted fracture involving the interphalangeal joint

B, The bony lesion was treated by immediate immobilization with Kirschner wires and direct closure of the defect. Patient ultimately had good pinch and grasp in spite of ankylosis of the proximal interphalangeal joint

or, as frequently happens in flaps with a retrograde attachment, the blood supply is not adequate to sustain the avulsed skin, the flap should be detached and should either be used as a full-thickness graft or be replaced by a fresh split-thickness skin graft

one or both surfaces of the hand are involved. These are *dorsal*, *palmar*, and *dorsopalmar avulsions*. This subdivision is practical because it reflects the increasing severity of injury and thus serves as a guide to appropriate early treatment (see Fig 647).

FRACTURES OF THE UPPER EXTREMITY

Dorsal Avulsions—These are usually produced by roller and wringer injuries and frequently involve damage to tendons or to the joint ligaments. Definitive repair is frequently possible in the injury of this type.

Palmar Avulsions.—A greater force is required to produce this type of injury because the palmar skin is tougher, thicker, and more intimately attached to the underlying structures. Usually only initial treatment is possible, and definitive repair should be delayed to some later date.

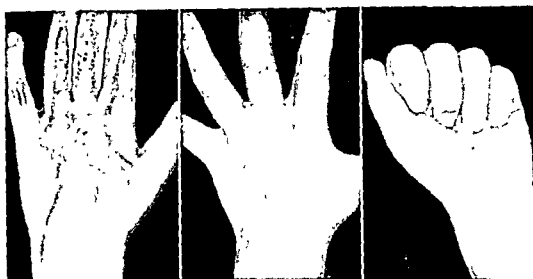
Dorsopalmar Avulsions.—This injury is frequently associated with loss of entire digits, immediate definitive reconstruction is rarely possible in this type.

Mixed Types of Injury.—Like the avulsing type of injuries, the mixed types fall into three subdivisions, based on the predominant localization of the injury. The dorsal injuries usually give better prognosis, whereas dorso-palmar injuries are frequently beyond repair.

Crushing-Avulsing Injuries.—In addition to the compression factor inherent in the crushing type, in the crushing avulsions there is also a severance of important structures, the tissues are torn away from the sources of blood supply; the blood vessels themselves are crushed and torn and become thrombosed, the avulsed skin, mauled by the crushing-compression force, has an impaired blood supply and, consequently, a reduced capacity



Fig. 645.—Crushing injury produced by a laundry wringer in a 68 year old female.
 A, Dorsal aspect of the right hand 2 hours after injury by laundry wringer. Note the extensive edema and subfascial hemorrhage.
 B, Appearance of the hand seen in A one week after injury, following repeated evacuation of hematoma, compression, and elevation.



A

B

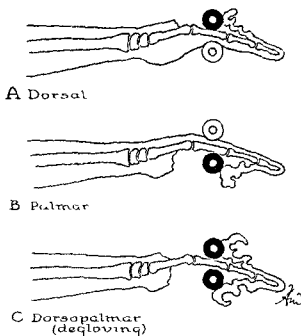
C

Fig. 616—Crushing injury, complicated by loss of skin and scarring

A, Appearance of dorsum of the hand in a 31 2 year old girl, 6 months after injury by a laundry wringer. Scar limits flexion of the fingers

B, Definitive repair was carried out by excision of the entire hypertrophic scar. Skin graft restored good cover and normal function

C, Appearance of the hand 6 months after grafting, showing normal flexion



A Dorsal

B Palmar

C Dorsopalmar
(degloving)

Fig. 647—Avulsing injuries. Clinical classification of avulsing injuries of the hand and forearm based upon whether one or both surfaces are involved



Fig 648—Crushing-avulsing injury of the dorsum of the hand in a 5 year-old boy

A. Right hand showing the extent of avulsion of skin from the dorsal aspect of the thumb, index finger, and hand. The metacarpophalangeal joints were exposed, and extensor tendons to index and middle fingers were severed.

B. The avulsed skin was discarded, and immediate closure of the defects was carried out by a sliding flap from the second web to cover the metacarpophalangeal joint of the index finger and skin grafting of the remaining areas.

C. Definitive reconstruction was carried out 6 months after injury. Widening of the first web was achieved by infraclavicular pedicle flap.

D. Photograph shows the extent of abduction of the thumb after application of pedicle to first web.

E. Appearance of the hand 10 months after injury. Flexion and extension of all fingers normal.

for healing and regeneration and a lowered resistance to infection.

Crushing-Avulsing-Amputating Type of Injury.—This type of injury results from exposure to mutilating machines, such as milling, corn-picking machines, or gears of roller presses. Usually the damage is so overwhelming that only salvaging procedures can be carried out. The initial treatment usually consists of providing skin cover to the remaining parts of the hand. Definitive repair is designed for restoration of some form of pinching and grasping motion.

niques, better understanding of the kinetics of hand function, atraumatic handling of tissues, and specific antibiotics. Although late repair requires specialized training, thorough understanding of the general principles of reconstruction of the hand and skills in providing good skin cover must be acquired even by the uninitiated who plan to carry out surgical procedures on the hand. Otherwise, tissues valuable for later reconstruction would be discarded at the initial treatment or hands left open to granulate, thus jeopardizing the success of later treatment.

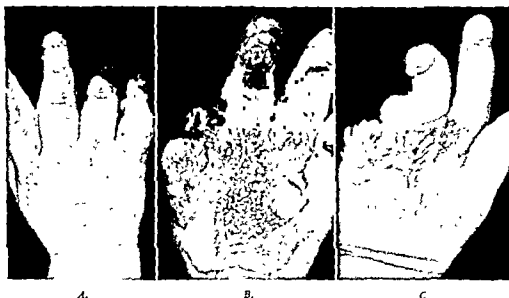


Fig. 649.—Crushing avulsing injury of the dorsopalmar type

A, The dorsal aspect of the right hand of a 1½-year-old boy one week after injury by a laundry wringer. The necrosis of the 2 distal phalanges of ring and little fingers is apparent.

B, Palmar aspect of the hand 2 weeks after injury. The flexor tendons as well as the skin have been destroyed, and denuded bones and joints are seen in the middle and index fingers.

C, In order to salvage this hand, abdominal pedicle flaps were applied to index and middle fingers. The skin graft was used to cover the defect of the palm of the hand. Photograph shows appearance of hand 3 months after injury.

Crushing Injuries, Complicated by Burns.—This type of injury is produced by mangles and heated rollers. The complicating features of thermal injury to the skin make this type of injury rather difficult to treat since a period of waiting has to intervene in order to determine the depth of damage.

Types of Injury Based Upon the Potentiality of Repair

Early definitive surgery has been made possible through the improved surgical tech-

Moreover, as was pointed out earlier, it is important to know the possibilities and potentialities of reconstruction of a given injury of the hand. First, in certain types of injuries, however mutilating, it may be possible to carry out immediate definitive treatment which will provide the patient with a useful hand through the primary operative procedure. Second, some very severe injuries can ultimately give a reasonably good function if judicious initial treatment, reconstituting the skeletal alignment and providing adequate



Fig. 640—Mixed type of injury of crushing avulsing amputating type

A. Dorsopalmar involvement of the hand of a 40-year-old male immediately after injury

B. Palmar aspect of the hand, at the end of the immediate operative procedure. The stump of the thumb was covered by advancing the dorsal flap of the index

skin. The palmar defect was closed by skin graft

C. Palmar aspect of the hand at the termination of the initial treatment 1 month after injury. Definitive reconstruction by pollicization of the index

finger would restore pinch and grasp to this hand

cover of good quality, is carried out. Finally, some tragically mutilated hands cannot be salvaged regardless of the number of procedures, and they should therefore not be subjected to useless surgery.

The following classification has been found useful:

Curable.—Complete restoration of function is possible by one surgical procedure. Most of the incised and slicing wounds and some of the dorsal avulsions fall into this category.

Reconstructible.—This group of injuries requires at least two operative procedures.

Initial treatment entails alignment of the hand in a position of function, possibly repair of severed nerves and covering of all wounds.

Definitive treatment, consisting of procedures aimed at restoration of lost functions, is carried out at a later date.

Salvable.—Milling machine injuries, corn-pickers' hands, severe electric burns, and amputations through the metacarpals are some of the examples of injuries of this type.

Initial treatment would consist of immediate cover if the injury is recent and cleansable. Pedicle flaps may be required to improve circulation in vital parts, as well as to provide cover.

Definitive reconstruction in one or more stages is carried out at a later date to provide some prehensile function.

Non-salvable.—In very severe mutilating injuries any surgical procedure other than amputation is useless. Milling machine injuries, traumatic amputation at the wrist, and some electric burns belong in this group.

PRINCIPLES OF MANAGEMENT

The management of seriously injured hands constitutes a battle between the surgeon and the obstacles that may interfere with good healing and restoration of function. These obstacles are lack of blood supply, threat of infection, replacement of specialized structures by nonspecific scar tissue, and inertia due to immobility.

A number of objectives must be attained for good management of injured hands

1. Proper first aid
2. Accurate assessment of injury
3. Selection of an over-all plan of treatment

4. Minimum number of procedures to attain the maximum of function

5. Correct operative principles

6. Intensive mobilization between operations

7. Definitive reconstruction, if impossible at initial operation

8. Rehabilitation to speed return to useful occupation

The final result of treatment of severely injured hands depend upon the successful achievement of these objectives, and they will now be considered in greater detail.

First Aid.—First aid is very important in the management of a severely injured hand. The most practical method of initial handling is to apply a clean towel with a bulky dressing, maintaining the hand in neutral position, followed by elevation to the level of the shoulder, either in a sling or resting on the chest. Such bulky dressing will serve the triple purpose of stopping the hemorrhage, preventing contamination, and providing support to the injured structures.

Examination of an Injured Hand.—This examination should be done with sterile precautions without undue contamination of the wound by probing or shifting of tissues and without digging for deeper structures. A great deal of information can be obtained by inspection: sensory loss can be assessed by rapid examination of uninjured parts such as finger tips which extend beyond the main dressing covering the wound. Guarded active motion of various parts by the patient helps to determine the extent of motor injury or the severance of tendons. One should not rely on the information supplied by the patient regarding the performance of these movements nor on his impression of perception or sensation.

Planning the Course of Management of the Injured Hand.—Surgery is a two-edged sword: on the one hand it helps to reconstitute deranged tissue and on the other hand it leaves a telltale scar. Several surgical procedures in the same region may leave a wake of cicatrix which almost negates all benefit. Therefore the number of surgical procedures should be minimized, and even the initial operation should form part of a definite plan.

Such a program is based upon the extent and nature of injury, the age and general condi-

tion of the patient, his occupation, time after injury, and the cooperation that can be evoked from the patient himself. The plan consists of the following:

1. Careful estimate of the functional potentialities of the injured hand

2. Initial operative treatment (if definitive restoration of function is not immediately possible)

3. Timing of the subsequent operative procedures, each following a period of active remobilization

4. Definitive repair to restore lost function or later reconstruction to give some useful function to the hand

The over-all purpose of such a plan is to serve as a guide to provide the hand with good sensation, stable parts, and painless motion.

Disadvantages of Too Many Operative Procedures.—It would be theoretically possible to restore any lost function of the hand given enough time and a sufficient number of operative procedures. However, this may not be practical because of the length of time required and because every surgical procedure produces a certain amount of scar tissue. The formation of such scars is cumulative and eventually detracts from what can be gained. For these reasons a minimum of surgical steps should be selected in order to achieve a practical degree of function.

Correct Operative Principles.—The immediate objective of any surgical procedure is the restoration of normal structure and function. This can be attained only through early healing with a minimal amount of scar and the least amount of injury. Closest adherence to the correct principles in operative techniques is required to obtain these aims.

1. Cleansing of severe wounds should be performed only in the operating theater. Adequate scrubbing of the areas around the wound is done first in order to avoid further contamination. Copious quantities of saline or Ringer's solution are preferred for irrigation of the wound. Removal of foreign bodies, debris, and nonviable tissue is done after draping of the area. Detached pieces of bone should not be discarded.

2. Aseptic and atraumatic surgery is carried out with gentle handling of tissues in an avascular field under a pneumatic tourniquet.

3. Meticulous débridement of all devitalized tissue should be done.

4. Stabilization of bony framework should be carried out, preferably by intramedullary pinning, because plaster casts may add further insult to injured tissues.

5. Severed nerves should be repaired immediately if not contraindicated.

6. Scar tissue must be minimized by avoiding edema, hemorrhage, infection, and open wounds.

7. Immediate cover of all wounds should be performed by direct closure, skin graft, or rotating, sliding, or distant flaps.

8. The hand should be placed in the position of function.

Management of Open Wounds Several Days Old.—Old open wounds are always infected. In addition, there is a variable degree of tissue edema and gaping of the wound edges. Such wounds are more difficult to cleanse than the fresh ones and the débridement has to be more extensive. Consequently, judicious preparation of such wounds before surgical intervention by frequent moist saline dressings, elevation, and compression of the limb, alternated with application of proteolytic enzymes (Varidase and the like), speeds preparation of old wounds. A culture should be obtained and specific antibiotics should be given systemically and, if suitable, applied topically.

Intensive Mobilization Between Operations.—Active mobilization after each operative procedure is extremely important for the attainment of maximum function of the hand. Every type of injury requires some degree of immobilization of the various parts of the limb. Restriction of motion leads to undesirable changes even in the uninjured parts: (1) the propulsion of tissue fluids is hampered; (2) the synovial fluid in the joints is reduced in quantity; (3) the collateral ligaments shorten from disuse; (4) the lubrication of the flexor tendons, especially within the sheath, is diminished. Prolonged immobilization leads to narrowing of the joints and produces difficulty in movement, due to stiffness, swelling of parts, and adhesion of tendons.

In injured areas these sequelae are superimposed over the processes of repair of disrupted

parts and may seriously interfere with restoration of motion. Consequently, it is imperative to evoke motion as early as possible in all intact parts proximal and distal to the area of injury.

Initial Versus Definitive Treatment.—The aim of treatment in all injuries of the hand is restoration of normal function and appearance. This can be accomplished in many types of wounds, but in severe injuries immediate restoration of normal function is not possible. However, it is imperative to carry out a number of procedures at the initial operation in order that subsequent reconstruction for restoration of function may be possible. Thus, in severe injuries, the management is divided into two phases

Initial Treatment.—The following procedures should be carried out at the initial treatment if it is not possible to restore normal function

1. All wounds must be closed and defects of skin covered.
2. Skeletal structures should be aligned in normal anatomic position
3. Disrupted nerves should be repaired, if practical.
4. The hand and arm should be placed in the position of function

The immediate aim, then, is early healing and early motion of all parts, followed by definitive treatment in order to restore normal function.

Definitive Treatment.—Definitive treatment is directed toward restoration of normal function and involves provision of a better cover, repair of tendons or tendon grafts, and repair of severed nerves. Occasionally, transposition of muscles may be required in order to provide motion to parts whose own original tendons and nerves could not be repaired.

When restoration of normal function is not possible, one must give to the hand perception and prehension, composed of pinch and grasp, for these constitute a minimum of usefulness of the hand, and without these reconstruction is not really practical.

Rehabilitation.—Earlier definitive surgery has been made possible through the improved techniques, gentle handling of tissues, specific

antibiotics, and early mobilization. However, the treatment of the injured hand is not really completed until the normal or useful function of the hand has been restored and the patient has returned to a productive occupation. The process of rehabilitation of the injured individual begins when the patient is first seen by the surgeon and involves a team, which includes the patient and, in addition, the surgeon, the social worker, the physiotherapist, the occupational therapist, and the employer.

The important phases in the rehabilitation program are as follows:

1. Reassurance to the patient by the surgeon, as early in the treatment as possible, that all efforts will be directed toward regaining useful function of the injured hand
2. Initial or primary repair of the injured hand
3. Intensive mobilization between operations
4. Definitive repair to restore full function or to provide the maximal useful function possible
5. Active exercises designed to provide muscle contraction and joint mobility, progressing to coordinating and purposeful synergistic action
6. Productive occupational and group activity
7. Return to original occupation
8. Selection and training for a new trade

The final outcome of any severe injury is affected by the age of the individual, duration of reconstruction, length of gaps between treatment, and the number of surgical procedures. However, in all long-term cases the surgeon and the other members of the team must continuously evoke an optimistic outlook in the patient, encourage attempts at early motion, and ignite a desire to get well quickly.

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Film References

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Surgical Approaches to the Scapulo-humeral Joint (1949) (By LeRoy C. Abbott, M.D., Donald B. Lucas, M.D., Max B. Shaffrath, M.D., and J. B. de C. M. Saunders, M.B., San Francisco)	36 min	Sound Color	Central Office Film Library, Veterans Administration Vermont Ave. and H St., N.W. Washington 25, D. C.
Surgical Anatomy of the Shoulder The Diagnosis and Surgical Treatment of Shoulder Lesions (Shows dissection of shoulder from posterior, superior, anterior, and lateral aspects supplemented by animated drawings) (1952) (By H. F. Moseley, D.M., Montreal)	45 min	Silent Color	American Cyanamid Co Surgical Products Division Danbury, Conn.
Recurrent Dislocation of the Shoulder (1958) (By H. F. Moseley, D.M., Montreal)	35 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn.
Ruptures of the Rotator Cuff (1949) (By H. F. Moseley, D.M., Montreal)	17 min	Silent Color	American Cyanamid Co Surgical Products Division Danbury, Conn.
Tendon Injuries (Presents a brief demonstration of the healing process of tendons to illustrate the principles underlying tendon suture) (1953) (By Michael L. Mason, M.D., Chicago)	26 min	Sound Color	American Cyanamid Co Surgical Products Division Danbury, Conn.
Stenosing Tenosynovitis at Radial Styloid Process (deQuervain's Disease) (1948)	23 min	Silent Color	Lyon K. Loomis, M.D. 116 S. Jefferson Davis Parkway New Orleans, La.
Surgical Approaches to the Elbow Joint (1950) (By LeRoy C. Abbott, M.D., Robert D. Ray, M.D., Donald B. Lucas, M.D., and J. B. de C. M. Saunders, M.B., San Francisco)	38 min	Sound Color	Central Office Film Library, Veterans Administration Vermont Ave. and H St., N.W. Washington 25, D. C.
Surgical Approaches to Joints of the Wrist (By using animated diagrams and actual dissection, approaches to the joints of the wrist are shown) (1951) (By LeRoy C. Abbott, M.D., Robert D. Ray, M.D., Paul A. Gregorieff, M.D., Philip D. Wilson, Jr., M.D., and J. B. de C. M. Saunders, M.B., San Francisco)	33 min	Sound Color	Central Office Film Library, Veterans Administration Vermont Ave. and H St., N.W. Washington 25, D. C.
Surgical Approaches to the Sternoclavicular and Acromioclavicular Joints (Surgical approaches, anatomy, and operation are shown for each joint by means of animation and dissection) (1952) (By LeRoy C. Abbott, M.D., Donald B. Lucas, M.D., Philip D. Wilson, Jr., M.D., and J. B. de C. M. Saunders, M.B., San Francisco)	18 min	Sound Color	Central Office Film Library, Veterans Administration Vermont Ave. and H St., N.W. Washington 25, D. C.

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Film References—Cont'd

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Obtainable From</i>
Dupuytren's Contracture (1937) (By J. Vernon Luck, M.D., Los Angeles, Calif.)	20 min	Color Silent	American Academy of Orthopaedic Surgeons 116 S. Michigan Ave Chicago 3, Ill.
Flexor Tendon Grafting in the Hand (1953) (By Walter C. Graham, M.D., Santa Barbara, Calif.)	28 min	Color Silent	American Academy of Orthopaedic Surgeons 116 S. Michigan Ave Chicago 3, Ill.
Trigger Finger in Adults and Children (1955) (By John J. Fahey, M.D., Chicago, Ill.)	15 min	Color Magnetic Sound	American Academy of Orthopaedic Surgeons 116 S. Michigan Ave Chicago 3, Ill.
Hand Reconstruction (1946)	17 min	Silent Color	William H. Frackelton, M.D. 324 E. Wisconsin Ave Milwaukee 2, Wis.
Open Injuries of the Hand (2 Parts). (1) Basic Problems, (2) Operating Technique (1956) (By Mr. Patrick Clarkson, England)	23 min 28 min	Sound Color Sound Color	Medical and Biological Film Library, Canadian Film Institute 142 Sparks St Ottawa 4, Ont.
The Anatomy of the Hand, Part I (1956) (By Joseph Markee, M.D., Durham, N. C.)	32 min	Color	The National Foundation for Infantile Paralysis 301 East 42nd St New York, N. Y.
The Anatomy of the Hand, Part II (1956) (By Joseph Markee, M.D., Durham, N. C.)	30 min	Color Sound	The National Foundation for Infantile Paralysis 301 East 42nd St New York, N. Y.

Fractures and Other Disorders of the Lower Extremity

H Fred Moseley, DM

PELVIS

The pelvic cage functions for the protection of the lower abdominal viscera and for the transmission of weight from the trunk to the inferior extremities "Within its borders are housed a portion of the urinary and intestinal systems and the female genitalia, through its foramina pass the great nerve trunks and blood vessels, while beneath its arches, passes all mankind, with few exceptions, the most notable Julius Caesar" (Howell).

Fractures of the pelvis are serious injuries and are frequently complicated by associated trauma to the urethra, bladder, and rectum. In this respect they are comparable to fractures of the skull and spine where involvement of the brain, cord, and meninges constitutes the major problem in therapy.

Mechanism of Injury.—Fractures of the pelvic ring are caused by lateral or anteroposterior compression. The accidents of travel in train, car, and plane account for some. Crushing beneath heavy weights or between cars account for others. Direct falls on the trochanteric area may fracture the acetabulum. Direct violence to different areas causes isolated fractures, e.g., the ilium. Indirect violence by muscular force produces avulsion fractures. Diastasis of the symphysis may occur during parturition or from hyperabduction of the thighs.

Classification.—

A Pelvic ring

- 1 Superior and/or inferior pubic ramus, unilateral or bilateral
- 2 Diastasis of symphysis pubis
- 3 Associated with 1 or 2, fracture through ilium or sacrum on same or opposite side or sacroiliac dislocation

B Acetabulum

- 1 Rim—with posterior or anterior dislocations of the hip
- 2 Floor—with varying degrees of central dislocation of the hip

C Isolated fractures

1 Avulsion

- a Anterior inferior spine by rectus femoris
- b Anterior superior spine by sartorius
- c Ischial epiphysis by hamstrings

- 2 Direct trauma—wing of ilium

D Sacrum and coccyx

Associated Injuries.—

A. Visceral

- 1 Urethra
- 2 Bladder, intraperitoneal or extraperitoneal rupture
- 3 Rectum

B Nervous—Injuries to lumbosacral plexus—S1,2 characterized by the clinical signs:

1. Toe drop
2. Loss of Achilles' reflex

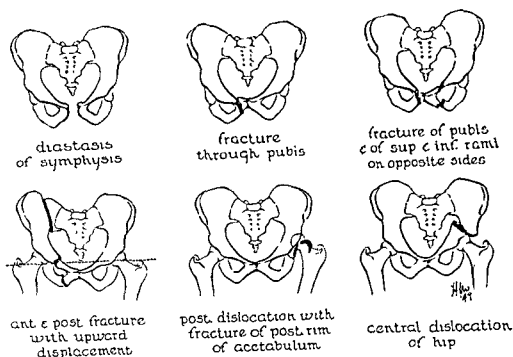


Fig 651—Type fractures of the pelvis

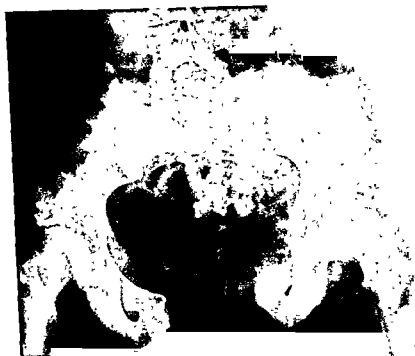


Fig 652—Male patient, aged 28 years, was cleaning the interior of a large cement mixer when the switch was accidentally turned on. Wide diastasis of the symphysis pubis and upward dislocation of the left sacroiliac joint. Ruptured abdominal muscles produced a traumatic ventral hernia. No injury to bowel, bladder, or urethra.

Clinical Picture.—The patient presents the general picture of shock in severe crushing injuries. Visible deformity is not often present. There may be pain localized to the pelvis on attempting to bear weight, or the patient may localize the pain and tenderness to different parts of the pelvis when lateral or anteroposterior compression is applied. Retroperitoneal hemorrhage commonly results from pelvic fractures as from fractured vertebrae. It is indicated by fullness and tenderness in the lower abdomen. Ecchymosis may be visible over this area after a few days, and ileus is a frequent complication.

The diagnosis is accurately made and any displacements of the fragments noted on x-ray examination, which is essential.

Because of the possibility of visceral injury, the patient is asked whether he has passed urine and, if so, whether blood stained. If urine has not been passed, the patient is advised against doing so. The presence of blood at the urethral meatus or a perineal hematoma indicates urethral damage. Suprapubic tenderness or early abdominal rigidity suggests rupture of the bladder (see Chapter 31).

A rectal examination is necessary to ascertain injuries to this viscus, and by this route fractures of the rami may be palpated.

Treatment.—

General.—If it is necessary to move the patient, this is best performed after binding the pelvis to prevent displacement. Therapy directed toward shock may be required. The patient is placed on a firm mattress supported by fracture boards except when diastasis of the symphysis pubis is present. In such cases, sagging of the unsupported mattress assists reduction of the displacement.

Visceral.—Catheterization should be carefully carried out. Failure to enter the bladder indicates rupture of the urethra. If the catheter enters the bladder but fails to drain urine, an intraperitoneal rupture must be suspected. The presence of a small quantity of blood-stained urine suggests extraperitoneal rupture and extravasation (see pp. 883-885 and Fig. 451). Lacerations of the rectal wall found on rectal examination or vesical injuries require laparotomy and repair when possible.

Local drainage, proximal colostomy and antibiotics are associated measures.

Fractures

Pelvic Ring

Fractures of the rami do not displace greatly and unite without residual disability after 3-4 weeks of bed rest.

Fractures of the anterior and posterior sections of the ring without displacement are best treated in bed for 4-6 weeks. A sling support often gives comfort to the patient.

Fractures of the anterior and posterior sections of the ring, with displacement, require treatment in a pelvic sling together with traction on the side of upward displacement of the innominate bone. This correction is best effected with skeletal traction through the lower end of the femur. Six to eight weeks are required for union in the reduced position.

In cases of disruption of the anterior and posterior segments of the ring, caused by anteroposterior compression, the innominate bone hinges outward on a posterior axis like a bivalve mollusc. This displacement is maintained, in the dorsal recumbent position, by the weight of the lower limb. Reduction is best carried out by placing the patient on his sound side on an orthopedic table, compressing the displaced innominate bone forward and medially, and applying a plaster spica to include the lower lumbar spine, the pelvis, and both thighs in moderate abduction to knee level (Watson-Jones). This method is also used for diastasis of the symphysis pubis. When upward displacement is present, downward traction is also required, and this skin or skeletal traction should be retained. Twelve weeks' immobilization is necessary.

Reduction of such displaced fractures is indicated when rupture of the bladder or urethra is present, in order to approximate the torn soft tissues as a preliminary to the urologic procedures of repair and drainage.

Acetabulum

Rim. This is usually part of the treatment of the primary dislocation of the hip. Crepitus may be noted at the time of manipulative reduction, or incomplete reduction or recurrent

dislocation may occur. When radiologic examination reveals a large posterosuperior fragment involving the weight-bearing surface or should recurrent dislocation ensue, operative exposure with internal fixation is indicated. The posterolateral (Gibson) approach affords the best access

Floor. In these cases the problem is how to avoid or minimize traumatic arthritis. When no displacement occurs, the treatment consists of gentle movements with avoidance of weight-bearing for 3-4 months. With central dislocation of the femoral head, clinical examination will disclose some shortening of the limb and

a fracture at the junction of the ilium and superior ramus of the pubis. This can be exposed through the iliofemoral (Smith-Petersen) approach, reduced, and internally fixated. Other types involving the junction of ilium and superior ramus of the ischium can be similarly treated by a posterior exposure

Improvement in the end results is being reported.

Weight-bearing should be avoided for 4-6 months and the progress followed for several years to note the development of aseptic necrosis of the femoral head and traumatic arthritis.

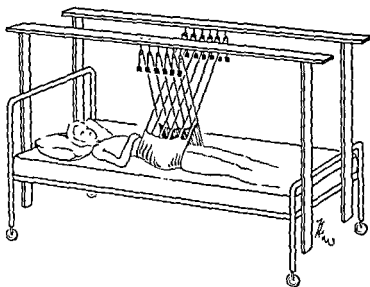


Fig 633.—Pelvic sling

lessened prominence of the trochanter, pressure over which will augment the discomfort, and there will be pain, crepitus, and restricted movement in rotation of the thigh. X-rays will indicate the position of the fracture line and the extent of the protrusion. Reduction is carried out at the earliest moment by strong traction on the abducted leg

Since the end results of such fracture-dislocations are invariably poor due to the mechanical arthritis resulting from incongruity of the acetabulum and frequently associated aseptic necrosis of the femoral head, primary operative reduction has been advocated. Elliott has pointed out that the common type presents

Isolated Fractures

In avulsion fractures rest for 3-4 weeks in the relaxed muscle position is indicated.

Sacrum and Coccyx

Avoidance of pressure and bed rest for 2-3 weeks are required. Fractures of the coccyx are rare, but ligamentous injuries are common and are prone to be followed by a painful neuralgia, i.e., traumatic coccydynia. This is best treated by avoidance of pressure on sitting by use of an air ring supplemented by local diathermy. Excision of the coccyx may be necessary in chronic cases

In all serious injuries of the pelvis, a firm canvas support should be provided when the patient becomes ambulatory. Ambulation on crutches is advisable before full weight-bearing is allowed. Any tendency to pain in the sacroiliac area merits, in addition, massage and diathermy treatments.

Late Complications of Pelvic Fractures.—The late complications are as follows:

1. Malunion with interference, in female patients, with the pelvic canal and parturition
2. Traumatic arthritis of the hip
3. Osteomyelitis following extravasation of urine
4. Sacroiliac pain
5. Traumatic urethral stricture
6. Coccydynia
7. Residual diastasis of the symphysis which results in little disability

THE HIP

The hip is a ball-and-socket joint formed by the articulation of the globular head of the femur in the acetabular fossa of the innominate bone.

The hip joint permits a greater range of mobility than any other joint in the body except the shoulder. At the same time this joint has remarkable stability due to the close fit of the femoral head into the acetabulum as well as to the support of the strongest capsular ligaments and the most powerful musculature of the body.

The hip joint is the most deeply situated of all joints. This increases the difficulty of diagnosis and, unfortunately, renders thorough operative exposure difficult.

The region of the hip is most easily localized by the prominence where the soft tissues overlie the greater trochanter. Anteriorly, the femoral head lies just below Poupert's ligament and lateral to the femoral vessels. The posterior aspect of the hip is bounded by the gluteal or buttock area.

Ligaments

The femoral head is attached at the fovea capitis to the acetabular notch and labrum by the ligamentum teres, which carries a vascular supply (foveolar artery) from the obturator

or medial femoral circumflex vessels to the head.

The capsule of the hip joint is especially strengthened by *Bigelow's ligament*, which extends as an inverted Y from the anterior inferior spine to insert along the full length of the intertrochanteric line. This is the strongest ligament in the body and is rarely ruptured even in dislocations of the joint.

When dislocation occurs, Bigelow's ligament acts as the pivot around which the head of the femur moves into the various characteristic positions to be described later. The ligament as a whole limits extension; its external band limits eversion.

The *pubofemoral ligament* extends from the pubic bone above the obturator foramen to the inner part of the intertrochanteric line. This ligament is made taut by abduction and serves to check this movement.

The *ischiofemoral ligament* passes from the tuber ischii to the upper part of the greater trochanter on its posterior aspect. This band arrests flexion and inversion.

Musculature

The superior and posterior aspects of the joint capsule are covered by a group of small external rotator muscles, i.e., the piriformis, obturator internus and gemelli, the obturator externus and quadratus femoris. Of these the obturator internus must be emphasized because of its great tensile strength and because dislocations of the hip posteriorly tend to displace either above or below this muscle into the gluteal or sciatic position, respectively.

This group of small muscles forms the bed on which the sciatic nerve and gluteal vessels lie. The close relation of this major nerve to the joint explains the frequency of its injury in posterior dislocations.

The glutei minimus and medius strengthen the joint superiorly and laterally.

The capsule on its anterior aspect is covered by the iliopsoas muscle passing to insert on the lesser trochanter. Between the capsule and this muscle lies the psoas bursa, which frequently communicates with the joint cavity.

More laterally the rectus femoris arising from the anterior inferior spine covers the anterior capsule. The most important anterior

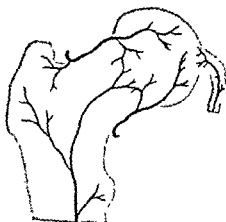
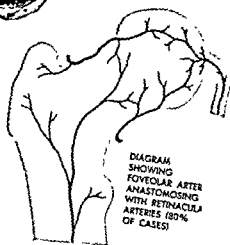
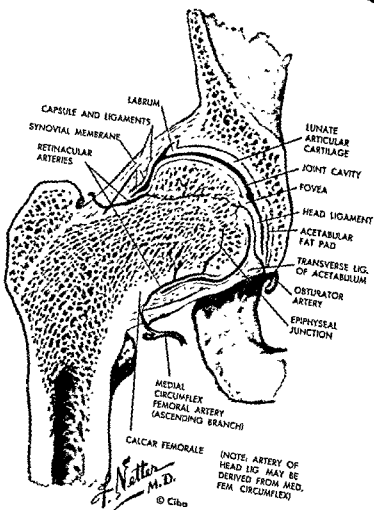
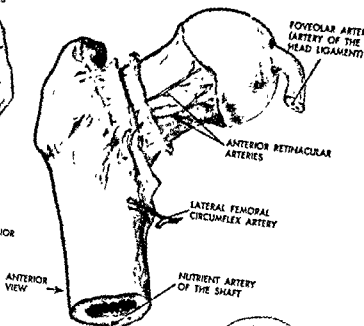
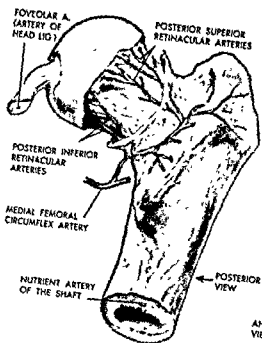


Plate 67.—Blood Supply to Upper End of Femur and Hip.

relations of the hip are the femoral vessels and nerves coursing over the anterior surface of the iliopsoas in the floor of Scarpa's triangle.

Extending superficially over the posterior and lateral aspects of the hip is a layer of aponeurosis and muscle corresponding to the deltoid at the shoulder and made up of the gluteus maximus and tensor fasciae femoris. Between the gluteus maximus and great trochanter is a large bursa which is characteristically involved by tuberculosis and also by calcified deposits similar to those found in relation to the subacromial bursa.

Arteries and Nerves

Blood is supplied to the hip by the gluteal and obturator arteries and by the circumflex femoral branches of the profunda femoris. The obturator and circumflex arteries nourish the acetabulum, head, neck, and trochanteric areas of the femur, whereas perforating branches of the profunda supply the femoral shaft.

The blood supply to the head and neck of the femur is of especial importance. The work of Wolcott, Tucker, and Trueta and co-workers indicates that the main blood supply is through the posterosuperior and posteroinferior retinacular vessels. The anterior retinacular vessels are of lesser importance. The nutrient artery sends branches which anastomose with the retinacular group. Some differences in opinion exist as to the significance of the foveolar vessels. Plate 67, based on Wolcott's observations, indicates that after the age of 14 years anastomoses occur between the foveolar and retinacular vessels in 80% of patients.

The above information should be correlated with the fact that in fractures of the neck of the femur impaction occurs superiorly and posteriorly, preserving the posterior retinacular vessels. It is in the displaced subcapital fractures that the retinacular vessels are most likely to be disrupted or thrombosed, leading to aseptic necrosis of the femoral head.

The muscles of the hip joint are dependent on the lumbosacral plexus for innervation. The iliofemoral group of muscles may be divided into an anterior group (iliopsoas), innervated by the lumbar plexus, and a posterior group (the gluteals, piriformis, and tensor

fasciae latae), supplied by the sacral plexus. The ischio-pubo-femoral muscles, or the lateral rotators of the thigh, also derive their nerve supply from the lumbosacral plexus. In the thigh, muscles are supplied by branches of the femoral, obturator, and sciatic nerves. It is interesting to note that the hip itself is little affected when the sciatic nerve is damaged by a dislocation. Nerve fibers to the muscles of the hip are given off at a high level so that damage from dislocation is shown only distal to the femoral condyles.

Functions of the Hip

The chief functions of the hip joint are (1) transmission of the superincumbent weight of the body to the lower limb and (2) movement at this line of junction.

Because of the transmission of weight, the hip joint is specialized for *stability*. This is in contradistinction to the shoulder joint, which is organized for *mobility*. This emphasis on stability is seen in the depth of the cup of the acetabulum as compared with the shallow fossa of the glenoid. Furthermore, the articular head of the femur protrudes from the femoral neck and is at some distance from the trochanters to which the small muscles are attached, whereas in the shoulder the articular head and tuberosities are continuous.

The weight of the body is transmitted through the roof of the acetabulum to the head of the femur and thence down the neck to the shaft of this bone. In the neck of the femur, the lamellae are architecturally arranged so as to give the greatest possible strength. This arrangement begins to develop in childhood with the beginning of weight-bearing. It attains its greatest development at the most active period of life and is reduced by bone absorption occurring during senescence.

A strong development of compact bone, called the *calcar femorale*, extends along the lower surface of the neck, from which radiating lamellae extend to the upper surface of the neck and head. As age advances, the loss of this bony structure, originally differentiated by stress and strain, constitutes the chief predisposing factor to fractures of the femoral neck.

It is possible that endocrine factors play a considerable role in this process of osteoporosis.

Movements.—The movements of the femur on the pelvis at the hip joint correspond to those of the humerus on the scapula at the glenohumeral joint but are much more restricted in range. These movements are as follows.

1. *Flexion*, which is finally limited by the thigh impinging on the abdomen

2. *Extension*, which permits the thigh to bend behind the coronal plane of the body. This movement is limited by the tension of Bigelow's ligament to about 10-15 degrees. Extension is best tested with the patient prone and the knee flexed at 90 degrees. The diseased hip with a fixed flexion deformity, when tested for this movement, reveals complete absence of extension

3. *Abduction*, which permits separation of the lower limbs. This is limited by tension of the pubofemoral ligaments and impingement of soft tissues between the greater trochanter and acetabular rim. Abduction is best tested with the hips flexed and feet together when the comparison with the normal side is readily made. Most hips when diseased have a fixed deformity of flexion and adduction. Hence, limitation of abduction is an important diagnostic sign

4. *Adduction*, by which the lower limb is brought across the sagittal plane of the body. When fixed adduction is present on both sides, as is seen in spastic paraplegia (Little's disease), the characteristic scissors gait is present

5. *Circumduction*, which is a composite of each of the preceding movements

These five movements constitute all those that occur at the hip joint. However, a complete understanding of the clinical examination and lesions of this joint would be lacking if we did not also consider the rhythm of movements in this region corresponding to the *claviculo-scapulo-humeral* rhythm of the shoulder mechanism. We refer to this as the *pelvic rhythm*, since the pelvis as a unit is moving synchronously with the lumbar articulations and the upper end of the femur, as well as with the knee, ankle, and foot joint mechanisms. This concept is of the greatest importance, since the simple act of bending

forward to touch the floor involves not only the question of hip flexion but also flexion throughout the vertebral column, especially at the lumbosacral joint. Furthermore, any stiffness or restriction of motion in one of these areas is compensated for by increased motion at the other joint mechanisms. An appreciation of these points should greatly assist the student in his examination of the hip joint disordered by injury or disease.

Examination of the Hip

The student may gain his first impression as the patient limps around the ward with or without the support of cane or crutches. The appearance of the older patient of sthenic habitus with excess weight immediately suggests degenerative arthritis in the weight-bearing joints. A careful history will throw light on the duration of symptoms and the disorder to be suspected. Clinical examination is best carried out on an examining table having a rigid surface

Inspection.—Observation of the patient lying supine will reveal the presence of any deformity, such as *eversion* of the whole limb, which is so characteristic of degenerative disease of the hip as well as of fractures of the femoral neck. The inability to flatten the thigh and leg on the couch will suggest the presence of a *flexion deformity*. *Muscular atrophy* affecting the thigh and buttock indicates organic disease of some duration

Before taking measurements for any *disference in limb length*, it is well to level the pelvis by seeing that the line joining the anterior superior spines is at right angles to the midline of the body. Such measurement is made from the anterior superior spine to the internal malleolus on each side.

Palpation.—Manual examination in the region of the anterior aspect of the femoral head and greater trochanter may disclose localized tenderness.

At this point, the level of the tip of the greater trochanter is compared on each side by palpation and also by drawing *Nélaton's line* upon the skin. This line from the anterior superior spine to the ischial tuberosity just crosses the tip of the greater trochanter when the leg is flat on the table.

Upward displacement of the trochanteric area is present in degenerative arthritis of the hip, in gluteal dislocations, in fractures of the femoral neck with displacement, and in non-union of such fractures with characteristic upward riding of the femur.

The shape of Bryant's triangle is another indication of how much, if any, the trochanter has been displaced. This triangle is formed by (1) Nélaton's line just described, (2) a line from the anterior superior spine dropped perpendicular to the examining table, and (3) a line from the tip of the greater trochanter perpendicular to the line just mentioned (or parallel to the examining table). The relative displacement of the trochanter can be determined by a comparison of the shape of this triangle on the affected side with that on the normal side.

Test of Mobility.—It is best first to examine the hip for fixed deformity. With the patient supine, the thigh on the normal side is flexed completely upon the abdomen. With the examiner's hand flat under the patient's lumbar spine, the normal lordotic curve is found to be obliterated. The thigh with knee flexed is held firmly against the abdomen, and the patient is instructed to hold his limb manually in this position.

If fixed flexion of the unsound hip is present, the thigh on the affected side must flex during this manipulation. With the knee on the involved side in extension and the pelvis fixed by the patient as just described, the unsound limb is lowered toward the couch until it reaches a stable position. In this position the angle of the thigh with the table indicates the fixed flexion deformity at the hip. This has been called the *Thomas test* for the demonstration of this deformity.

To determine fixed adduction, the pelvis is first leveled by movements of the lower limbs until the anterior superior spines are aligned perpendicular to the midline. The angle made by the affected limb with the midline of the body when the leg is carried outward as far as it will go will indicate the extent of the adduction deformity. This deformity can also be brought out by testing the range of abduction when the hips are flexed and the feet side by side. As the knees are then pushed apart, any limitation of abduction (indicating

a fixed adduction deformity) on the side of the lesion will be apparent.

By manually moving the sound limb through its various ranges of motion and by repeating this on the side of derangement, a good idea of all the limitations of motion can be obtained.

Radiologic Examination.—X-ray examination should include a view of the whole pelvis, with both hips on one film, taken in the anteroposterior plane. Special oblique and lateral views are also indicated in cases of fracture of the femoral neck or acetabular roof.

The localization of the femoral head in the normal, compared with the displaced, position is determined by x-ray examination (see Fig. 659). Nélaton's line also is invaluable in such determinations.

Examination of the lumbar spine should be included in cases of degenerative disease of the hip, since advanced disease in this vertebral area will contraindicate an arthrodesis for the hip disease. Fusion would throw too great a strain on the lumbar spine and cause pain more intolerable than that from the hip itself.

FRACTURES OF THE NECK OF THE FEMUR

These acute injuries constitute the chief group of emergencies involving the hip. Although these fractures can occur at any age, they are characteristically found in the elderly patient. They occur with increasing frequency from the 6th decade onward.

Fracture of the femoral neck, formerly called the "unsolved fracture," suddenly converts the the ambulatory aged person into a completely bedridden one. If this situation is not corrected, physical and mental deterioration usually ensue. Before the general use of internal fixation, this accident was a frequent cause of death.

Types of Fracture

Subcapital Fracture.—The thin old lady who has passed her biblical span of life is the typical case in whom subcapital fracture occurs. The predisposing factor is senile osteoporosis involving the lamellae of the femoral neck with loss of the *calcar femorale*. A sudden slip on a scatter rug, polished floor, or

linoleum and the bone gives way, the patient sinking to the floor. There the doctor finds her, unable to rise because of pain, with tenderness localized over the femoral head and the limb everted in the typical deformity.

Transcervical and Intertrochanteric Fractures.—These also occur in the aged but are often found in the younger age groups. The mechanism of fracture may be similar to that just described. However, it is frequently due to a forcible fall on the trochanteric area.

On palpation, the trochanter will be found less prominent and more highly placed in the displaced fractures of the subcapital, transcervical, and intertrochanteric types. Nélaton's line and Bryant's triangle can be outlined to confirm this point. In the displaced subcapital fracture, shortening will vary from $\frac{1}{2}$ "-1"; in the extracapsular types, the shortening is still greater.

Movements of the limb are painful. The complete inability of the patient to raise the

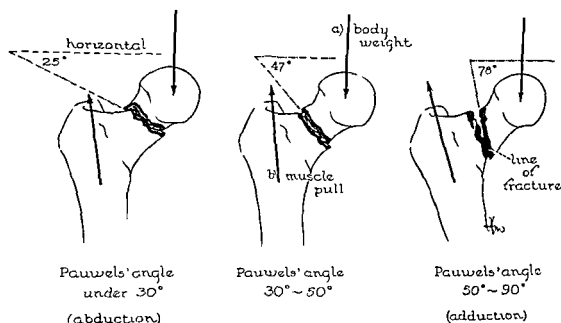


Fig. 634—This illustration stresses the angle formed by the line of fracture to the horizontal. Pauwels showed that in the abduction type of fracture, this angle was under 30 degrees, and it can be seen that the two opposing forces, (a) body weight and (b) muscle pull, tend to impact the fracture. However, when the angle is greater than 30 degrees, there is an increasing shearing force at the site of fracture caused by the opposing forces of body weight and muscle pull. The greater this shearing force, the greater the necessity for internal fixation and the more secure must be the means of internal fixation.

The extent of the eversion deformity is usually greater in the intertrochanteric cases because of the loss of attachment of Bigelow's ligament to the trochanter and shaft fragment.

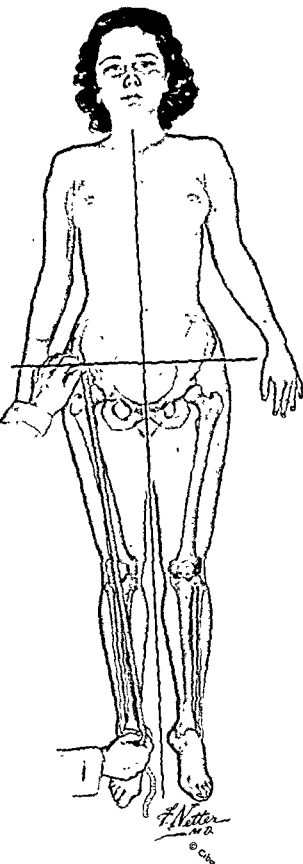
Diagnosis.—Fractures of the femoral neck will be suspected from the history and the age of the patient. Eversion of the limb is characteristic, but it may be absent or replaced by inversion in an occasional case.

The patient complains of pain in the area of the psoas insertion and adductor region, which sometimes confuses the examiner.

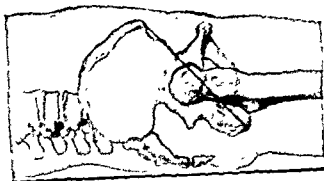
leg from the couch and the localization of pain to the groin on attempts to do this are important points in diagnosis. Acute tenderness can be elicited over the anterior aspect of the femoral neck.

If gradual traction is made, the limb can be readily brought down to its proper length and crepitus elicited by gentle rotation.

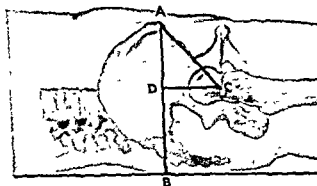
All these findings may be absent in IMPACTED fractures at the different locations in the femoral neck. The student must remember that a patient may actually walk with such a fracture, and if x-rays of the hip are



MEASUREMENT OF LIMB LENGTH (PELVIS LEVELED WITH LINE THROUGH ANTERIOR SUPERIOR SPINES AT RIGHT ANGLES TO AXIS OF THE BODY)

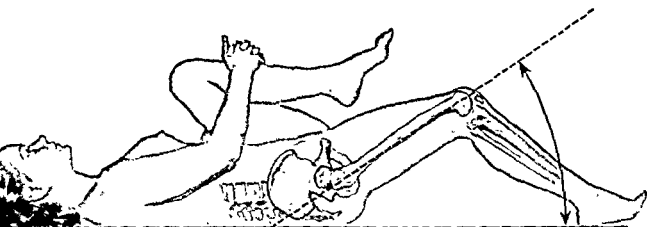


NÉLATON'S LINE

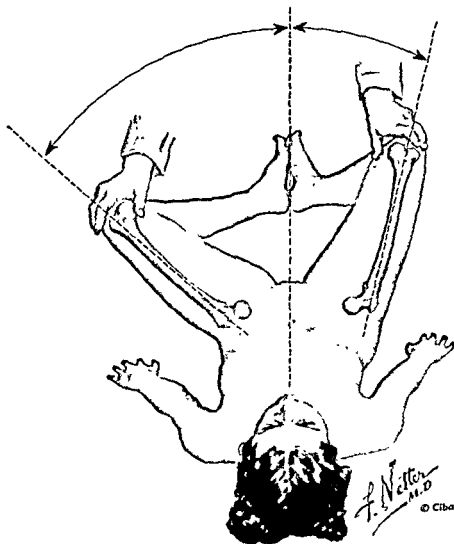


BRYANT'S TRIANGLE (A, C, D)

Plate 68.—Measurement of Limb Length, Nélaton's Line, and Bryant's Triangle.



THOMAS TEST FOR FIXED FLEXION



TEST FOR FIXED ADDUCTION

Plate 69.—Thomas Test for Fixed Flexion and Test for Fixed Adduction.

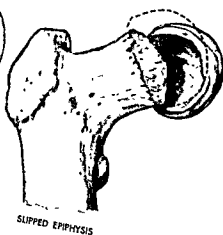
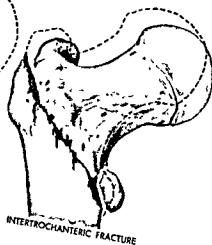
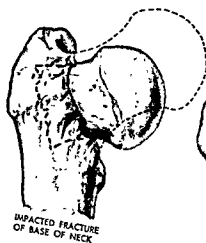
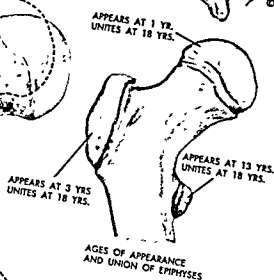
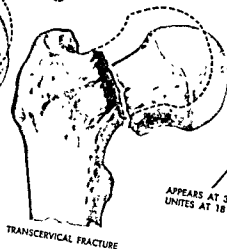
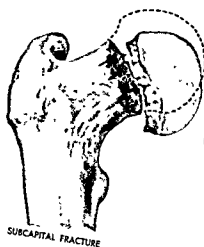
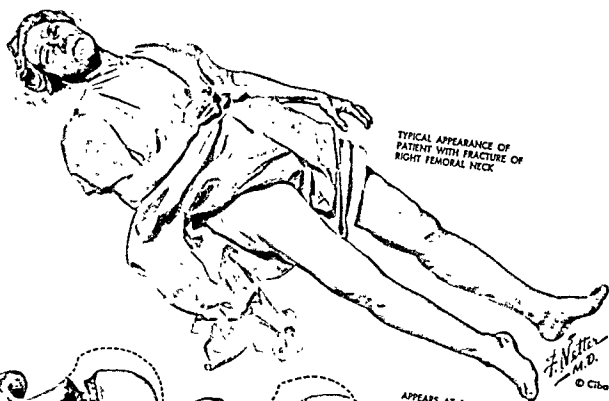
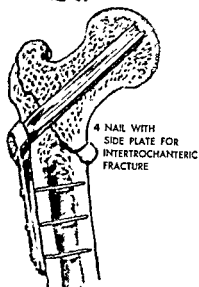
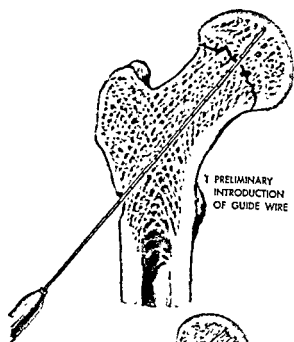
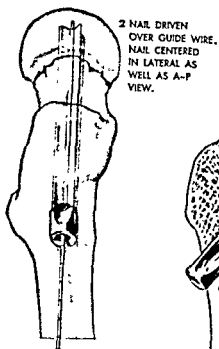


Plate 70.—Fractures of the Neck of the Femur.

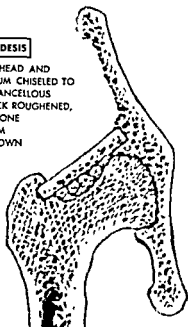


THE SMITH-PETERSEN NAIL

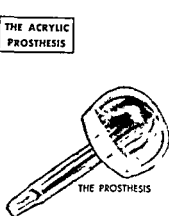


ARTHRODESIS

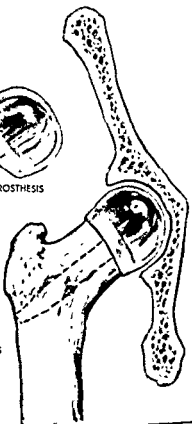
FEMORAL HEAD AND ACETABULUM CHISELED TO EXPOSE CANCELLOUS BONE. NECK ROUGHENED, FLAP OF BONE FROM ILIUM TURNED DOWN



THE ACRYLIC PROSTHESIS



FEMORAL HEAD REMOVED, NECK DRILLED AND PROSTHESIS DRIVEN INTO PLACE.



A. Netter M.D.
© Ciba

Plate 71.—Operative Procedures on the Hip.

The acrylic prosthesis has now been replaced by models of Vitallium or stainless steel with a shaft extending into the medullary canal (See Fig 656.)



Fig. 655—*A*, Prior to operation. *B*, One year after operation. Comminuted fracture of neck and head of femur with aseptic necrosis appearing after application of pin and plate for internal fixation. There was probably injury to the cervical branches of the medial femoral circumflex vessels at the initial injury. Aseptic necrosis and nonunion with fracture of screws are present one year after operation.

C and *D*, Subcapital fracture 2 years after insertion of Smith-Petersen pin, showing union and loosening of pin, track visible in lateral view, early extrusion.

not taken in such cases, unfortunate medico-legal complications may ensue.

The clinician must, therefore, arrange for x-ray examination of all patients suspected of having fracture of the hip

Emergency Treatment.—The patient should be transported in a Thomas splint arranged with fixed traction. Morphine 15 mg. should be given to relieve pain.

Definitive Treatment.—The introduction by Smith-Petersen of the three-flanged nail has been one of the great contributions to fracture treatment. Although other methods of

tality of about 25%. With the proper use of the Smith-Petersen nail, union can be expected in 75-80% of cases, with a mortality of about 10%. These statistics are only rough; however, they are sufficiently accurate to give an indication of what can be expected from this method of treatment.

The displaced fracture of the subcapital type will not unite without reduction and internal fixation. Reduction consists of traction on the flexed thigh while adducted and externally rotated. This is followed by movement into internal rotation, abduction, and extension. If



Fig. 656—A. Aseptic necrosis of femoral head following subcapital fracture treated by Smith-Petersen nail. Note track from which the nail has been removed.

B. Result following excision of head and replacement by Moore prosthesis.

internal fixation, such as multiple pins, lag screws, bolts, etc., have been successfully employed, most surgeons rely on the Smith-Petersen nail.

Prior to the introduction of this method, the use of a Whitman plaster cast secured union in possibly 40% of cases, with a mor-

tal reduction is stable and the heel of the affected extremity is placed in the surgeon's palm, the limb will not immediately fall into the externally rotated position. This reduction has been called *Leadbetter's maneuver* and the test for stability, *Leadbetter's test*. The author has not found this test of great practical value.

In these cases, therefore, the nail should be expertly inserted at the earliest possible moment. Most surgeons will agree that little is gained by delay, and elderly patients withstand the operation well.

Recently, in the *adduction* type of subcapital fracture in the patient 70 years or older, the use of a prosthesis has been tried because this type of fracture in the aged has been the one with the greatest percentage of nonunion and the longest periods of immobilization. With the prosthesis, the patient is immobilized for only 3 weeks and thereafter is encouraged to move about freely. Because of the short life expectancy in such cases, this method holds considerable promise.

The *impacted* subcapital fracture will unite without internal fixation but should be protected by a plaster spica in the more vigorous patients. In the elderly patient, it is probably safer to insert a nail to obviate the possibility of a later displacement.

Transcervical and *intertrochanteric* fractures do not present the same difficulties in securing union. However, in the hands of specialists, the use of internal fixation causes the least inconvenience to the patient and makes the problem of nursing much easier. An *intertrochanteric* fracture requires a side plate in addition to the nail, since the tongue of bone extending from the shaft is usually not sufficiently strong to support the nail.

In the younger group of patients presenting these fractures, reduction and fixation may be secured by the use of skeletal traction arranged through a Kirschner wire in the lower end of the femur. This must be maintained in place for 8-12 weeks. Sometimes a sufficient degree of union has occurred in 6-8 weeks to warrant change to a plaster spica, which will permit the further bed confinement to be carried out at home. Progress of union and position should be followed in these cases by repeated x-ray examination.

Postoperative Therapy.—If the fracture is well nailed, patients with the above types are allowed to move freely in bed. Six to eight weeks of such confinement is conservative in this type of treatment. If necessary, the patient may be allowed to sit up in bed immediately or be lifted into a chair. This will obviate

pressure sores from prolonged rest in one position.

Ambulation on crutches is determined by the patient's capacity. Often this is too dangerous to consider.

Union occurs in 3 months in the *intertrochanteric*, 4 months in the *transcervical*, and 4-6 months in the *subcapital* fractures. However, these are only arbitrary time factors, and union is best judged by radiologic examination. Internal fixation is only a means of maintaining position. Time and the healing process are the essentials for union.

The nail need not be removed except when complications develop.

Physiotherapy in the Elderly Patient.—In all elderly patients with fractures of the femoral neck, the physiotherapist can supervise deep-breathing exercises from the beginning to prevent postoperative pulmonary complications.

With the use of a beam over the bed, the patient can be taught to assist her own movements when a bedpan or change of sheets is required. With a well-nailed fracture, the therapist can encourage movements of both lower limbs from the beginning to obviate venous stasis and the possibility of phlebotrombosis and pulmonary embolism. The patient can be assisted to sit on the side of the bed and even to a chair shortly after operation. Once the stitches are out and the wound is soundly healed, a walker can be used. The exact timing of graduated activity will be determined by many factors, including the general capacity of the individual, the type of fracture, and the security of the fixation obtained by the nail and plate.

Complications.—

General.—A fractured hip in the senile patient is still an all-too-frequent cause of death. The initial shock, together with the early development of pulmonary, cardiovascular, or renal complications, is often too much for the frail body to bear. Before the internal fixation technique was employed, pressure sores from long confinement to bed were additional factors disposing the patient to deterioration.

Local.—The most important local complication of the fracture is *nonunion*. This is most common in the *subcapital* type but is rare

in the transcervical and intertrochanteric types, which unite well although sometimes with a *varus deformity*. The nonunion is often associated with *avascular necrosis* of the capital fragment. The initial displacement ruptures the cervical ligaments that carry the main vascular supply, leaving only an inadequate supply via the ligamentum teres. The density of the head is increased as seen on x-ray, and if weight-bearing is permitted, collapse of the head and traumatic arthritis ensue.

In those cases in which the nail does not secure effectual fixation of the fragments, a shearing force is exerted at the fracture line and *absorption of the femoral neck results*. This is a frequent cause of nonunion and of extrusion or fracture of the nail.

Treatment of nonunion of femoral neck fractures depends upon the viability of the head, absorption of the neck, and general factors such as the patient's age, general health, and expectation of life. In the senile patient, if treatment because of pain is necessary, the most promising results are from the use of a prosthesis. In the younger patient with a viable head, bone-grafting operations or a McMurray osteotomy can be used. An arthrodesis can be performed if immobilization for 3-4 months can be justified.

Complications of Nailing.—

Extrusion of the Nail.—In cases where aseptic necrosis of the head develops or where a shearing force at the line of fracture occurs, the nail moves in the line of least resistance. In cases where the head of the nail is fixed to the shaft, the nail will penetrate into the joint. Where it is not so fixed, the nail will be extruded. This is due to the fact that the more vascular bone of the shaft area atrophies more rapidly around the nail and therefore permits the extrusion. Treatment depends upon the individual case and follows the principles outlined under nonunion.

Penetration of the Nail.—In cases where the nail has been hammered too far and has penetrated the joint, withdrawal is required because of the painful arthritis which develops.

Fracture of the Nail.—Occasionally the nail may break and require removal or replacement.

End Results.—A follow-up of 242 cases admitted to the Royal Victoria Hospital for the years 1951-1955, inclusive, has recently been completed by Dr. I. Bitenc.

TYPES AND INCIDENCE

Femoral Neck (108)

Impacted subcapital (valgus)	11.1%
Vertical (Pauwel's III)	11.1%
Transverse subcapital	51.8%
Transverse mid-cervical	24.1%
Basocervical	1.9%

Trochanteric (134)

Intertrochanteric	25.2%
Intertrochanteric (comminuted)	51.8%
Intertrochanteric and subtrochanteric	7.8%
Subcervical (reversed intertrochanteric)	2.6%
Subtrochanteric	8.6%
Greater trochanter	4.0%

99 patients showed signs of advanced cardiovascular disease

18 patients were diabetics

18 patients had other fractures

8 patients presented pathologic fractures

Highest incidence of neck fractures occurred in 8th decade—female:male = 4:1

Highest incidence of trochanteric types occurred in 9th decade—female:male = 2:1

Treatment.—Patients moribund on admission were afforded palliative treatment. Whenever possible, internal fixation was employed at the earliest moment. Displaced neck fractures in patients of advanced age were sometimes treated by prosthesis or excision of the head. Impacted subcapital fractures were treated both conservatively and by internal fixation.

Results.—In displaced femoral neck fractures treated by internal fixation, union occurred in 87.7%, avascular necrosis in 14.2%, operative mortality being 3.1%; mortality in cases treated conservatively was considerably higher.

In 12 impacted subcapital fractures, all united; one developed avascular necrosis.

In the trochanteric types, all united whether treated conservatively or by internal fixation. The mortality was halved by the operative treatment, 8.1% and 16.2%, respectively.

The mortality in those operated upon was least when the operation was performed within 48 hours and increased considerably thereafter.

SEPARATION OF UPPER FEMORAL EPIPHYSIS (ADOLESCENT COXA VARA)

This disorder occurs typically in the Fröhlich type of endocrine obesity in the age range 10-16 years. It will be remembered that the capital epiphysis fuses with the neck about the 19th year.

The condition may occur acutely, but more often it develops gradually. The displacement is frequently bilateral. However, in such cases one hip is usually involved before the other.

The onset may follow different degrees of trauma but may also occur spontaneously. The soft tissues are intact, but the capital epiphysis rotates backward as the neck and shaft fragments rotate forward.

Diagnosis.—The patient is usually an overweight adolescent 10-16 years of age, with a history of pain in the groin. Whether acute or chronic in nature, the earliest possible radiologic examination is required. Anteroposterior views are not adequate to demonstrate early



Fig 657—Male patient aged 16 years. Case of delayed puberty with early slipping of left femoral epiphysis.

A, Anteroposterior view of both hips. Little abnormality to be seen.

B, Oblique view of abducted hips. Note inferior displacement of head on left side.

C and D, Similar views showing result after Smith-Petersen nailing.

displacement, lateral views are essential to show the projection backward of the capital epiphysis.

Treatment.—A period of observation in the orthopedic ward is essential. If early diagnosis by x-ray is secured, internal fixation by a Smith-Petersen nail is the accepted treatment. Should displacement be marked, reduction by gentle, graduated traction is preferred to forcible manipulation, which may interfere with the circulation to the head and lead to aseptic necrosis.

With reduction secured by traction, plaster immobilization or preferably internal fixation may be used. Supervision must be continued until epiphyseal union has occurred. X-ray films of the opposite limb should be made repeatedly because the process is frequently bilateral. The avoidance of weight-bearing during the period of waiting for synostosis of the epiphyseal plate is one of the essentials of treatment.

TRAUMATIC DISLOCATION OF THE HIP

These major accidents are much less commonly seen than are fractures. A dislocation usually occurs in the younger age group. It is rare in the elderly patient because in such a person the bone is relatively weaker than the joint capsule. Dislocations occur chiefly in automobile accidents. The so-called "dashboard dislocation" is the one seen most commonly.

Types of Dislocation.—It is a surprising fact that little can be added to the descriptions of dislocation given by Hippocrates. However, we are greatly indebted to Bigelow, whose studies on this subject published in 1869 remain the classic today. This surgeon emphasized the significance of the inverted Y-shaped ligament of the hip, which bears his name, and demonstrated that most dislocations leave this ligament intact to act as a controlling band around which the head rotates. Bigelow referred to the dislocations with this ligament intact as *regular* in type and those with rupture of this ligament as *irregular*.

The regular dislocations occurring clinically are characterized by the same position of the femoral head in relation to the acetabulum and the same corresponding deformity of the

limb that occurs when the identical dislocations are produced on the cadaver. Bigelow also showed that reduction can best be secured by circumducting the limb around this ligamentous pivot. With the ligament of Bigelow ruptured, however, the head assumes irregular positions determined by the direction and intensity of the force that has been applied.

Bigelow's studies were continued by Allis, who published his observations in 1896.

For our purposes, it will be sufficient to recognize four positions of the regular dislocations:

1. Posterosuperior—also called dorsal or gluteal
2. Posteroinferior—below the tendon of the obturator internus—also called sciatic
3. Anterosuperior or pubic
4. Anteroinferior or obturator

Variations of these will be found when one or both branches of Bigelow's ligament have been ruptured. Most classifications describe the fracture of the acetabular floor, with varying degrees of pelvic protrusion of the femoral head, as *central dislocation of the hip*.

Diagnosis.—The diagnosis will be made on the history of a serious accident, often with multiple injuries, of which the hip is one site. The patient will usually be of the younger age group and almost certainly younger than 50 years of age.

In the *dashboard dislocation*, caused by direct force against the knee with the thigh flexed and adducted, the femoral head is driven out into the gluteal position. This type is frequently associated with a fracture of the acetabular roof in its weight-bearing portion. This is a most serious complication, and if the fragment is large, internal fixation of this weight-bearing surface in perfect position will be required to prevent traumatic arthritis after closed reduction of the dislocation has been performed.

The femoral head will be palpated in its dorsal position; the limb will be shortened and adducted. X-ray examination will confirm the diagnosis and position.

Other types of dislocation can be diagnosed by the abnormal position of the head, the abnormal shortening or lengthening of the limb, and the x-ray appearances.

*A**B**C**D*

Fig. 658—Various types of dislocation
A, Posterosuperior or dashboard
B, Anterosuperior or subspinous

C, Anteroinferior or obturator
D, Central

Treatment.—The first important point is to secure complete relaxation of muscle spasm by spinal or general anesthesia, which should be induced at the earliest possible moment after the accident.

DORSAL DISLOCATIONS.—These are the more frequent. Three methods of reduction are available:

Gravity Method.—The patient is placed face downward on a firm surface, with the affected thigh and leg flexed at right angles over the side of the table. Gentle, graduated traction to the thigh will usually secure reduction.

Method of Allis.—The patient is placed supine on a firm mattress on the floor.

1. Flex the thigh to 60-70 degrees and the knee to 90-100 degrees. The pelvis is fixed by an assistant. Gentle traction is applied to the thigh.

2. Apply force sufficient to lift the head into the acetabulum. If resistance is felt, give gentle rotation inward, then outward, and follow this by lifting upward.

3. The limb is now extended while the lifting force is maintained. Reduction usually occurs at this point.

Circumduction Method of Bigelow.—The position of the patient and limb is as for the method of Allis.

1. The limb is first flexed, rotated inward, and adducted, which relaxes the taut capsular ligaments.

2. While maintaining traction, the limb is abducted, rotated outward, and extended in one continuous movement.

All surgeons agree that, with complete muscle relaxation, reduction can be secured with single or repeated manipulations without the use of excessive force, which would further damage the capsule, vascular supply to the head, and the sciatic nerve.

ANTERIOR DISLOCATIONS.—These can be reduced by the methods of Allis or Bigelow as follows:

Method of Allis.—Abduct the slightly flexed thigh while the assistant steadies the femoral head with his fingers. While applying gradually increasing traction to the thigh, adduct the limb as the assistant pushes the head laterally into the socket.

Method of Bigelow.—The limb is first flexed to a moderate degree, and with gentle

traction the abducted and externally rotated limb is successively adducted, internally rotated, and finally extended.

Postreduction Treatment.—The affected hip should be placed in balanced traction for 3-4 weeks. After this time the patient should be instructed in the re-education of hip movements. Because of the high incidence (as high as 50%) of aseptic necrosis and traumatic arthritis, differences of opinion exist as to the optimal time at which weight-bearing should be permitted. However, it would appear that graduated return to full weight-bearing can be permitted after 4 weeks, since little evidence exists that prolonged rest minimizes these sequelae.

Complications.—Sciatic nerve paralysis found at the initial examination is a serious complication. This and fracture of a major part of the acetabular roof afford the chief indications for operative exploration.

Late cases presenting traumatic arthritis with marked symptoms will require relief by arthrodesis or arthroplasty, depending on the indications in the particular case.

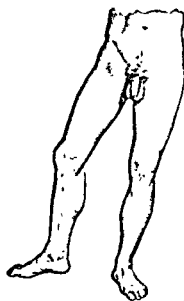
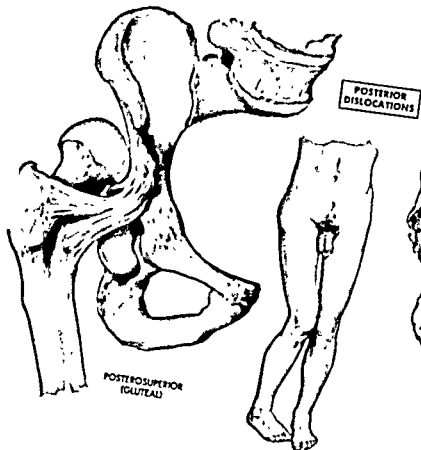
CONGENITAL DISLOCATION OF THE HIP

The hip is the joint which most frequently presents a congenital dislocation. It occurs more frequently in girls than in boys, in the ratio of 4:1, and 1 out of every 3 cases presents a bilateral lesion. There is a geographic and racial incidence noted. This congenital anomaly is especially common in northern Italy, where Putti made extensive studies and contributions on this subject. It is rare among Negroes. There appears to be a hereditary tendency to this defect.

The term congenital dislocation includes the various degrees of displacement of the head from its normal position and thus includes subluxations.

Pathologic Anatomy.—The acetabulum is shallower, and its roof presents a greater obliquity, than normal. There is a postero-superior defect of the acetabular rim. In many cases the acetabulum is obliterated by fibrous tissue and capsular adhesions. There is a false joint above the acetabulum on the dorsum ilii.

The upper end of the femur is also abnormal. The epiphysis of the head, which



ANTERIOR
DISLOCATIONS

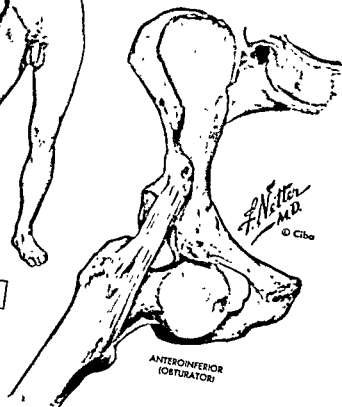


Plate 72.—Dislocations of the Hip.

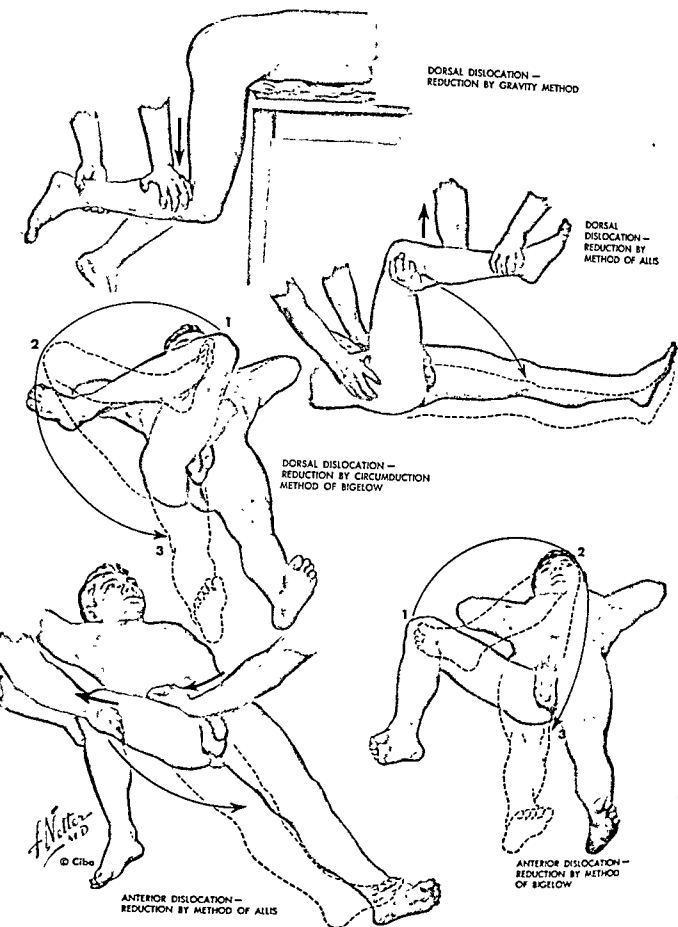


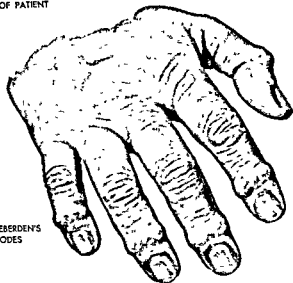
Plate 73.—Reductions of Dislocated Hip.



CHARACTERISTIC
HABITUS OF PATIENT



DEGENERATIVE CHANGES IN ACETABULUM
AND FEMUR IN OSTEOARTHRITIS



HEBERDEN'S
NODES



Plate 74.—Osteoarthritis of Hip.

*Courtesy Moseley, H F: CIBA
CLINICAL SYMPOSIA 5 35, 1953*

appears in infants 6-8 months of age, is hypoplastic and sometimes undergoes an osteochondritic process. In the early stages before ambulation the head is more laterally placed from the floor of the acetabulum than normal. In the later stages the shape of the head is altered from pressure changes, being flattened on its medial and posterior aspects. The normal anteversion of the neck is increased and the neck points almost directly forward. There is an increased neck shaft angle. In patients 20-30 years of age the x-ray appearances of degenerative arthritis, characterized by cystic areas and osteophytic changes, are noted in the head.

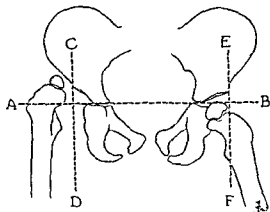


Fig. 659.—The method for localization of the head of the femur in relation to the acetabulum. AB = horizontal line through points of fusion in the acetabula. CD and EF = perpendicular lines drawn from the superior rims of the acetabular roofs. Normally, the head lies inferior to the horizontal line and medial to the vertical line. In congenital dislocation, as shown on the right side, the head lies superior to the horizontal line and lateral to the vertical line. The head, in such cases, is usually late in development.

The capsular changes have been studied at operation, autopsy, and lately by arthrography. In some cases the capsule is drawn out in hourglass arrangement, and part of the capsule intervenes between the head and the false socket on the ilium. The hourglass constriction permits the passage of the ligamentum teres carrying part of the blood supply to the head. This constriction is a barrier to manipulative replacement, as it intervenes between the head and acetabular socket on closed reduction.

It is generally agreed that before ambulation has occurred the head is usually capable of being replaced in the acetabulum by abduction of the limbs. With the passage of time, adaptive structural changes occur which make anatomic position more difficult to obtain and retain. Early diagnosis will depend on the careful examination of the hip joints of all infants with radiologic studies, if deformity or limitation of movements is found. If the preluxation state is demonstrated, treatment can be initiated at the earliest moment.

Clinical Features.—

Preambulant Period.—The radiologic examination is the key to the discovery of the preluxation. At birth an increased obliquity of the acetabular roof can be noted. At 1 month any increased lateral displacement of the upper and inner corner of the femoral metaphysis may be seen and after 6-8 months the delay in the development of the upper femoral epiphysis or its smaller size can be demonstrated.

Ambulant Period.—As soon as the child begins to walk, a definite limp will develop. In the unilateral case there will be lurch to the affected side. There is a *positive Trendelenburg test* which means that when the child stands on the affected leg and raises the other from the ground, the pelvis will lower on the normal side because of the lack of the stabilizing influence of the gluteal muscle group. Clinical examination will also disclose an increased number of folds on the medial and posterior aspects of the thigh. Palpation will reveal the absence of the head in Scarpa's triangle, and the femoral pulsation will be more difficult to obtain for this reason. The head may be palpated in the gluteal area. Movements of the hip are altered, and there is limitation of abduction and internal rotation with increased adduction and external rotation. It may be possible to telescope the upper end of the femur. There is a greater than normal prominence of the great trochanter, and measurements will reveal that the trochanter rides above Nelaton's line, joining the anterior superior spine to the ischial tuberosity.

X-ray at this period will show that the head is more superiorly and laterally placed than normal and the line of Shenton is broken.

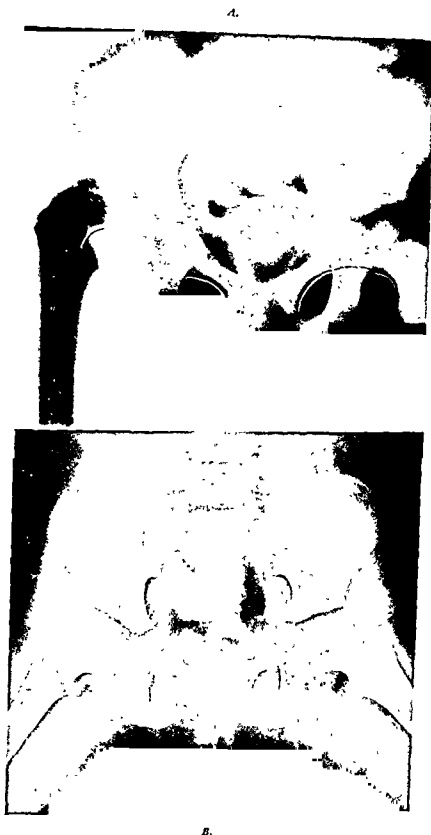


Fig. 660—A. Congenital dislocation on right side. Shenton's line, which is shown in its normal arrangement on the left side, is broken on the right side.

B. The same case after reduction by traction. Note the increased slope of the acetabular roof and the delay in the development of the femoral head on the right side.

In the bilateral case the child walks with a waddle. There is greatly increased lumbar lordosis and protuberant abdomen, giving the appearance that the trunk is falling forward on the posteriorly displaced hip mechanisms. There is a bilateral positive Trendelenburg test.

Diagnosis.—The work of Putti has shown that by public education this anomaly can be diagnosed in the early months of life if the possibility is suspected and radiographic studies are made. Any infant presenting limited abduction of the flexed hip merits investigation and supervision. After ambulation has begun the diagnosis is made on the characteristic limp and clinical features mentioned above.

Treatment.—If this congenital anomaly is diagnosed in the preluxation period, positioning the limbs in abduction and internal rotation on various type of mattresses, cushions, or braces for periods of 6 months or more can effect a cure. This is only of value within the first 6 months of life.

After this time, the treatment consists of gentle reduction of the dislocated head by traction followed by positioning the limbs in full internal rotation until stability is secured, which averages 9 months. This position is maintained by plaster fixation which is changed at 3 monthly intervals. As soon as the reduction is stable, dynamic splinting in abduction may be employed instead of plaster fixation (Denis Browne).

In those cases that relapse or where reduction was impossible, the displacement may be corrected by open operation. This group often shows a high degree of anteversion of the neck which requires correction by external rotation osteotomy.

When the patient presents later with such dislocations a variety of reconstructive procedures may be employed.

OSTEOARTHRITIS OF THE HIP (DEGENERATIVE ARTHRITIS, MALUM COXAE SENILIS)

Apart from the traumatic disorders of the hips, which are chiefly the fractures and dislocations previously discussed, osteoarthritis of this joint is the lesion most frequently encountered. This is a progressive and disabling

disease, more common in the male than in the female patient, which occurs with increasing frequency from the 5th decade onward. There is a constitutional predisposition to this form of arthritis. It tends to occur in families of the sthenic habitus with a tendency to be overweight. Their joints are of the close-knit, stable variety, and the summation of minor and major traumas, the wear and tear of daily use, and the effects of aging all play their part in its development.

Macroscopic Joint Changes.—Articular cartilage changes are most marked on the non-weight-bearing area of the head, where the cartilage becomes fibrillated, softened, and finally desquamated, exposing the bare bone. The bone becomes dense, polished, and eburnated.

At the periphery of the head, excess production of *enchondral* bone takes place to give the exostoses, or lipping, which accounts for the term *hypertrophic* frequently applied to this type of arthritis. These changes in the articular cartilage are reflected in the x-ray, which discloses a diminution of the joint space and lipping on the head and acetabular rim.

The *subchondral bone* undergoes atrophy and cyst formation. The bone marrow shows *fibrosis* and fatty infiltration. The *synovial membrane* presents signs of chronic inflammatory change. Villous processes develop. The synovial fluid loses its mucous character and becomes more serous in consistency. The *joint capsule* and *ligaments* lose their elasticity and become thickened from fibrous tissue proliferation. The *surrounding muscles* undergo contractures and the hip assumes a position of deformity characterized by *flexion*, *adduction*, and *external rotation*. The muscles of the buttock and thigh atrophy from disuse.

Clinical Picture.—A middle-aged man walking with a limp and supported by a cane is the typical picture. Usually of the sthenic habitus and carrying excess weight, he comes for medical aid, complaining of increasing pain and stiffness in his hip and thigh. At first the pain is an ache on overuse and is noted only at the end of the day. It is relieved by rest from weight-bearing. In time the ache becomes more constant and reaches a stage where *night pain*

interferes with sleep and relief is difficult to secure. The pain is aggravated by dampness, cold, overuse, and postural strain. Often it increases with work in the patient who is of the laboring class.

As the pain and stiffness increase, the typical deformity of eversion, flexion, and adduction develops. This places a strain on the lumbar spine, and an associated low backache is a frequent complaint.

In some cases, there is a history of adolescent coxa vara, Perthes' disease, or a previous dislocation or fracture, which has contributed to premature wearing out of the joint. Initially, the disease is unilateral, but in time the opposite hip is also affected



Fig. 661—Osteoarthritis of the hip. Note loss of joint space, sclerosis of the head and acetabular roof in the weight bearing portions, as well as cystic changes in these areas and osteophytic formations at the acetabular rim.

Radiologic Examination.—The early x-ray picture is one of diminution of the joint space, with cystic areas of decreased density in the subchondral zones of the weight-bearing areas of the head and acetabular roof. Lipping on the periphery of the head and acetabular rim is seen in later stages, and the joint space

appears to be absent. However, bony ankylosis practically never occurs.

Diagnosis.—The diagnosis is readily made on the history of gradually increasing pain and stiffness, with the clinical picture of a limp, in a patient presenting deformity characterized by flexion, adduction, and eversion of the leg. The radiologic examination will conclude the evidence. The condition may be unilateral or bilateral and varies in the degree of involvement.

The presence of Heberden's nodes on the terminal finger joints and symptoms and signs in other joints will indicate the systemic process present in these cases.

There is little difficulty in differentiation from involvement of the hips in Marie-Strümpell spondylitis, in which clinical examination will disclose a rigid poker spine with limited chest expansion. X-rays will show the typical sacroiliac sclerosis.

Conservative Treatment.—Conservative measures will be devoted to the relief of pain. The general health should be improved by conservative weight reduction and limitation of activity within the capacity of the joint as indicated by the presence of pain. During an acute phase, relief from weight-bearing by bed rest associated with massage, diathermy, and gentle repetitive hip exercises is beneficial. When the patient is ambulatory, a raised heel of the shoe or the support of crutches or a cane will give relief. Analgesics can be employed as required.

Operative Treatment.—In cases where pain becomes a burden and especially in the younger patient who is prevented from doing his work by the hip disease, operative intervention should be carried out.

In the unilateral case where the patient's health will permit immobilization in plaster casts for 3-4 months, a combined intra-articular and extra-articular arthrodesis will secure a painless hip that will give good service. The hip is best fused in 20-30-degree flexion, 5-degree abduction, and slight external rotation.

However, arthrodesis must not be considered in the case presenting marked pain in the low back with x-ray changes of advanced lumbar arthritis. In these cases and in

those presenting a bilateral lesion, an arthroplasty to retain a partial range of movement is indicated. The various methods of securing this need not be detailed here. Suffice it to say that at the present time, the replacement prosthesis for the femoral head made of stainless steel or Vitallium is replacing the mold arthroplasty, which has been in vogue for the past 14 or 15 years. This method appears to give the best results to date in this difficult field of reconstructive surgery.

Other operative procedures, such as denervation of the hip or capsulectomy and its variants carried out for the relief of pain, all have their advocates. Similarly, the McMurray osteotomy to correct the flexion-adduction deformity has been successfully used in certain cases. There is a multiplicity of procedures, all selected by different workers for individual cases, which time will finally evaluate.

OTHER DISORDERS OF THE HIP

Space will not permit the discussion of the many other clinical lesions that involve this joint. The student must remember the *congenital subluxations and dislocations* presenting at birth and in the early years of life. All suspected cases must be subjected to careful and repeated clinical and radiologic examinations.

From the ages of 5-10 years, the possibility of *Perthes' disease (osteochondrosis of the upper epiphysis)* will be suggested in the youngster with aching pain and a limp. At a later age, around puberty, a *slipped epiphysis* requiring detailed lateral views of the hip may be encountered.

At all ages the possibility of *tuberculous or pyogenic arthritis* must be considered, but again these disorders are most frequently found in the younger age groups.

Atrophic arthritis commonly involves the hips as part of the polyarthritis of rheumatoid arthritis or ankylosing spondylitis. This type generally occurs in the female during the 3rd, 4th, and 5th decades.

Inflammation may involve the *psaos bursa*, giving rise to a swelling which presents in the groin and requires differentiation from *hernia*, *saphenous varicosity*, and *psaos abscess*. The *ischial bursa* may enlarge and give a fluctuant

swelling under the lower border of *gluteus maximus*.

Finally, the *trochanteric bursa* may be chronically involved by *tuberculosis* or by secondary spread from amorphous calcium deposits in the *gluteus medius tendon*. This latter gives rise to an acutely painful swelling over the trochanter. X-ray examination will usually show the calcium. The course is similar to that found in the subacromial bursitis so common in the shoulder.



Fig. 662—X-ray of Legg-Perthes' disease. Note the fragmented appearance of the head and widening of the metaphyseal region. The revascularization and creeping substitution of the head require about 3 years.

SHAFT OF THE FEMUR

The shaft of the femur is the longest and strongest bony structure in the body. It has normally an anterior and lateral bowing. Fractures result from great violence and are difficult to reduce. Following reduction they are difficult to maintain in position because of

the powerful muscle forces acting upon them. There is a tendency for fixation of the quadriceps to the healing area with consequent limitation of function. This is one of the factors causing the stiff knee, which is the chief disability after such injuries.

Mechanism.—Direct violence of great severity is the usual cause. Indirect violence giving a torsion force and resulting spiral fracture accounts for a smaller number.

The displacements taken up initially depend on the direction of the causative forces, but the powerful muscles gradually pull the fragments into positions of characteristic deformity.

In *supracondylar fractures*, the lower fragment is angulated backward by the *gastrocnemius*. Reduction must therefore be carried out with these muscles relaxed by flexion of the knee and a counterforce applied to maintain the corrected position.

Clinical Picture.—The patient is in great pain and may be in shock, with associated injuries to other structures. Deformity is sometimes marked, and the fracture area is surrounded by a large hematoma. Local tenderness and crepitus will be found. The patient is moved with great difficulty. X-rays reveal the type of fracture and displacements.

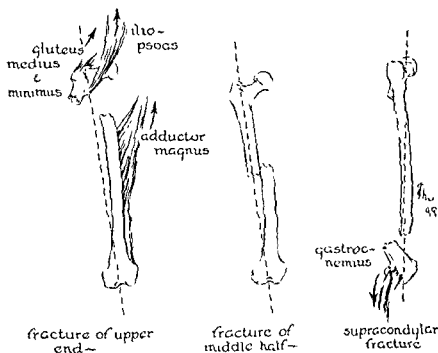


Fig. 663.—Influence of muscle pull on fractures of the femur.

Fractures of the upper end present flexion, abduction, and external rotation of the upper fragment and adduction of the shaft produced by the adductor magnus. Because of this, reduction and fixation are best arranged in flexion and wide abduction of the whole limb.

Fractures of the shaft proper in its middle half present adduction of the lower in relation to the upper fragment, with overriding caused by the spasm of the hamstrings and quadriceps. External bowing is produced frequently in these cases. Traction must therefore be maintained in moderate abduction.

Treatment.

Emergency Treatment.—The limb should be splinted before moving the patient. A Thomas splint with fixed traction or a plaster spica is the ideal method and is used if the patient is to be transported long distances. If such facilities are not available, fixation of the two limbs together and both fixed to a rigid support can be used. Morphine should be given to alleviate pain.

Definitive Treatment.—Various methods of treatment may be considered for shaft fractures occurring in adults, and the choice will

depend on the judgment of the surgeon in charge:

1. Traction and countertraction
2. Manipulation and plaster spica
3. Open operation and internal fixation
4. External-skeletal fixation

Principles Underlying Therapy.—Reduction in *adults* should be anatomic if possible in cases of femoral fractures, but this is sometimes impossible to achieve. Angulation must be avoided at all costs and the proper length of the limb maintained. Furthermore, alignment of the bones should be such as to avoid abnormal stresses and strains on the adjacent joints above and below. In *children*, because of the overgrowth in length of the shaft due

to the hyperemia of repair, an average of 1 cm. overlap is the ideal. In *children under 4 years*, Bryant's suspension or a plaster spica is the usual method, whereas skin traction is the routine after this age.

If reduction in *adults* can be secured by conservative measures such as skin or skeletal traction, these methods should be employed. Sometimes interposed tissue or injuries to other bones or the patient's general state may demand the use of open operative reduction and internal fixation to facilitate aftercare.

If traction is employed in *adults*, skeletal is preferable to skin traction. The Kirschner wire or Steinmann pin may be placed through the lower end of femur or upper end of tibia.

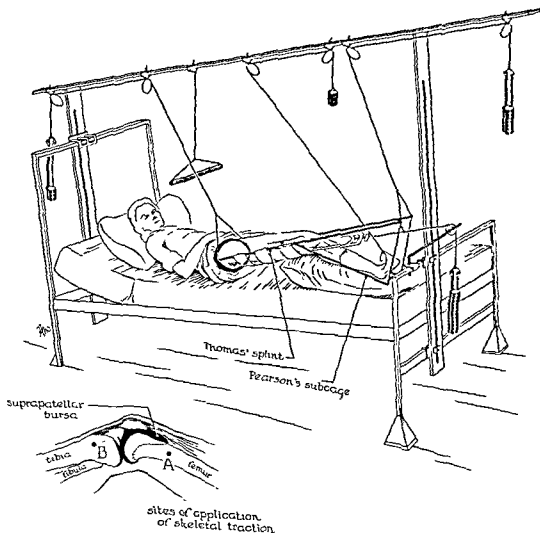


Fig. 664—Balanced traction for fractured femur. Inset shows site for insertion of Kirschner wire or Steinmann pin. A in femur; B in tibia

Either site has its advantages and disadvantages.

The pin should not be placed through the lower end of the femur in the vicinity of the fracture hematoma which may thus become infected.

The pin should be driven from the inner to the outer side to avoid injury to the femoral vessels in Hunter's canal and should not penetrate the suprapatellar pouch since spreading infection may cause inflammation and obliteration of this pouch, with resulting stiffness of the knee.

It is technically easier to put the pin through the upper end of the tibia at the level of the tibial tubercle. When skeletal traction is applied at this site, however, the force is transmitted through the knee joint of which the ligaments may be stretched. This danger is avoided if the extension is not maintained too long or with too great force.

When the patient is under anesthesia the pin is introduced and traction is set up. The

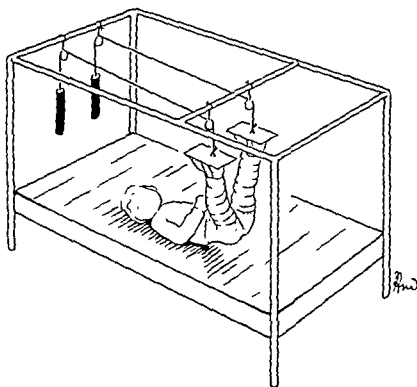
fracture is manipulated to the correct length of limb and correct position, and traction and countertraction are instituted. Approximately one-seventh the body weight is adequate for purposes of extension, but x-ray follow-up gives the best indication of the amount to use in the individual case.

It is wise to avoid pin traction from one site of longer duration than 4-6 weeks.

When x-rays show satisfactory position and good callus formation (8-12 weeks), traction is replaced by a plaster spica or Thomas caliper. The Thomas caliper may be used subsequently as a support when the patient becomes ambulatory on crutches.

When it is decided that open operation is indicated, the anterolateral approach is employed and a 6- to 8-screw plate is applied, with the screws transfixing both cortices.

A Thomas caliper may be used as a splint in the postoperative period preferably to a plaster spica. Ambulation should not be permitted until 6-8 weeks have passed, and pro-



utilized for children under 3 years of age. The weight of the traction and the weight of the patient, on the average, must be clear of the bed.

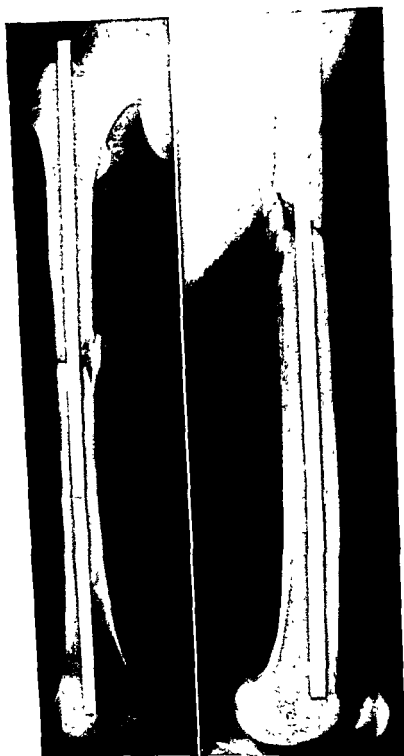


Fig 666—Fracture of the femoral shaft, treated by intramedullary nail

tective splinting is necessary for approximately 4-6 months

The subtrochanteric fracture presents a special problem and is probably best treated by open operation and internal fixation with a Smith-Petersen nail and long side plate. Intramedullary nailing may be considered for this type of case.

Recently, for fractures of the upper two thirds of the shaft, intramedullary fixation by Kuntscher nail has been employed. The site of fracture is exposed by the anterolateral or posterolateral approach, the guide wire inserted retrograde through the distal end of the proximal fragment and through the trochanteric area followed by the insertion of the nail over the guide wire. The nail is driven down through the fracture line into the distal fragment under direct vision. This method enables immediate movements of the knee and ambulation on crutches in 10-14 days. It has become the treatment of choice in selected cases when expert care is available.

Some cases in the young show spiral fractures with little displacement. These can be treated by plaster spica fixation.

External skeletal fixation may also find a place in selected cases.

It is most important in fractures of the femur to maintain the activity of the quadriceps muscle and at the earliest moment obtain the return of knee function.

Transverse fractures of the femur usually unite more slowly than the spiral fractures. In the adult, the time loss from work averages 8-12 months.

Complication.—The chief complication is stiffness of the knee which should be prevented by avoiding prolonged or excessive traction through the knee or placement of the pin or wire too near the suprapatellar pouch. This stiffness may be minimized by maintenance of activity of all muscles of the limb during therapy. Intramedullary nail fixation is the method which best preserves muscular power and knee motion.

When seen as a late complication, a trial of quadriceps drill and active movements of the knee is recommended before manipulation under anesthesia or quadriceps lengthening is considered.

THE KNEE

The knee joint marks the articulation of the two longest and strongest bones in the human body; namely, the femur and the tibia. A third bone, the patella, which is the largest sesamoid, forms the third osseous component of this joint. Even though the fibula is frequently involved in injuries to the knee, it is not part of the joint.

The femoral condyles articulate in the shallow fossae on the superior surface of the tibia. These bony fossae are deepened by the superimposed medial and lateral semilunar cartilages.

The strength and stability of this joint do not depend on its bony contours but rather on the strong ligaments that bind the bones together and the powerful muscles that control its movement in different positions of flexion and extension.

Because of the tremendous leverage exerted on this joint by forced movements, injuries to the ligamentous structures and intra-articular menisci are very common. Sports such as football, ice hockey, and skiing, hazardous occupations, and modern rapid transportation all contribute to the increasing number of knee disorders requiring treatment. Injuries of the soft tissues predominate.

Musculature

Attention should be directed first to the quadriceps muscle, so named from its four components, the vastus intermedius, vastus lateralis, vastus medialis, and rectus femoris. This important group of muscles has as its antagonists the hamstring group of muscles, consisting of the biceps femoris, the semimembranosus, and the semitendinosus. These two groups of muscles, the extensors (quadriceps) and the flexors (hamstrings), are reciprocally innervated and act with other muscles, such as the gastrocnemius, popliteus, gracilis, and sartorius, to give the synchronized movements characteristic of normal knee joint function.

Interest is usually centered on the quadriceps. It is therefore necessary to understand evolutionary changes that have taken place in these muscles. In the anthropoid, the knee is somewhat flexed when the animal assumes

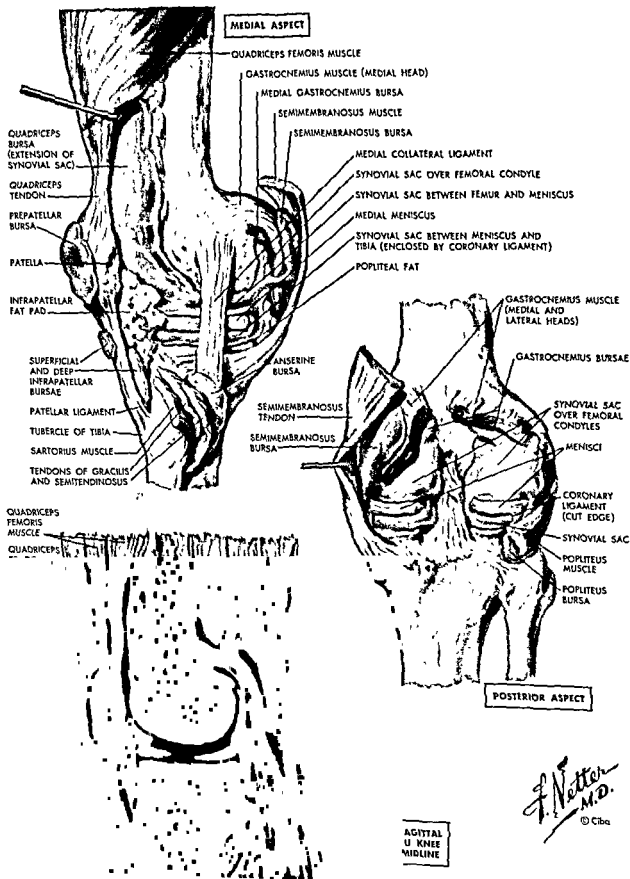


Plate 75.—Anatomy of the Knee Joint.

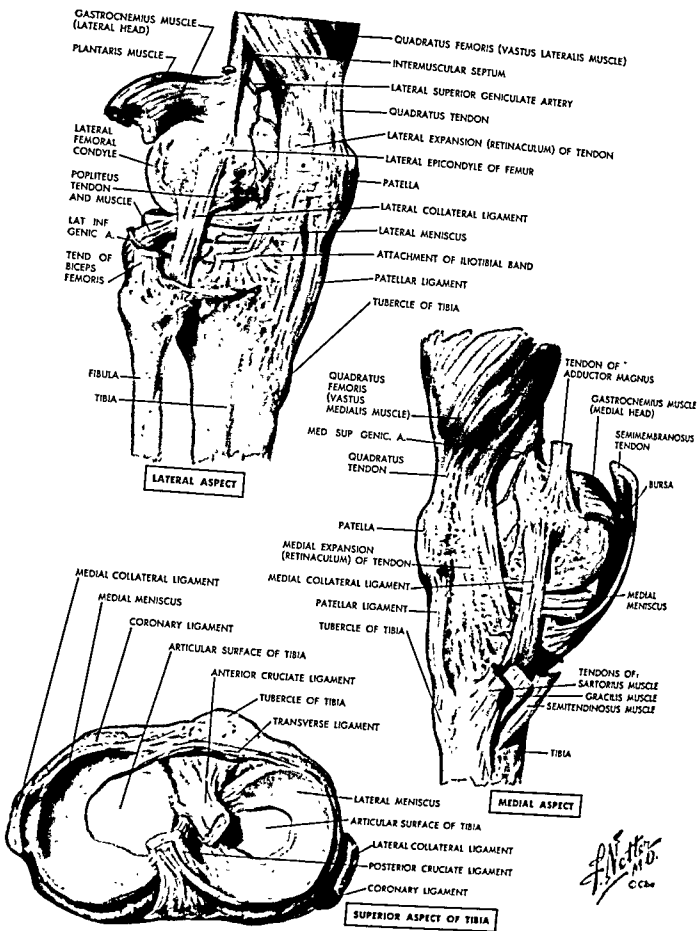


Plate 76.—Anatomy of the Knee Joint.

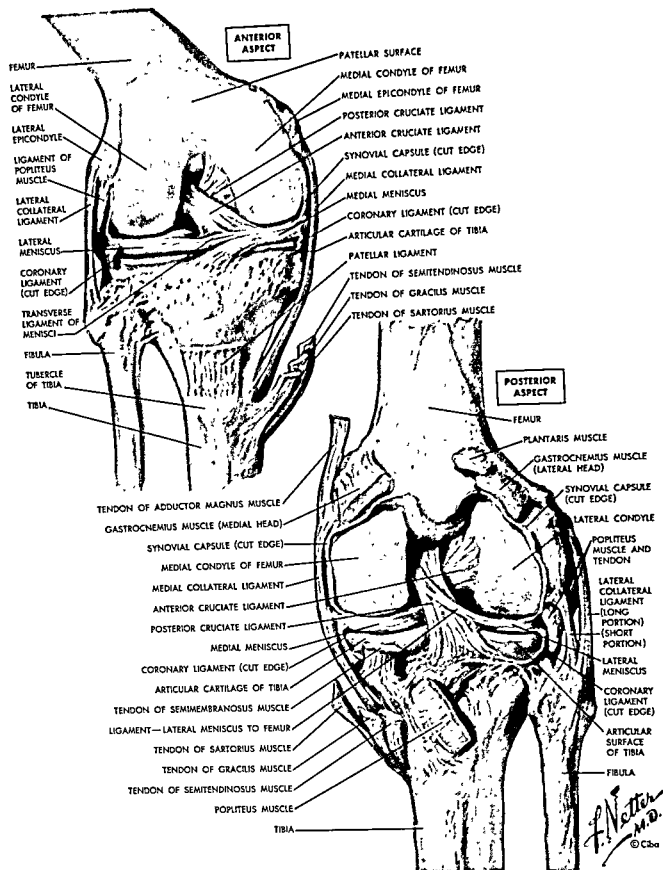


Plate 77.—Anatomy of the Knee Joint.

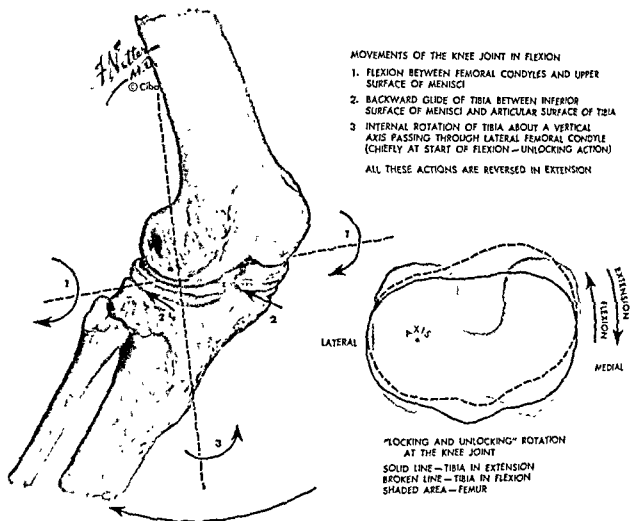


Plate 78.—Movements of the Knee Joint.

Courtesy Moseley, H. F. CIBA
CLINICAL SYMPOSIA 5: 171, 1953

the upright posture. However, man has evolved further and, when erect, the knee is maintained in complete extension. This is possible because of further functional development of the quadriceps. Although not completely true, it is helpful to regard the vastus medialis as the muscle that has developed the function of attaining and maintaining the final 15 degrees of extension. Here is one reason why disorders of the knee cause the joint to assume the position of slight flexion, since a study of evolutionary processes discloses that man loses first those functions last evolved. This also serves to explain the characteristic atrophy of the vastus medialis in chronic disorders of the knee.

Synovial Cavity

The *synovial cavity* of the knee is the most extensive found in any articulation. It consists of two main parts:

1. The inferior cavity arranged in relation to the articular surfaces of the femur, tibia, and patella.

2. The superior cavity, or suprapatellar pouch, forming an upward extension of the joint between the quadriceps tendon and the femur above the articular surface.

The synovial membrane covers the intra-articular fat pads and cruciate ligaments. When distended with effusion, the superior reflection of the suprapatellar pouch will extend about 2-3 inches above the superior pole of the patella. In this area, the membrane, when thickened by chronic disease such as tuberculous synovitis, can be palpated through the atrophied muscles.

Position of Rest

When a tense effusion distends the synovial cavity, the knee is held in a position of 15° to 20-degree flexion. Before chronic stretching of the tissues has occurred, this is the position in which the joint cavity has its maximal capacity to hold fluid. This fact can be confirmed by insertion of a wide-bore needle with manometric connection. When in this position of rest, either extension, flexion, or long-axis traction applied to the joint will increase further the intra-articular tension.

In this posture of 15° to 20-degree flexion, tension in the vastus medialis (which is largely responsible for the last 15 degrees of extension) is reciprocally inhibited, and it rapidly atrophies. *Atrophy of the vastus medialis is therefore a cardinal clinical sign in organic disorders of the knee.*

Ligaments

The important ligaments stabilizing the bones are the medial collateral and cruciate ligaments which require careful description at this point, since they are the ones most frequently deranged by trauma.

Medial Collateral Ligament.—This is the ligament that is most frequently injured. The mechanism is a severe strain caused by forced abduction or external rotation.

This ligament consists of two parts: (1) a short, deep, thick posterior portion and (2) a longer anterior band, approximately 7.5-9 cm in length, extending from the femoral epicondyle and fanning out into a broad expansion on the anteromedial surface of the tibia. This insertion is strengthened by the common tendinous expansion of the sartorius, gracilis, and semitendinosus.

Cruciate Ligaments.—These ligaments are the fibrous bands which stabilize the tibia and femur in their anteroposterior glide upon one another. The *anterior cruciate ligament* is tight on extension and limits excessive anterior mobility of the tibia on the femur. When ruptured, there is increased anterior mobility of the tibia on the femur when tested in flexion.

The *posterior cruciate ligament* limits posterior mobility and is tight on flexion. Rupture of this ligament permits the tibia to assume a position of posterior displacement on the femur when in flexion. When tested clinically by the "drawer sign," there is an increased posterior mobility.

An aid in remembering the anatomy and function of the cruciate ligaments is the mnemonic—ATE A PIE.

Anterior cruciate is
Tight on
Extension and runs from the
Anterior surface of the tibia to the
Posterior part of
Internal surface of the
External condyle of the femur

The posterior cruciate is tight on flexion and runs in the opposite direction

Semilunar Cartilages

These intra-articular menisci develop as a condensation of the mesoderm, dividing the joint into superior and inferior cavities. In time a central division occurs from the development of the ligamentum mucosum, giving rise to a lateral and medial meniscus of solid discoid form. These develop into the semilunar-shaped structures with which we are all acquainted.

The evolution of the medial cartilage tends to be more uniform than that of the lateral cartilage. In the latter, arrests in development account for the various forms of congenital discoid cartilages, which are found almost exclusively on the lateral side.

The menisci differ somewhat in shape. The lateral cartilage constitutes a greater portion of a circle of smaller radius than is the case for the medial meniscus. This corresponds to the differences in the shapes of the femoral condyles, which will be discussed later in relation to the motions of the joint.

The medial meniscus is firmly attached in its posterior portion to the deep surface of the medial collateral ligament, whereas the lateral meniscus is not so closely adherent to the lateral collateral ligament and is therefore more mobile than its counterpart. The tendon of the popliteus muscle bears a close relation to the lateral meniscus. Both cartilages are peripherally attached by the coronary ligaments to the adjacent areas of the upper aspect of the tibia.

Movements of the Knee Joint

This articulation is classed as a hinge joint. However, its movements are more complicated than the flexion and extension of a simple hinge. To help in understanding the movements of the knee, compare them with those of the elbow.

Motion at the elbow consists of two main mechanisms; namely, the *hinge mechanism* between the lower end of the humerus and the greater sigmoid notch of the ulna and

radial head and the *pivotal mechanism* between the radial head and the capitellar surface of the humerus and lesser sigmoid notch of the ulna.

These two mechanisms exist in a variant way in the knee joint (Plate 78). Flexion from the position of complete extension occurs as a hinge movement between the femoral condyles and the upper surfaces of the menisci and tibial articular cartilage. At the same time that this hinge movement is occurring, the tibia glides backward, this latter motion taking place between the inferior surface of the menisci and the articular surface of the tibia. Therefore, flexion of the knee is a movement that occurs on a transverse axis of changing position, and at any particular point only a small area of femoral articular cartilage is in contact with a small area of the articular surfaces inferiorly.

At the same time, as flexion occurs, there is also an internal rotation of the tibia through a vertical axis situated in the outer part of the lateral femoral condyle and tibial tuberosity. This internal rotatory movement is reversed in extension. Maximal stability is obtained by this external rotation, which locks the joint on complete extension.

Association of ideas assists in memory. Therefore, one might remember that just as complete elevation of the arm above the head is possible only with external rotation of the humerus, so complete extension of the knee is possible only with external rotation of the tibia on the femur.

The above exposition will explain the reasons for the differences in size and shape of the lateral and medial femoral condyles and menisci. Since the axis of the rotating movement of the tibia on flexion and extension is vertically placed through the outer part of the joint, the medial condyle and menisci must move farther and therefore possess the larger radius of movement.

Besides this movement of rotation, the student should recognize that in the flexed position, a certain amount of rotational and side-to-side motion is possible on clinical examination. This varies in extent when comparing the close knit with the lax-knit type of joint.

Examination of the Knee

This articulation, unlike the hip and shoulder, is superficially placed, and a great deal can be learned on clinical examination by inspection and palpation, as shown in Plates 79 and 80.

Inspection.—The patient may walk with an obvious limp, carrying a cane or crutches. When assisted by a friend on either side, the method of hopping on the sound leg should be noted. Complete absence of weight-bearing and partial flexion of the knee will indicate the acute injury so often seen in the young athlete.

With the patient supine on the examining table and the affected limb next to the examiner, the region of the knee is inspected for obvious deformity. Gross changes due to severe injury will be apparent. The most frequent finding is that the knee is held in the flexed position previously mentioned. Also, the joint is swollen from synovial effusion, the normal hollows superior to and on each side of the patella being lost due to distention.

In injuries by direct violence, contusion of the skin over the patella or in the region of the tibia will be seen. When the injury has occurred several days previously, ecchymoses of varying hues will be present. In chronic disease, as well as in acute derangements of 10 or more days' duration, wasting of the vastus medialis will be perceived by the experienced examiner. Note will be given to the prominence of the superior reflection of the synovial membrane. At this point, the patient may be asked whether he can raise the limb from the couch and whether flexion and extension are possible voluntarily.

Palpation.—The student should first handle the limb to determine the range of passive motion. He should ascertain whether complete extension and flexion are possible, or whether they are limited by pain and muscle spasm in the acute cases, or by contracture in chronic disorders.

During the manipulation, the typical elastic block to complete extension in cases of displaced menisci with the locked knee will be discerned. With the palm of one hand compressing the patella to the femoral articular

area, crepitus will be noted on passive flexion and extension when degenerative changes are present. Fixation of the patella can be demonstrated by attempting side-to-side manipulations of the bone in the chronically stiffened joint.

The *abduction test* is valuable in differentiating lesions of the internal collateral ligament from those of the meniscus. With the knee in complete extension and with the examiner's knee as a fulcrum, a valgus strain is applied to the joint. This will greatly augment pain in the ligamentous injuries, and digital palpation will localize the point of maximal tenderness as being near the femoral epicondyle rather than on the joint line over the medial meniscus. This can be confirmed by forcible external rotation in flexion, when ligamentous injury will present digital tenderness over the femoral origin of the ligament, while joint line tenderness will be maximum with lesions of the meniscus.

McMurray's sign is valuable in posterior tears of the meniscus. With a finger flattened along the joint line, the knee is fully flexed. Rotation of the tibia internally and externally while extending the joint will transmit a clicking sensation to the finger when the torn meniscus moves in and out of place.

The *drawer sign* is diagnostic of associated injuries of the cruciate ligaments. With the knee flexed to a right angle, the tibia is pulled forward and pushed backward. Increased anterior mobility denotes rupture of the anterior cruciate ligament. Increased posterior mobility indicates rupture of the posterior cruciate ligament, while increased anterior and posterior mobility is present when both cruciates have been avulsed. This increased anteroposterior glide present in cruciate ligament avulsions constitutes the *drawer sign*.

Examination for Effusion.—This determination is based on the law of the equal transmission of pressure in all directions when applied to fluid within an enclosed space. Plate 80 shows how one hand compresses the fluid in the suprapatellar pouch, while alternating pressure on the patella causes that bone to tap against the femur. The second method, useful when only a moderate effusion is present, is to apply a finger on each side of

the patella and perceive the transmission of pressure from one side to the other

When a tense effusion is present, examination of the knee may be impossible until the joint has been aspirated. Aspiration is done under local anesthesia from the lateral aspect. It should be noted whether the fluid withdrawn is serous, sanguineous, or pure blood. The presence of blood indicates a severe injury involving the cruciate ligaments or an intra-articular fracture. The settling out of a fatty layer on the top of blood is pathognomonic of an intra-articular fracture, usually of the upper end of the tibia. After aspiration, it is possible to make an adequate examination of the joint.

Palpation of the Suprapatellar Pouch.—Careful examination of this area gives valuable information. Palpation may reveal the presence of a joint mouse most frequently lodged in this recess of the synovial cavity.

Thickening of the membrane in chronic synovitis is best ascertained by rolling the superomedial reflection of this pouch under the fingers placed on the atrophic vastus medialis.

Mensuration.—Careful measurements are valuable additions to the patient's record and are especially easy to obtain in injuries of the knee.

The range of motion is accurately indicated by the goniometer, using the position of full extension as 180 degrees.

Measurement of the circumference of the thigh and calf at fixed points from the superior edge of the patella on each limb will give an exact record of the extent of muscle atrophy.

Radiologic Examination.—X-rays taken in the standard anteroposterior, oblique, and lateral positions are necessary. Tangential views show the opposing articular surfaces of the patella and intercondylar area of the femur. In cases of severe injury with ligamentous avulsion and fracture, examination in the positions of strain after instillation of procaine into the joint will indicate the degree of joint laxity and the extent of displacement before recoil. Arthrography, using contrast media, has proved valuable in cases of doubtful meniscus lesions.

Classification

1. Injuries of the extensor apparatus
 - Hematoma of thigh
 - Rupture of quadriceps expansion
 - Patellar lesions
 - Fractures, stellate or transverse
 - Chondromalacia
 - Dislocations
 - Rupture of patellar ligament
 - Osgood-Schlatter's disease
2. Injuries of other soft tissues
 - Synovitis
 - Hemarthrosis
 - Internal collateral ligament
 - Chronic postural strain and Stieda-Pellegrini's syndrome
 - Cruciate ligaments
 - Semilunar cartilages
 - Bursal lesions
3. Fractures of component bones
 - a Femur
 - Lower femoral epiphysis
 - Condylar and intercondylar
 - Articular; osteochondritis
 - b Tibia
 - Bumper fracture
 - Tibial spine
4. Dislocations
 - Knee
 - Patella, see above
5. Arthritides
6. Congenital anomalies

INJURIES OF THE EXTENSOR APPARATUS

Hematoma of the Thigh

Large volumes of extravasated blood may accumulate in the quadriceps mass as a result of a direct blow or kick at football, 500-1,000 ml is not unusual.

Diagnosis.—The history of the injury together with a swollen tender thigh is typical. Fluctuation is present in the early cases.

Treatment.—If seen early, treatment includes applications of a compression bandage, ice bags, and the institution of quadriceps exercises consisting of repeated straight-leg raising, tensing the quadriceps by forcing extension, and contracting and relaxing the muscle groups by alternate gentle flexion and extension. These exercises constitute quadriceps



Plate 79.—Examination of the Knee Joint.

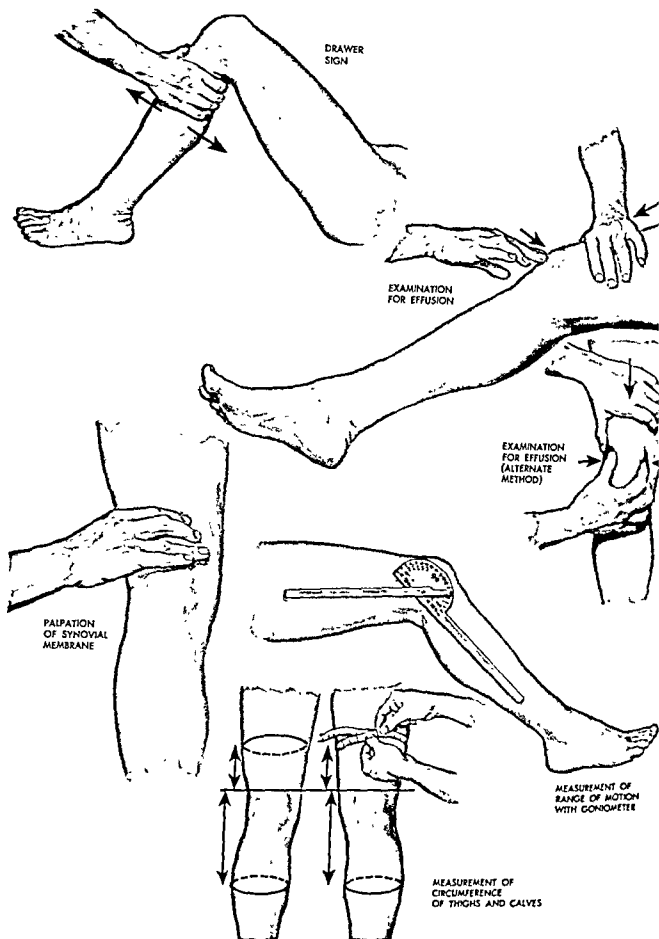


Plate 80.—Examination of the Knee Joint.

drill and should be carried out for 5 minutes on the hour every hour. Early ambulation on crutches is desirable. If seen within 2-3 days aspiration with a wide-bore needle or evacuation through a small incision is indicated. A compression bandage is applied for 12-24 hours followed by quadriceps drill. Bed rest for 2-3 days and then ambulation on crutches and graduation to normal activity is the post-operative routine.

If seen later, the hematoma may be clotted, and it is best to apply hot packs and initiate quadriceps drill. The hematoma begins to liquefy between 10-14 days, and the serum may then be evacuated. If the femoral peri-

osteum has been raised, myositis ossificans sometimes develops. The use of a Thomas caliper to prevent knee movements is then beneficial, and the progress must be followed by x-rays at regular intervals. *Passive movements, which predispose to myositis ossificans, are avoided at all stages.*

Rupture of the Quadriceps Expansion

This corresponds to rupture of the rotator cuff in the shoulder. The tendon ruptures at the critical zone near its insertion on the patella. Degenerative changes constitute the predisposing factor. The central portion, consisting of the tendons of the rectus femoris and vastus intermedius (which corresponds to the supraspinatus tendon) may separate alone, or the expansions of the vasti on the medial and lateral aspects of the patella may separate as well. This major injury corresponds to massive avulsion of the rotator cuff. The cause of such tendon ruptures is a sudden strain in a middle-aged, often overweight patient while endeavoring to prevent a fall. The condition may be bilateral.

Diagnosis.—The diagnosis is often overlooked but can be made readily, if suspected, from the history of a sudden onset due to strain and the inability of the patient to raise the extended leg from the table. The absence of active extension while power of flexion is preserved in a patient with a hemarthrosis and a deep visible and palpable sulcus above the patella is pathognomonic. If seen later, extensive ecchymosis, indicating a severe injury, will be present.

Although radiologic examination may reveal no abnormality, sometimes the lateral view will show an abnormal tilt of the superior pole of the patella away from the intercondylar articular surface. Soft tissue films may disclose the tendinous defect with a distended joint. Late cases present considerable ossification extending from the patella into the area of tendon rupture.

Treatment.—The major avulsions are best treated at the earliest moment by open operation. The hemarthrosis is evacuated and the tendon reattached, sutures passing through drill holes in the patella. When massive ruptures have been neglected, late repair with



Fig. 667—Myositis ossificans following hematoma of thigh.

fascia may be indicated if a period of active quadriceps training has failed to restore adequate power to the extensor mechanism.

Patellar Lesions

The patella is the largest sesamoid bone in the body and constitutes a fulcrum over which the quadriceps expansion passes to the patellar ligament and tibial tubercle. It presents a bony barrier to injuries directed from the anterior aspect and determines the normal cosmetic appearance of this region.

The patella is an integral part of the extensor mechanism. It can be removed and the extensor apparatus reconstituted. However, accurate evaluation of knee joint function after its ablation, taking into consideration power, range, speed, and coordination of motion, will disclose that the articulation has lost an important though not essential part of its mechanism.

Fractures

Mechanism.—Fractures of this bone may result from a direct blow or fall whereupon it is crushed against the intercondylar area of the femur. Such forces result in a stellate fracture but it must be stressed that damage to the articular cartilage of the patella and intercondylar surface of the femur may be the most important component of the injury.

Sudden contraction of the quadriceps with a flexed knee may break the patella transversely with minimal or maximal separation of the fragments. The extent of bony separation indicates the degree of rupture of the lateral expansions of the quadriceps. The shape of the patella appears to predispose to knee injury, and some patients are subject to bilateral and even recurrent fractures.

Clinical Picture.—The region of the knee is grossly swollen, and evidence of bruising may be present. The injury may be associated with a wound compounding the fracture or communicating with the prepatellar bursa. Hemarthrosis is usually present. In transverse fractures with separation a groove is palpable between the fragments which increases when the patient flexes the knee or attempts quadriceps contraction.

The patient usually is unable to lift the extended limb.

Diagnosis.—The diagnosis is readily made on the clinical picture. X-ray examination in different planes affords conclusive proof and indicates the degree of comminution and separation.

Treatment.—The perfect result to be obtained from the treatment of patellar fractures is determined by three factors:

1. Restoration of the continuity of the extensor apparatus
2. Preservation of the fulcrum of the patella which gives the power in the final 5 to 10 degrees of extension
3. Re-formation of a smooth gliding articular surface between patella and femur

In stellate or transverse fractures without displacement, this result may be obtained by the use of hot packs, compression bandages, and bed rest for 2-3 days, followed by quadriceps drill and early ambulation in a tubular cast with the knee in a position of complete extension. The cast should be maintained for 4-6 weeks, and this is followed by re-education of knee movements.

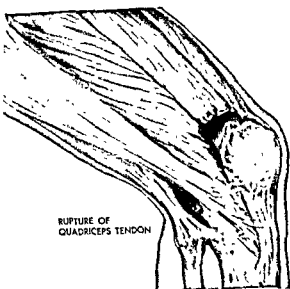
Transverse fractures with separation are best treated by open operation. The hemarthrosis is evacuated and the patella and lateral expansions meticulously sutured. The patella must be so approximated as to give a perfect articular surface. Sometimes a small fragment at the upper or lower pole is best excised.

When the patella is comminuted or when a smooth articular surface cannot be obtained, excision of the patella is indicated. The extensor tendons must be plicated and the continuity with the patellar tendon securely restored.

Postoperative therapy consists of a plaster cast with the knee completely extended for 4-6 weeks with quadriceps drill enforced. After splintage is discarded, re-education of knee movements is initiated.

Chondromalacia of the Patella

Chondromalacia of the patella is a clinical entity receiving more and more attention as a cause of internal derangement of the knee. As a result of bruising of its articular surface by a fall or blow on the kneecap, the cartilage



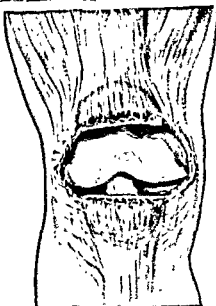
RUPTURE OF
QUADRICEPS TENDON



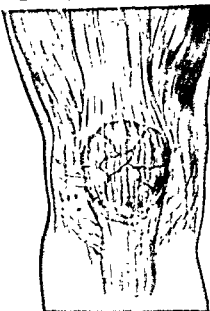
DEMONSTRATION OF
SUPRAPATELLAR DEPRESSION
IN QUADRICEPS—TENDON
RUPTURE



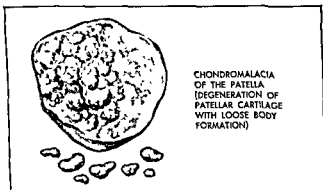
TRANSVERSE FRACTURE OF
PATELLA WITHOUT SEPARATION
(LATERAL EXPANSIONS INTACT)



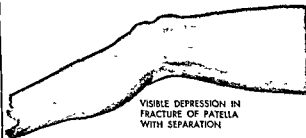
TRANSVERSE FRACTURE OF
PATELLA WITH SEPARATION
(LATERAL EXPANSIONS TORN)



COMMUNUTED FRACTURE
OF PATELLA



CHONDROMALACIA
OF THE PATELLA
(DEGENERATION OF
PATELLAR CARTILAGE
WITH LOOSE BODY
FORMATION)



VISIBLE DEPRESSION IN
FRACTURE OF PATELLA
WITH SEPARATION

F. Netter
M.D.
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Plate 81.—Patellar Lesions.

Courtesy Moseley, H. F.: CIBA
CLINICAL SYMPOSIA 5 171, 1953.

degenerates and sequestrates, giving rise to loose body formation. With separation of cartilaginous fragments, recurrent effusions develop and traumatic arthritis ensues.

Clinical Picture.—The patient may be an active youth with symptoms and signs of internal derangement. There may be complaint of pain centrally located in the knee deep to the patella. This is accentuated by movements of the knee with the patella compressed against the femur. Crepitus may be elicited in this maneuver.

An older patient may present with localized patellofemoral arthritis.

part bone to give a smooth surface. In some cases, however, the damage is so severe that excision of the patella must be carried out.

In all cases, a careful search for loose fragments of cartilage is made and their removal from the joint must be complete.

Dislocations

Dislocation of the patella is usually to the lateral side and is predisposed to occur in patients with valgus deformity or when the patella is small, highly placed, and the lateral femoral condylar buttress poorly developed.

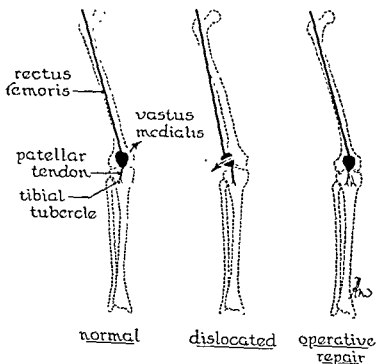


Fig 668—Mechanism of recurrent dislocation of patella

Diagnosis.—The study of x-ray films carefully taken to show the articular surface in various planes may disclose irregularities which, with the clinical picture, concludes the diagnosis. The presence of loose bodies in the joint is suggestive.

Treatment.—Exploration of the knee joint is indicated, and all components of the joint must be carefully studied through a medial parapatellar incision.

If the articular surface is not grossly diseased, the cartilage is shaved off down to com-

plete bone to give a smooth surface. In some cases, however, the damage is so severe that excision of the patella must be carried out.

Mechanism.—In most cases the resultant force of the quadriceps contraction tends to displace the patella to the lateral side. In the complete displacement the patella rotates 90 degrees or more around the lateral condyle.

Diagnosis.—When the patella is dislocated, the knee is held locked in a slightly flexed

position. The abnormal position of the patella is seen. Any genu valgum deformity is noted, and x-rays are taken to show the characteristics of the patella and femoral condyle.

Treatment.—Acute dislocation is readily reduced by direct pressure in a medial direction. The patient should then be placed on quadriceps drill to develop the musculature. This should also be initiated in recurrent cases and a firm bandage applied for a period if the patella is unstable.

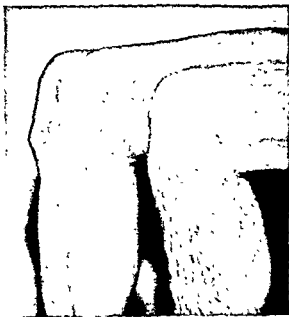


Fig. 669—Photograph of Osgood-Schlatter's disease

In most recurrent dislocations operation is eventually required. This consists of transposition of the patellar tendon with its tibial attachment into a slot cut more medially on the tibia. The capsule at the same time is plicated on the medial side. This places the line of quadriceps pull more directly in line with its tibial insertion.

Rupture of the Patellar Ligament

This can occur at the patellar or tibial attachment with or without a fragment of bone. The diagnosis is usually obvious. Treatment consists of careful suture, followed by splinting of the knee in extension until firm union has occurred (6-8 weeks).

Osgood-Schlatter's Disease

This is sprain of the patellar tendon attachment to the tibial tubercle. The tubercle develops its ossification as a separate center or as a tongue-shaped extension of the upper tibial epiphysis. This epiphysis fuses at 17-18 years. Before this period and especially from 12-15 years of age, athletic youths may develop pain and swelling of this area associated with limitation of knee movements and consequent limp.

Clinical examination shows a prominence of the tibial tubercle more marked than on



Fig. 670—X ray showing fragmented appearance of tibial tubercle during the period of creeping substitution. The unaffected side is shown for comparison.

the opposite side. The swelling may be red-
dened and tender. All grades of severity are
seen.

X-ray examination may be negative or show
a fragmentation of the tibial tubercle which
with time undergoes the creeping substitution
seen in the osteochondritic process.

Treatment.—This consists of rest to the
part. In mild cases strapping suffices. In severe
cases with separation of the epiphysis, reddening,
and swelling, a tubular cast with the knee
in extension is applied for 6-8 weeks.

If the severe cases are not so treated,
a permanent failure of fusion of the bony
tubercle may result. This is marked in later
years by recurrent pain on excessive use and
limitation of functional capacity. This results
from the sprain of the central attachment of
the quadriceps tendon, the lateral expansion
attachments to the tibia being undisturbed.

X-ray reveals the separated tubercle, and
treatment consists of roughening its deep aspect
and fusing this to the tibia. Excision of the
fragment with suture of the central tendon
to a raw bed on the tibia is an alternative
procedure.

INJURIES OF OTHER SOFT TISSUES

Synovitis

A serous effusion may occur in the knee
as an acute or recurrent condition. The acute
process may result from any irritation to the
synovial membrane resulting from a sprain
or from the presence of some mechanical
derangement in the joint. Loose bodies or
torn semilunar cartilages may be the irritant
factors, and an intra-articular lesion should
always be suspected in the recurrent cases.

Treatment.—Aspiration of the fluid, fol-
lowed by the application of a firm elastic
support and quadriceps drill, is the treatment
of choice. Recurrent effusions may require
exploration of the joint to ascertain the cause.

Hemarthrosis

The knee may fill with blood as a result
of a severe sprain, rupture of cruciate ligaments,
or fracture communicating with the joint. A
tense joint may develop rapidly within one
hour, whereas serous effusions take the greater
part of a day.

Clinically, the joint is held in the position
of maximum joint capacity, i.e., 160 degrees.
There are diffuse tenderness and considerable
pain on attempted movement. X-ray should be
taken to exclude bony injury.

Treatment.—Aspiration should be per-
formed. When the joint is lax, further ex-
amination may be carried out to ascertain
the extent of injury. A firm bandage and
quadriceps drill is the routine postoperative
therapy. Exploration may be advisable.

Internal Collateral Ligament

This ligament is subject to acute partial
and complete tears. Many cases of prolonged
disability of the knee are due to chronic pos-
tural strain of its femoral attachment. Calcified
deposits may occur at this point and give
rise to a syndrome named after Stueda and
Pellegrini.

Mechanism of Rupture.—The femoral is
much weaker than the broad tibial attachment
of this ligament and separates partially or
completely in varying degrees of valgus strain
of the joint. This constitutes the first stage,
which passes on to external condylar or tibial
fractures or lateral dislocations of the knee.

The causative force may be a tackle at foot-
ball from the side or a bumper injury from
a car.

Diagnosis.—This is made on the type of
injury and the location of the tender point
on the femoral condyle. In mild cases this may
constitute the clinical picture. In severe cases
the ligament is completely separated. Lateral
instability is present. A serous effusion or
hemarthrosis indicates the serious nature of
the injury. The knee is held flexed and the
patient bears weight with pain and a limp.

Treatment.—Mild cases require little more
than a firm support. Complete ruptures with
separation are best sutured by open operation
followed by plaster immobilization for 4-6
weeks and concomitant quadriceps drill.

The majority of cases present an inter-
mediate group. No instability is present, but
the final 20 degrees of extension and complete
flexion are painful. Treatment consists of
heat to the affected area, quadriceps drill, and
firm bandaging. Infiltration with 1% procaine
assists both the diagnosis and treatment.

Ambulation on crutches may be helpful for a week or more. Elevation of the inner side of the heel $\frac{3}{16}$ " and in-toeing will relieve the strain when weight-bearing begins.

Chronic Postural Strain and Stieda-Pellegrini's Syndrome

Both these clinical entities present the same clinical picture. They both occur in valgus strain of the knee of chronic type, whether in women of the sthenic type from the relative genu valgum or from the transmitted strain of pes valgus.

The patient complains of pain in the knee with inability to flex or extend the joint completely. There is the consequent limp, increasing with overuse. Tenderness is present over the femoral attachment and along the ligament.

X-rays may show degenerative arthritic changes. In the latter syndrome, an area of calcification will be seen at the femoral attachment.

Treatment.—This consists of diminishing the valgus strain, which may be accomplished by general measures such as the reduction of excessive weight. Raising the inner side of the heel and sole of shoes $\frac{3}{16}$ " and teaching the patient to in-toe will diminish the aggravation. With this is associated re-education of the muscular balance to both lower limbs working from the foot musculature upward. Local heat is an adjuvant, as are active movements of the knee on bicycle or sliding seat.

Cruciate Ligaments

The cruciate ligaments are ruptured in marked displacements of the tibia on the femur. They are therefore associated lesions in the various dislocations.

The *anterior cruciate* is tight in extension and limits hyperextension. It is injured in forcible hyperextension of the joint.

The *posterior cruciate* is tight in flexion and is injured in the dashboard type of injury when the flexed tibia is driven backward on the femur.

Clinical Picture.—The injury is usually a severe one with hemarthrosis present. Rupture of the cruciates results in an increased

anteroposterior glide of the tibia on the femur in the flexed position.

Rupture of the anterior alone permits increased forward movement; rupture of the posterior alone increases posterior movement.

Treatment.—This is determined by the concomitant injuries. Immobilization of the joint in a tubular plaster for 3 months associated with routine quadriceps drill is the most effective initial treatment. If associated with a dislocation of the knee, open operative suture of the ruptured soft tissue structures is indicated.

Late cases must be evaluated carefully and a thorough trial made of the stabilizing influence of quadriceps re-education before operative reconstruction is considered.

Semilunar Cartilages

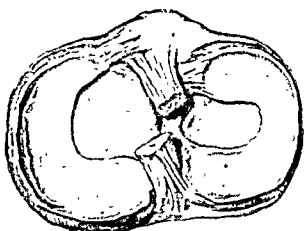
Injuries of the menisci constitute one of the most frequent and interesting groups of internal derangements.

Development.—The semilunar cartilages originate as a plate of mesoderm, which divides the joint into superior and inferior compartments. Later this plate is divided centrally by the ligamentum mucosum into a lateral and medial disc. The medial disc differentiates more rapidly and completely than the lateral disc into the semilunar shaped structures, which serve to deepen the sockets for the femoral condyles. Arrest in differentiation is more common in the lateral meniscus, which may present various forms to give the congenital discoid cartilage.

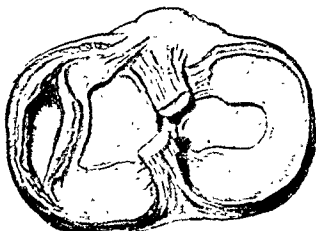
Mechanism of Injury.—The internal cartilage is injured 7-8 times more frequently than the lateral cartilage. This is because there is more tendency to an external rotation strain of the tibia on the femur in the flexed position of the knee. During this movement the internal cartilage is drawn between the articulating surfaces and, if ground between these surfaces with a shearing force, is fractured. Various types of meniscus injuries are produced as illustrated (see Plate 82).

The external cartilage is injured by an internal rotation strain with adduction of the tibia on the femur.

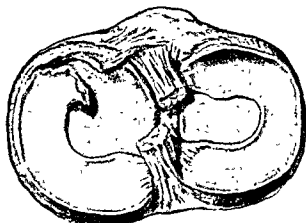
Clinical Picture.—The patient is typically a young athlete who suffered a severe rota-



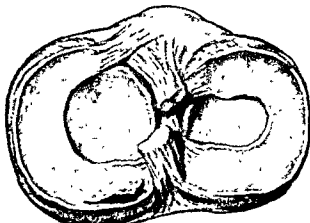
LONGITUDINAL TEAR



LONGITUDINAL TEAR WITH DISPLACEMENT
(BUCKET—HANDLE TEAR)



TEAR OF ANTERIOR POLE WITH FLAP



POSTERIOR TEAR

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Plate 82.—Lesions of the Semilunar Cartilages.

*Courtesy Moseley, H. F.: CIBA
CLINICAL SYMPOSIA 5: 171, 1953.*

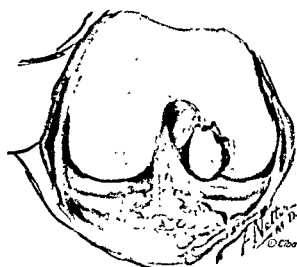


Plate 83.—Osteochondritis Dissecans.

*Courtesy Moseley, H. F.: CIBA
CLINIC II. SYMPOSIA 5: 171, 1933.*

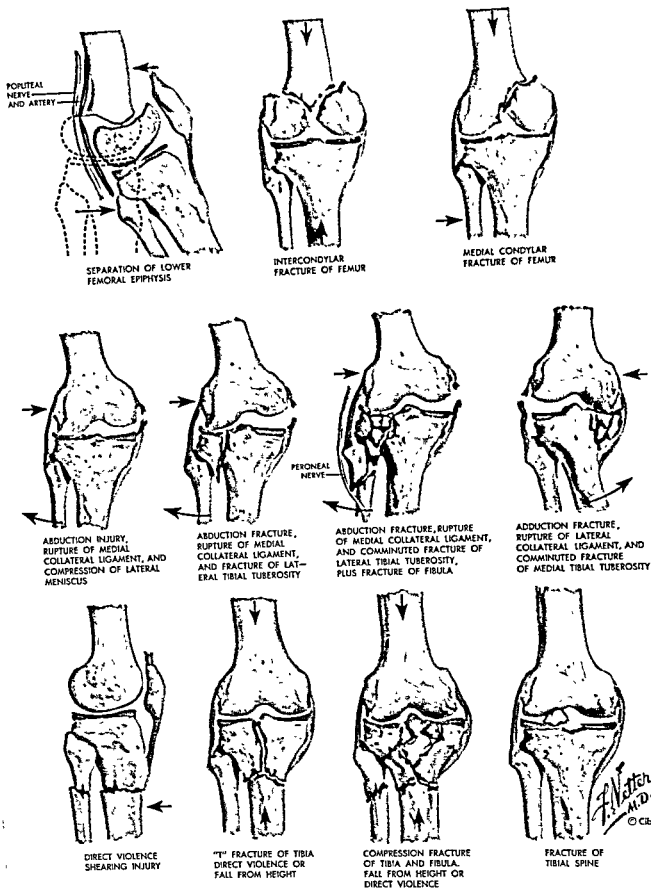


Plate 84.—Fractures of the Lower End of Femur and Upper End of Tibia.

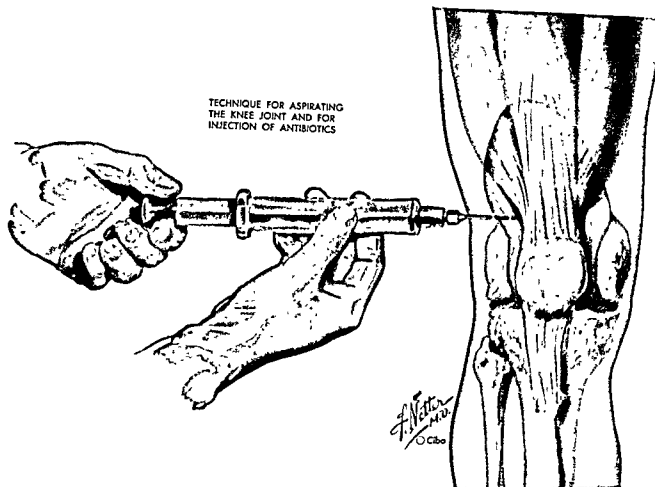


Plate 85.—Procedure for Aspiration or Injection of Knee Joint.

Courtesy Moseley, H. F. CIBA
CLINICAL SYMPOSIA 5 171, 1953

tion strain of the knee during a football game. He felt a sickening pain at the time and possibly heard or felt a click in his knee. The knee locked in a position of flexion and he was unable to carry on. When seen shortly afterward the patient is in pain. The knee is held by muscle spasm at 140-150 degrees, and attempts to walk on it or straighten it cause severe pain. Palpation over the inner joint line discloses a point of maximum tenderness.

Diagnosis.—This is made on the typical clinical picture with the locked knee and tenderness localized to the joint line.

Manipulation of the knee through a full range of movement under anesthesia unlocks the joint and adds confirmation to the diagnosis. X-rays in these cases disclose no bone injury but are taken routinely to exclude articular fractures or loose bodies in the joint.

The case may be seen later, when the displaced cartilage has been reduced. Here the diagnosis may be more difficult. Characteristic findings are atrophy of the vastus internus associated with effusions in the joint and joint line tenderness.

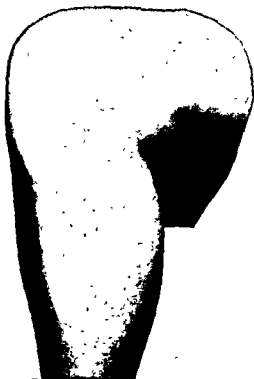
Manipulation of the knee in flexion and extension with varying degrees of rotation may elicit a click to the fingers placed along the joint line on the affected side. This test described by McMurray is of great diagnostic significance in doubtful cases. There may be a history of recurrent locking, recurrent effusion, or repeated giving way of the joint. In tears of the lateral meniscus, a sudden click in the final degrees of voluntary extension is most significant.

Arthrograms with contrast media (8-10 ml 35% Diodrast) may be helpful in cases of doubt.

Differential Diagnosis.—

1. *Quadriceps insufficiency* After all injuries of the knee, there is rapid atrophy of the quadriceps unless this is prevented by muscular drill. Insufficiency of the extensor mechanism can result in recurrent effusion, giving way of the knee, and all the points in the clinical picture of internal derangement. When suspected as the cause, the therapeutic trial of quadriceps drill is prescribed as a diagnostic and curative measure.

2. *Sprain of the internal collateral ligament* is the most important injury to differentiate in acute cases from injuries to the internal meniscus. The mechanism of injury here is a direct abduction or valgus strain, whereas the injury of the cartilage is caused by a rotation strain. Confusion arises where the two are associated.



A.



B.

Fig 671—Cyst of external cartilage. A, Clinical photograph B, Specimen

The point of maximum tenderness is over the femoral attachment in the ligamentous injuries, and procaine infiltration may be used to abolish the local pain and demonstrate the absence of mechanical block to knee movements. In cartilage injuries the tenderness is directly over the joint line and the McMurray test will be helpful in differentiation.

3 *Osteochondritis of the femoral condyle and chondromalacia of the patella* must be considered. X-ray examination disclosing the defect of articular cartilage and the presence of loose bodies in the joint demonstrate the chief differentiating points.

4 *Hypertrophy of the infrapatellar pad of fat* may cause the symptoms of internal derangement when villous processes are nipped in the joint. Arthrotomy often is required to establish the diagnosis.

5 *Loose bodies*. Fibrous, cartilaginous and osseous loose bodies can give all the signs of internal derangement.

6 *Cysts of the semilunar cartilages*. Ganglionic degeneration which chiefly occurs in the lateral meniscus will be indicated by the tense local swelling on the joint line.

Treatment.—The locked knee caused by the displaced cartilage should be manipulated under anesthesia and the cartilage replaced. The joint is put through its full range of flexion and extension in varying degrees of rotation. This usually results in replacement of the displaced fragment.

Following manipulation a firm bandage is applied and quadriceps drill is initiated.

In recurrent cases and in cases of initial injury where the diagnosis is certain, arthrotomy with complete removal of the cartilage is the ideal therapy. Postoperative treatment consists of immediate quadriceps drill. Ambulation on crutches may begin as soon as the patient feels able. Stitches are removed on the 7th-8th day and flexion exercises begin between the 8th and 10th days. Good function is present after 3-6 weeks.

Bursal Lesions

Numerous bursae exist around the knee, and each one can be the site of an inflammatory process. The prepatellar and semimembranosus bursae are of most interest.

Prepatellar Bursitis.—This bursa is based on the patellar ligament and lower half of the patella and enables the skin and subcutaneous tissue of the kneeling surface to slide over the underlying hard tissues.

In the days before household appliances removed the hard work from domestic cleaning, the amateur housemaid who attempted the wide-swath technique of scrubbing would suffer a traumatic bursitis in this "buffer" between patella and floor. Thus, "housemaid's knee" received its designation. It consisted of a painful swelling of the prepatellar bursa due to repeated trauma. Today it is occasionally seen in those forced to kneel or it can follow a direct blow or a fall, or it may be associated with stellate fracture of the patella.

Gout may produce a bursitis at this site, and bilateral chronically thickened bursae may be due to gummatous infiltration. Syphilitic bursitis is much less common because of the present efficient primary treatment of this disease.

Treatment of traumatic bursitis consists of removing the cause. If the bursa is distended, the sanguineous fluid should be aspirated and a pressure bandage applied. When the disorder is recurrent or chronic, excision of the entire bursa is the treatment of choice.

Semimembranosus Bursitis.—This bursa is situated in relation to the semimembranosus tendon, the adjacent posteromedial capsule of the joint, and the medial head of gastrocnemius. In some cases the bursa communicates with the knee joint. Indeed, Wilson has referred to it as a "posterior herniation" of the joint. Clinically, the student should recognize two groups of patients who seek advice for a painful distention of this sac.

Most commonly the patient is a youth below the age of puberty who presents pain in the popliteal area extending into the upper calf and lower thigh. He complains that his knee feels stiff and that pain and stiffness are more severe after activity. The parent may have also noted a swelling at the back of the knee.

Examination discloses an ovoid, tense swelling somewhat to the medial side of the popliteal space which is prominent when the knee is in full extension. On palpation a tense swelling may be felt. On flexion it seems to dis-

appear. Considering these points, diagnosis is readily made.

The same clinical picture may be found in a patient past middle age who has an effusion into the knee joint associated with degenerative or other forms of arthritis, posterior meniscus tears, or following arthrotomy. Tense swelling in the popliteal space is noted on examination. Here the semimembranosus bursa communicates with the joint and acts as a blow valve for release of intra-articular pressure (Baker cyst).

The treatment in the two cases is different. In the primary bursitis of the youth, excision will produce a cure. In the bursal involvement secondary to articular disease the treatment is that of the primary lesion. Excision of the bursa alone may result in a synovial fistula.

FRACTURES OF COMPONENT BONES

Femur

Lower Femoral Epiphysis

Separation of the lower femoral epiphysis occurs before its fusion (18-20 years). The mechanism of injury is a forcible hyperextension by which the epiphysis displaces forward with the leg, the lower end of the femoral shaft projecting backward into the popliteal space. As in supracondylar fractures, there is a danger of injury to the popliteal vessels and nerves by the prominent shaft fragment. These epiphyseal injuries are uncommon but must be considered serious because of their tendency to interfere with growth and thus shorten or deform the limb.

Reduction.—Manipulation in the flexed position of the knee with traction on the leg and countertraction to the thigh and direct pressure backward on the displaced epiphysis secures an easy replacement.

The limb is splinted in a plaster cast with the knee at a right angle for 4 weeks. The cast is then changed to one with the knee in slight flexion for a further 3-4 weeks. When reduction is unstable, or as an alternative treatment in later adolescence, internal fixation by two wires inserted in cruciate manner may be employed. Avoidance of weight-bearing for 3 months by ambulation on crutches tends to lessen the tendency to premature synostosis.

Condylar and Intercondylar Fractures

These fractures are serious because of their involvement of this weight-bearing joint and the tendency to restriction of knee movements as a complication.

Mechanism.—Falls from a height, a direct blow, or valgus or varus strain gives the various types of fracture. Shearing off of one condyle is most commonly produced by forceful displacement of the leg to the corresponding side associated with a compression force through the knee by the body weight.

Clinical Picture.—The knee presents a grossly swollen appearance with marked hemarthrosis. X-rays disclose the site of fracture and the displacements.

Treatment.—Aspiration of the hemarthrosis is beneficial. Reduction must be anatomic. If this is possible through tibial skeletal traction and external compression, conservative treatment is indicated. In the grossly displaced fractures, open operation with evacuation of extravasated blood and internal fixation by screw or bolt, securing meticulous alignment of joint surfaces, is the ideal treatment.

Articular Fractures (Osteochondritis Dissecans, Paget's Quiet Necrosis)

This is a specific entity causing internal derangement of the knee. It is one of the group designated *osteochondritis* and in the knee most commonly occurs on the anterolateral aspect of the medial femoral condyle. Most authorities regard the etiology as traumatic. An acute articular fracture may be produced possibly by pressure of the cruciate ligament in a severe rotatory strain. Possibly a contusion through the patella may be the mechanism. Following such an injury an area of cartilage and underlying bone begins to sequester, and the patient may be seen either before or after the separation of the fragment. When first separated, there is cartilage on the one side and bone on the other. As time passes, fibrocartilage forms over the whole surface, giving a uniform appearance to the loose body when seen at operation.

Clinical Picture.—The patient is a young male of athletic habits, who gives a history

of injury to his knee some months previously. He now complains of the typical signs of internal derangement, i.e., recurrent effusion, weakness, locking, or giving way.

X-ray examination is conclusive. A loose body is seen when separation is complete. In earlier cases the lateral view shows the sequestrating fragment on the medial condyle.

Treatment.—Exploration is indicated. If the fragment is loose, it is removed and the joint thoroughly searched for other loose bodies. The condylar area is examined. Usually no surgical treatment is required to the healing area. If the fragment is in the process of separating, it should be removed and the area smoothed with gouge and chisel.

often a longitudinal fracture makes communication with the joint.

When the bumper strikes from the lateral or anterolateral aspect, a valgus strain of the knee develops, and the tibial tuberosity on the outer side is depressed and sheared off. The varying stages of injury are shown in Plate 84.

Clinical Picture.—This is similar to that presented by femoral condylar fractures. A hemarthrosis is usually present.

Treatment.—In cases without displacement of the tibial articular surface, aspiration of the hemarthrosis, manipulative reduction by traction in varus position, and compression of the tuberosities when spread, followed by plaster immobilization in this position, are used. Three

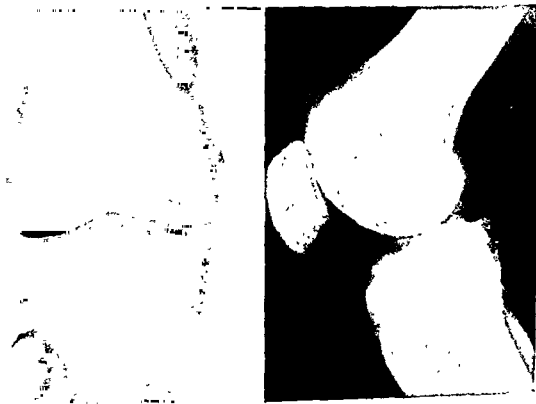


Fig 672.—Osteochondritis dissecans

Tibia

Bumper Fracture

Bumper fracture is so named because of the frequency with which it is caused by the car bumper in street accidents. When the leg is struck from the anterior aspect, the tibia is fractured just below the articular margin, and

to 4 weeks' immobilization is an average time, and this is followed by re education of knee function.

Where the lateral tuberosity is markedly displaced, open operation by a lateral parapatellar incision is performed. The displaced lateral meniscus is removed and the articular surface realigned. The limb is immobilized in varus

position in full extension for six weeks. Re-education of knee movements follows. Weight-bearing in the displaced articular fractures is best avoided for 3-4 months to minimize the traumatic arthritis.

Tibial Spine

Avulsion of the tibial spine occurs with anterior cruciate injuries. The fragment is displaced laterally and wedges between the lateral condyle and tibia, preventing complete extension.

The diagnosis is made by x-ray examination which demonstrates the displaced fragment.

Treatment.—Manipulation of the knee into complete extension and immobilization in a plaster cast for 6-8 weeks in this position may suffice. Open operation with replacement of the fragment and suture may be preferred. The bony fragment may be excised.

DISLOCATIONS OF THE KNEE

The knee is not frequently dislocated. The causative force may be direct to the upper end of the tibia or indirect through the leg. Lateral dislocation is the most common, although anterior and posterior displacements of the tibia on femur occur.

Severe capsular and ligamentous damage results. The cruciates are disrupted.

The serious nature is due to frequent pressure on the popliteal vessels, leading to gangrene. This occurs from the damage of the initial injury and sometimes is due to delayed reduction.

Clinical Picture.—The severe deformity is easily recognized. Careful examination to ascertain vascular and nervous injury is essential. X-rays are important.

Treatment.—The earliest possible reduction is imperative, and this is readily obtained under general or spinal anesthesia. If an anesthetic is not available, reduction should be attempted as a first-aid measure, using gentle continuous traction with the knee at 90 degrees, associated with pressure on the displaced tibia. Open operative suture of the torn capsule and ligaments is being used more frequently on these cases.

Postoperative therapy consists of protection of the capsular tissues from strain until well healed (3 months), associated with quadriceps drill.

ARTHRITIDES

The knee joint is frequently involved in the various forms of arthritis found in other joints. A simple classification is as follows:

1. Traumatic arthritis, including the stiff knee from quadriceps fixation
2. Degenerative or osteoarthritis
3. Infective arthritis, including pyogenic and specific types
4. Rheumatic and rheumatoid
5. Various other types, including gouty arthritis

We cannot deal here with each in detail, but the student should be reminded of the great improvement in the results of treatment in the *pyogenic group* following the use of parenteral and intra-articular penicillin. In these cases, aspiration is carried out and the initial intra-articular injection of penicillin given, combined with massive systemic antibiotic therapy. The antibiotic to which the strain of organism is proved most sensitive should be selected.

Gonorrheal arthritis is common in the knee. It is usually treated with penicillin. If penicillin is not available, sulfadiazine is applied to this disorder. Conservative therapy is routine in the young, whereas arthrodesis after a period of rest with streptomycin, isoniazid and para-aminosalicylic acid therapy is acceptable for the adult. (See also page 65.)

The bilateral *gummatous synovitis* (Clutton's joints) seen in the congenital syphilitic at puberty and the *Charcot's joints* of the tabetic should also be kept in mind in differential diagnosis.

The knees are frequently involved bilaterally in *rheumatoid arthritis*. Here advance in treatment has come with the use of cortisone, gold therapy, and the prevention of deformity. Late cases may require synovectomy or arthrodesis.

Traumatic arthritis and *quadriceps fixation* are most commonly seen following fractures of the femoral shaft. This complication can be minimized by maintaining muscular exercises

throughout the period of immobilization, by keeping the patella mobile, and by the avoidance of excessive traction through the joint and of prolonged fixation in splints

The improvement obtainable in cases of *degenerative arthritis* and *osteoarthritis* has been most interesting. The x-rays here give the diagnosis when characteristic osteophytic formations are found. Weight reduction, physiotherapy, and intra-articular hydrocortisone give marked symptomatic relief in a large proportion of cases

CONGENITAL ANOMALIES

To complete the picture, the three common anomalies should be mentioned, i.e., *genu valgum* (knock-knee), *genu varum* (bowlegs), and *genu recurvatum* (hyperextension, bandy legs), which can be greatly helped by orthopedic procedures applied in the early stages of the deformities

SUMMARY

Disorders of the knee have been considered largely from the point of view of the derange-

ments characteristic of this articulation. The student should again be reminded of the importance of the extensor musculature in maintaining the stability and movements of the knee joint, and he will greatly improve the results in these disorders by enforcing quadriceps exercises in all cases of traumatic disorders. Resistance exercises will also assist the final rehabilitation of these common clinical problems

TIBIA AND FIBULA

Fractures of the tibia and fibula are of frequent occurrence. The subcutaneous position of the tibia accounts for the fact that its fractures are more often open or compound in nature than those of any other major bone.

Mechanism.—Fracture of either bone singly is usually the result of direct trauma. Both bones may be broken by direct or indirect violence. Direct violence gives rise to transverse fractures at the same level and such may be directly compounded. Indirect violence accounts for the spiral types at varying levels,



Fig. 673.—X-rays of fracture of tibia and fibula immediately after plating and 1 month later, showing clinical union

and the compounding occurs when a sharp spicule of bone penetrates the skin from within.

Displacement is frequent and overlap occurs from muscular spasm. The oblique fracture is especially prone to interposition of soft parts and is unstable after manipulative reduction.

Clinical Picture.—The nature of the accident determines the clinical picture. Fracture of a single bone causes little alteration in the normal contour of the leg, but involvement of both bones may be accompanied by considerable deformity.

The presence of a surface wound and the degree of swelling must be carefully noted.

X-rays show the type of fracture and presence of comminution.

Treatment.—Fractures of the tibia and fibula are serious injuries because of the time required to secure union. This time factor in-

creases as the line of fracture is placed lower in the tibia. The upper third consists of cancellous bone and is surrounded on two thirds of its surface with muscles. The blood supply is good to both fragments. The lower third has little muscle in relation to its surface, tendons having arisen to pass into the foot. The bone has a poor blood supply and little cancellous tissue. The middle third is intermediate in this respect. The presence, therefore, of butterfly fragments devoid of circulation in fractures in the lower half indicates a prolonged period before union.

Four methods of treatment are available.

- 1 Manipulation and plaster cast
- 2 Skeletal traction
- 3 Open operation with internal fixation
- 4 External skeletal fixation

Manipulation and Plaster Cast.—Fracture of the fibula alone requires little treatment apart

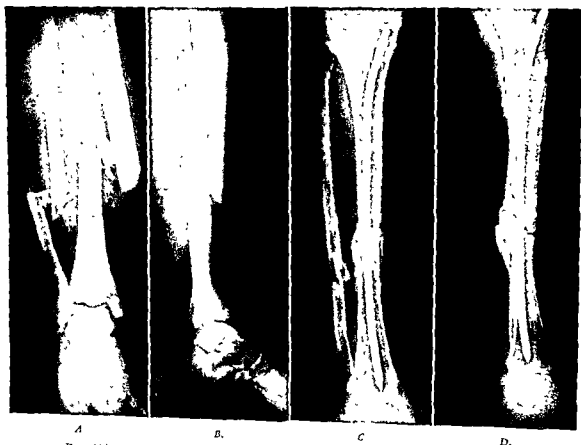


Fig. 674—A and B, Gross open fracture of tibia and fibula treated by débridement, primary suture, and skeletal traction until wounds healed.

C and D, Similar anterior and lateral views 3 months after intramedullary nailing (Lottes' nail) and cancellous bone chaps to the area of the freshened bone ends.

from protection. The tibia affords adequate splinting.

Fracture of the tibia alone without displacement is best treated in a midhigh cast with the knee slightly flexed and foot at a right angle.

Fractures of the tibia and fibula without displacement, or those in the young or adult that are transverse and stable after manipulation, can be treated in a similar way.

Skeletal Traction—Displaced fractures of the tibia and fibula, especially if oblique or comminuted, can be well treated by skeletal traction through the os calcis on a Braun splint. (See general section on Fractures.) Preliminary manipulation under anesthesia is used to obtain alignment.

Weights vary from 6-12 pounds, and the position is followed by x-rays. After 3-4 weeks the limb is placed in a cast from midthigh to toes, and ambulation on crutches is begun.

Internal Fixation—In cases of fracture of both bones resisting reduction by manipulation or traction, or when compounded or both limbs are fractured, or other injuries justify, open operative correction and fixation by screws, plates, or intramedullary nail is used. Post-operative therapy includes protection by plaster cast and ambulation on crutches.

External Skeletal Fixation—This method has been successfully used for displaced fractures of both bones.

Time Factor.—In all cases a period of 12-16 weeks is required, depending upon the patient's age and the position of fracture. Fractures in the lower third in the older patient require even longer immobilization.

Complications.—

Delayed Union.—Delayed union is difficult of definition but in the case of tibial fractures may be said to be present when movement still persists on changing the cast between 3-4 months. When this is found, it is wise to accelerate healing by the application of an onlay bone graft to the anterolateral surface or by a sliding bone graft on the anteromedial surface.

Nonunion.—When clinical examination after 6 months discloses movement at the site of fracture, and x-rays show sclerosis of the bone ends and minimal callus, union should be

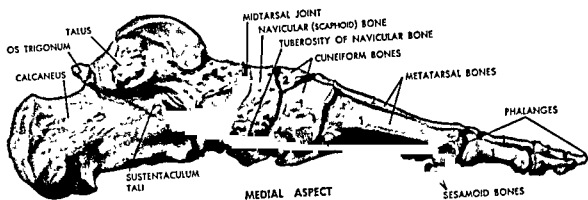
assisted by removing the scar tissue and metallic fixation if present. This is followed by application of a sliding or onlay bone graft. Cancellous bone can be placed around the fracture line. Adequate plaster splintage is required postoperatively. *Delayed and nonunion of tibial fractures may be caused by an intact fibula which prevents contact compression of the fracture surfaces. When such a problem exists, an oblique osteotomy of the fibula should be performed.*

In both delayed union and nonunion, general treatment directed to correct dietary or mineral deficiency should be instituted.

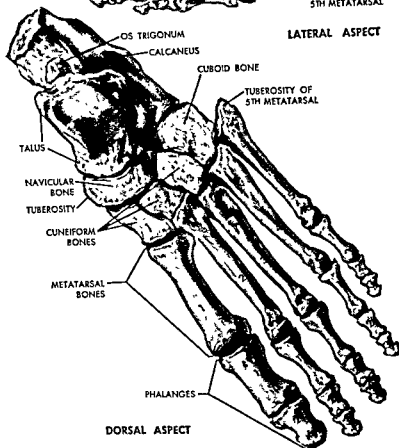
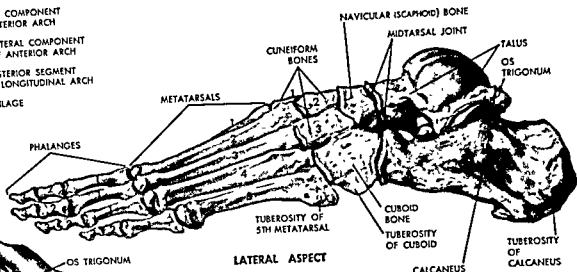
Chronic Osteomyelitis.—This complication of open fractures involving the tibia, lessened by chemotherapy, is best treated by saucer-



Fig. 675.—Fracture through area of saucerization which had been completely healed with split thickness skin graft.



RED MEDIAL COMPONENT
OF ANTERIOR ARCH
YELLOW LATERAL COMPONENT
OF ANTERIOR ARCH
GREEN POSTERIOR SEGMENT
OF LONGITUDINAL ARCH
BLUE CARTILAGE



ARTICULAR SURFACE
OF TIBIO-FIBULAR
MORTISE

F. Netter M.D.
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Plate 86.—Bones of the Ankle and Foot.

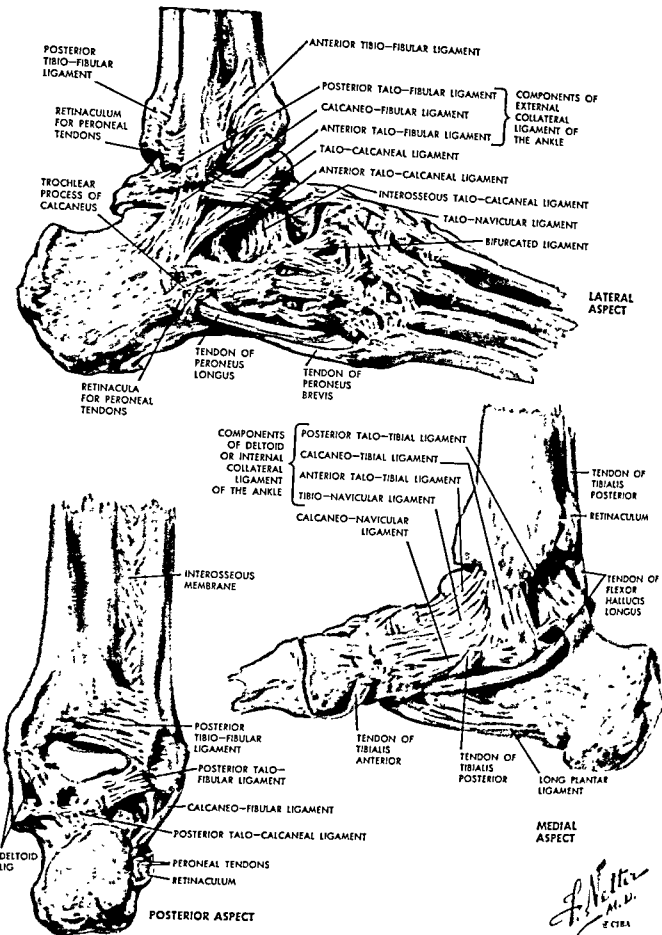


Plate 87.—Ligaments of the Ankle and Foot.

zation of the diseased bone and application of a split-thickness skin graft. Cancellous bone may be used later to fill cavities. Plastic procedures supplement the initial treatment.

ANKLE AND FOOT

TRAUMATIC DISORDERS OF THE ANKLE AND FOOT

Anatomic Considerations

Osseous Components (Plate 86)

The foot is composed of 14 phalangeal, 5 metatarsal, and 7 tarsal bones, articulating through the talus, with the mortise of the ankle joint formed by the lower ends of the tibia and fibula. Accessory bones of minor clinical significance are also present.

The foot has a main longitudinal arch, of which the talus is the keystone. The calcaneus forms the posterior segment. The anterior segment is best regarded as composed of two functional components, a *medial component* which is mostly concerned with propulsion and a *lateral component* which chiefly serves for balance.

In the erect posture the body weight is transmitted through the ankle joint to the foot. Depending on the stance, the weight is largely borne by the heel and ball of the great toe, with the lateral border of the foot, the lateral metatarsal heads, and the toes acting for balance. In walking, the heel first receives the weight, which is transferred to the ball of the great toe as plantar flexion occurs. As the heel leaves the ground, the whole weight is finally taken on this part of the forefoot.

Ligaments (Plate 87)

The ankle and knee are the articulations most prone to ligamentous sprains, and such disorders have the greatest practical significance. For the understanding of sprains, whether or not associated with the fractures or fracture-dislocations, detailed knowledge is necessary of the interosseous relationships and ligamentous attachments.

Most important in this respect are the interosseous ligament and membrane and the anterior and posterior tibiofibular ligaments strengthening the capsule of the inferior tibiofibular articulation. These fibrous bands, together with the external and internal collateral ligaments, maintain the integrity of the ankle mortise.

The Interosseous Ligament and Membrane.—The interosseous ligament binds the lower ends of the tibia and fibula together and, with the anterior and posterior tibiofibular ligaments, stabilizes the inferior tibiofibular syndesmosis. The fibers of the interosseous ligament run from above outward and downward from tibia to fibula, and this ligament is continuous above with the interosseous membrane whose fibers run in the same general direction. The interosseous membrane functions to prevent bending of the fibula when under strain.

The interosseous ligament and membrane are torn in diastasis of the inferior tibiofibular syndesmosis.

Anterior Tibiofibular Ligament.—The fibers of this ligament have a course downward and outward from the tibia to the fibula. It is ruptured in diastasis of the inferior tibiofibular articulation due to abduction force. When the deforming force is carried to the extreme, there is an associated rupture of the interosseous ligament and membrane with fracture of the fibula 3" or more from its tip (*Dupuytren's fracture*).

Posterior Tibiofibular Ligament.—This fibrous band corresponds to its anterior counterpart in direction of fibers. In spiral fractures of the fibula due to an external rotation force, this posterior ligament remains intact, while the anterior is ruptured. In complete diastasis both the anterior and posterior ligaments are torn.

External Collateral Ligament.—This ligament is the one most frequently involved in soft tissue disorders of the ankle. It consists of three distinct bands:

1. The anterior talofibular ligament passing from the tip of the fibula to the base of the neck of the talus on its lateral aspect
2. The calcaneofibular ligament extending downward and slightly posteriorly to the calcaneus

3. The posterior talofibular ligament passing horizontally backward to insert on the tubercle of the talus

The anterior talofibular ligament and the calcaneofibular ligament are the bands avulsed by forced inversion. The rupture is most commonly at the fibular attachment and the tip of the external malleolus may be avulsed at the time of injury. This injury permits subluxation of the ankle and, if neglected, gives rise to recurrent subluxation of the joint.

Internal Collateral or Deltoid Ligament.—The deltoid ligament is really the strong fibers of the medial portion of the articular capsule of the ankle joint. Its fibers course from the internal malleolus to the navicular, sustentaculum tali, and posterior area of the talus. It forms four main bands, i.e., the tibionavicular, the anterior talotibial, the calcaneotibial, and the posterior talotibial ligaments.

This ligament is ruptured by forces of abduction or external rotation, and the tear usually takes place at its tibial attachment. The insertion of the ligament to the tibial malleolus is so strong that avulsion of the internal malleolus is the rule.

Muscles and Their Functions

(Plates 88-90)

A simple basic comprehension of the muscle balance of the foot can be gained by examining some of the important muscles and their functions.

Gastrocnemius and Soleus.—These calf muscles act through the tendo achillis to produce plantar flexion of the foot and of the lateral component of the anterior segment of the longitudinal arch. When contraction of the muscles is carried further, it produces an adduction and inversion of the forefoot.

The proximal attachment of the soleus to the tibia, in contradistinction to the origin of the gastrocnemius on the femoral condyles, enables the above function to be carried out when the knee is in flexion.

Peroneus Longus.—This muscle also functions to secure plantar flexion. It balances the tendency to inversion of the lateral component of the forefoot, just described, by a force of eversion applied to the medial component of the forefoot exerted through the head of the

first metatarsal. The ball of the great toe joint is therefore held firmly to the ground, affording the fulcrum necessary for propulsion.

Tibialis Anterior.—This powerful muscle produces dorsiflexion and inversion of the foot, in which movement it is assisted by the extensor hallucis longus acting chiefly on the proximal phalanx of the great toe.

Extensor Digitorum Longus.—This muscle acts as a balancing force with the tibialis anterior and extensor hallucis longus as a dorsiflexor of the foot. By its power of eversion of the forefoot, it counterbalances their action of inversion, thus assisting the movement of dorsiflexion.

Tibialis Posterior and Peroneus Brevis.—The first of these two muscles produces adduction and the second produces abduction of the forefoot. Both assist in plantar flexion.

Abductor Hallucis, Flexor Hallucis Brevis, and Adductor Hallucis.—These all act in forcible plantar flexion of the proximal phalanx of the great toe, leaving the distal phalanx lax and thus facilitating propulsion. The muscles moving the toes correspond to those for the fingers. It should be reiterated that the toes, apart from the hallux, are chiefly concerned with balance.

Muscles Acting in Walking.—As the heel strikes the ground, tension in the dorsiflexors (tibialis anterior, extensor digitorum communis, and extensor hallucis longus) holds the foot approximately at right angles to the leg until the weight is transferred to the ball of the foot. Then the gastrocnemius and soleus lift the heel. Assisting in this are the peroneus longus which depresses the medial component of the foot and the adductor, the abductor, and the flexor hallucis brevis which flex the great toe to give the final "push off."

General Examination of the Ankle and Foot

This region should receive careful inspection with the patient standing, walking, and sitting. Any deformity or abnormality should be noted. The skin and underlying soft tissues of the lower extremities afford a good indication of the circulatory status, impairment being revealed by dependent edema or discoloration. The dorsalis pedis and posterior tibial arteries

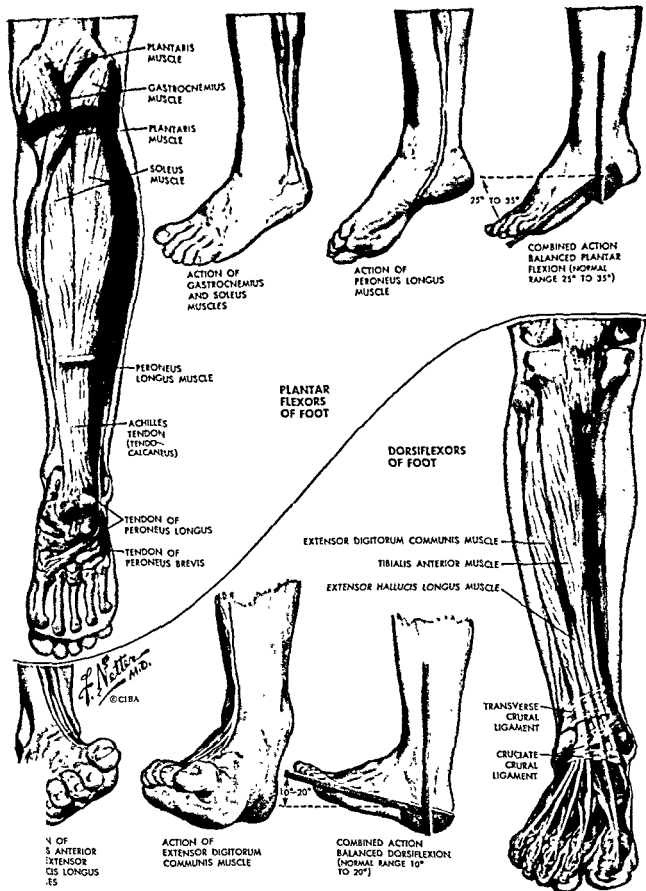


Plate 88.—Plantar Flexors and Dorsiflexors of the Foot.

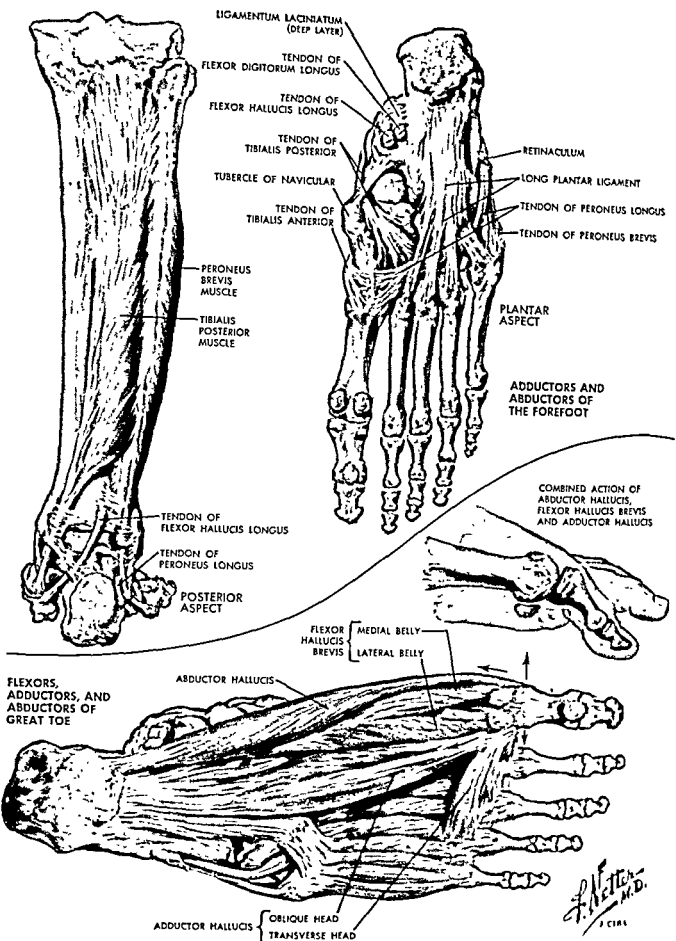


Plate 89.—Adductors, Abductors, and Intrinsic Muscles.



GASTROCNEMIUS AND SOLEUS
RAISE HEEL FROM GROUND
AND THROW WEIGHT ON
HALL OF FOOT



2 PERONEUS LONGUS
COMES INTO PLAY,
DEPRESSING MEDIAL
COMPONENT OF FOREFOOT
AND THROWING WEIGHT ON
HEAD OF FIRST METATARSAL



3 ADDUCTOR, ABDUCTOR
AND FLEXOR HALLUCIS BREVIS,
FLEX THE GREAT TOE AND
THUS EXERT A FINAL LIFTING
MOTION OR 'PUSH OFF' TO
THE FOOT

J. Netter M.D.
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Plate 90.—Muscle Action in Walking.



INVERSION SPRAIN
(RUPTURE OF
CALCaneo-TIBULAR
AND TALo-TIBULAR
LIGAMENTS)



INVERSION SPRAIN-FRACTURE
(AVULSION OF
FRAGMENT OF
TIBIAL)



ABDUCTION SPRAIN
(RUPTURE OF DELTOID
LIGAMENT)



ABDUCTION SPRAIN
(RUPTURE OF
ANTERIOR
TIBIO-FIBULAR
LIGAMENT;
DIASTASIS)



DIASTASIS
WITH AVULSION
OF TIBIAL
FRAGMENT

must always be palpated to determine relative patency.

The movements of the ankle should be recorded, remembering the variability between the hypermobile and stable types. Dorsiflexion of 10-20 degrees and plantar flexion of 25-35 degrees constitute an average range. Dorsiflexion will be limited in the female patient accustomed to high heels, in whom a contraction of the calf muscles is present.

The subtalar joint between the talus and the calcaneus is tested by a side-to-side rocking movement to elicit pain or demonstrate stiffness. This articulation is the shock absorber of the foot, enabling adjustment when traversing irregular ground. The midtarsal joints should be moved to note their range in adduction and abduction.

Sprains (Plate 91)

A *sprain* may be defined as the rupture of some or all of the fibers of a ligament. If the injury does not result in instability of the joint, the sprain may be termed *minor*, but if instability results, the designation *major* is given. When the ligament itself does not rupture but avulses the fragment of bone to which it is attached, a *sprain fracture* has occurred.

Inspection.—The ankle region is observed to note the presence of general or localized swelling, discoloration, or blister formation.

Palpation.—The areas of the malleoli and tarsal bones are palpated to localize the points of maximum tenderness, indicating the points of ligamentous rupture.

Manipulation.—The foot is moved on the ankle into external rotation, internal rotation, abduction, adduction, plantar flexion, and dorsiflexion to bring out the movements that cause pain. This information is correlated with the findings on palpation. It may be necessary to infiltrate the painful torn ligaments with a local anesthetic before manipulation can be properly performed. Excessive mobility will indicate an instability from a major sprain. Separation of a fragment of bone will indicate a sprain fracture. X-rays taken in positions of strain will add confirmatory information.

Treatment of Minor Sprains.—Swelling due to minor sprains may be controlled by the

immediate application of a pressure dressing, a cold pack, and rest from weight-bearing. Strapping is applied in such a manner as to relax the affected ligament. Procaine infiltration will alleviate pain and facilitate ambulation. Infra-red heat, massage, and active exercises will secure a rapid recovery.

Major Sprains

Two important groups of major sprains and sprain fractures of the ankle occur. These are *inversion sprains*, injuring the fibulocalcaneal and anterior fibulotalar components of the external collateral ligament, and *abduction sprains*, involving the superior attachment of the deltoid or internal collateral ligament.

Major Inversion Sprains.—Close-knit and lax types of joints are found in the articulations of the ankle and foot. The *lax joint mechanism* gives rise to the hypermobile ankle and tarsal joints found in slender individuals. Bonnin places the incidence at 4%. Such persons have feet that are hypermobile on inversion, and the foot can be moved into a position so that the sole is inverted at practically a right angle with the ground. Examination of the lateral aspect shows the prominence of the fibular malleolus, the talus, and the cuboid. In the stable foot, this marked inversion is prevented by the fibulocalcaneal ligament which stands out as a firm, palpable band when such a movement is forced.

A major sprain or sprain fracture of this ligament in the stable foot results in the same clinical appearance found normally in the hypermobile foot. The foot can be markedly inverted. In the acute lesion, the diagnosis is made by finding acute tenderness and swelling over the tip of the lateral malleolus. Pain often prevents the foot from being placed in the position of the complete displacement that occurred at the time of the accident. Satisfactory evaluation requires local procaine infiltration of the area, with radiologic examination in the position of strain. Such investigation will disclose subluxation of the talus from the ankle mortise (Fig 676).

Treatment.—Diagnosis of a major sprain of the fibulocalcaneal ligament necessitates plaster immobilization for 8-12 weeks with the ligament in a position of relaxation. Because of

this prolonged period many surgeons consider that primary suture is justified and, with more exact diagnosis in the early stages, this form of initial treatment is more and more frequently performed. Ambulation is permitted in a below-knee walking cast. Following the period of plaster immobilization, an elastic bandage or stocking should be worn and physiotherapy given.

habitus or as a result of previous fibulocalcaneal rupture, there is the clinical syndrome of recurrent weakness, giving way, going over, or further stages of inversion sprain associated with pain and swelling.

In the patients without an initial major sprain, palliative treatment consists of a laced ankle support, care to avoid worn shoe heels, and a raise on the outer side of the heel and



Fig. 676—Subluxation of left ankle with avulsion of the anterior fibulotalar and fibulocalcaneal ligaments. Both the left and right ankles are in forced inversion, demonstrating the subluxation on the left side when compared to the normal right ankle.

Recurrent Subluxation of the Ankle—The stable ankle has the taut fibulocalcaneal ligament and adequate muscle balance to maintain this state. The hypermobile or lax joint has a relaxed fibulocalcaneal ligament due to inherent laxity in the asthenic individual or to previous rupture with inadequate treatment in cases resulting from trauma. In the slender type with powerful muscle balance, as found in ballet and acrobatic dancers, prevention of sprains is due to muscular control. In those individuals with poorly developed musculature who have hypermobile ankles as part of their

sole, associated with active exercises directed especially to develop the muscles, such as the peroneus longus and brevis, which oppose the inversion of the foot produced by the tibialis anterior and posterior and triceps surae.

In cases resulting from a major sprain of the fibulocalcaneal ligament, primary treatment by adequate plaster immobilization, with or without operative suture, will prevent its development. In neglected cases, reconstruction of the ligament by tenodesis, using part of the peroneus brevis tendon (Watson-Jones) or with fascia lata, has given satisfactory results.

Major Abduction Sprains.—Major sprains of the abduction types are second in frequency of the neglected traumatic disorders of the ankle, and they often lead to disabling sequelae.

When the foot is forcibly abducted, tension develops in the internal collateral or deltoid ligament. A minor sprain may result, which is readily relieved by bandaging the part with the foot adducted, thus relaxing the injured ligamentous fibers.

Should the deforming force continue beyond this point, the ligament may rupture near its periosteal attachment or, more commonly, may avulse a varying portion of the internal malleolus. In other cases, forced abduction may produce diastasis of the inferior tibiofibular articulation, or these two lesions may occur together.

These cases are diagnosed by finding acute tenderness and swelling around the tip of the internal malleolus. A sulcus indicating the separation is sometimes palpable. Radiologic examination will disclose any avulsed fragment of bone, and the extent of separation will determine the advisability of operative intervention and repair by suture or screw fixation.

Sometimes the abduction force springs the inferior tibiofibular and interosseous ligaments which bind the tibia and fibula together, and a *diastasis* results. When this occurs clinical examination affords evidence of a severe ankle injury. However, the radiologic report is frequently negative because the bones recoil into position and, unless x-rays are repeated in the position of abduction strain, the diagnosis will be overlooked and a serious disability will ensue (See Plate 91 and Fig. 678).

The student should have in mind the following points to assist in the recognition of these cases:

1. *The clinical picture is that of a severe injury to the ankle. There is gross swelling all around the ankle which appears broader than normal. The patient avoids weight-bearing because of the severity of the pain.*

2. *The point of maximum tenderness is localized on the anterior aspect of the tibiofibular joint just medial to the fibula. There may be a second point of tenderness at the tip of the internal malleolus.*

3. *Radiographs, which on casual inspection may seem negative, on careful study will often reveal a widening of the mortise. This may appear on the medial side in the anteroposterior view. Also, a flake of bone may have been avulsed from the lateral aspect of the tibia. This flake of the tibial tuberosity is often concealed by the fibular shadow but will be perceived on careful examination, if present and suspected.*

The absolute proof of diastasis is obtained by local anesthetic infiltration of the joint, followed by radiologic studies with the foot in forced abduction.

Treatment.—Once the diagnosis is established, two approaches to treatment are possible.

1. *Immobilization in a below-knee plaster cast with the malleolar areas carefully compressed; the foot held at 90-100 degrees.* The cast, which should be changed when the swelling subsides, is maintained for 12 weeks. No weight-bearing through the affected limb should be permitted for at least 6 weeks, but ambulation on crutches is allowed.

2. *Internal fixation by a long screw through the fibula into the tibia.* Plaster immobilization is again required for 12 weeks. The screw must always be removed, as some movement in the tibiofibular joint is essential. This removal may be performed after 6 weeks. In cases where the ligaments have avulsed the tuberosities of the tibia, the healing appears more rapid, and immobilization for 8 weeks is sufficient.

Tendon Lesions

A sudden forced effort of plantar flexion while the foot is supporting the body weight, with or without a superadded load, may cause either a rupture of the plantaris or a rupture of the tendo achillis.

Rupture of the Plantaris

This occurs more frequently in the young athlete at the beginning of a spring, jump, or dive. There is a sudden pain in the calf, a distinct feeling of something giving way, and sometimes an audible snap. The calf becomes swollen, hard, and tender. Walking may be difficult. A "Charley horse" is the

usual lay diagnosis. In the older person a deep phlebitis must be differentiated.

Treatment.—The condition clears in 10-21 days and is assisted by firm elastic bandaging of the limb from the foot to below the knee. A raised heel on the shoe will diminish the tension in the calf when walking and afford considerable relief. Heat, massage, and active exercises will accelerate the recovery after the first 48 hours. There is no contraindication to ambulation or weight-bearing once the discomfort lessens.

Rupture of the Tendo Achillis

(Plate 92)

This tendon, which is the strongest in the body, ruptures only on a sudden forcible effort, and only after undergoing degenerative changes due to aging or a specific condition, such as syphilis or gout. The great John Hunter sustained this injury while dancing, and there exists an excellent record of his case with the findings at autopsy years later. Rupture frequently occurs in entertainers who carry on beyond their prime of life.

The history is that of a sudden effort which is accompanied by a snap with pain in the calf, after which the foot becomes useless. Plantar flexion of the foot is impossible.

Examination.—With the patient in the prone position, examination discloses a loss of the normal outline of the Achilles tendon and, with the foot passively dorsiflexed, the sulcus at the site of rupture is palpable. Sometimes the rupture occurs at the musculotendinous junction, and then the diagnosis is more difficult. At other times avulsion of the calcaneal insertion occurs. Typically, however, rupture leaves a part of the tendon attached to the bone.

Treatment.—The ideal treatment is operative suture at the earliest moment, with the site of rupture exposed through a longitudinal incision placed along its lateral border.

The limb is encased, postoperatively, in a below-knee walking cast with the foot at 90-100 degrees. The author does not use the position of plantar flexion to relax the tendon because of the tendency to contracture of the calf muscles.

Plaster fixation is continued for a period of 6 weeks. During this time ambulation is permitted. Following this period, an elastic bandage is employed, and massage and active exercises are used to restore function to the part. Return to violent exercise such as acrobatic work is delayed for 4-6 months. When a shoe is worn, a raised heel, gradually diminished in height over several weeks' time, is advisable.

Recurrent Dislocation of the Peroneal Tendons

The tendons of peroneus longus and brevis pass behind the fibular malleolus in a bony groove of varying depth. Their displacement in the different positions of the foot is prevented by the superior and inferior peroneal retinacula. Stretching or rupture of the superior retinaculum results from a forced eversion while the foot is in plantar flexion.

This rupture permits the peroneal tendons to slip out of the bony groove onto the lateral aspect of the external malleolus. Should the initial lesion remain undiagnosed and untreated, this tendency to recurrent dislocation of the tendons persists as a painful syndrome which occurs on sudden eversion with plantar flexion of the foot.

Treatment.—Primary treatment consists of replacing the tendons and firmly bandaging the foot so as to limit eversion and plantar flexion for 3-4 weeks.

When seen as a recurrent disorder, if the patient's disability is sufficiently severe, a reconstruction of the superior retinaculum, using fascia or a strip of tendon from the peroneus longus or brevis, gives excellent results. Deepening the bony sulcus is usually unnecessary.

Avulsion of the Insertion of Peroneus Brevis

The peroneus brevis is inserted on the prominent tubercle located at the base of the 5th metatarsal. A forcible inversion of the foot against resistance may avulse this tubercle. This injury is often called "dancer's fracture" because of its occurrence while dancing. The diagnosis will be made by the history of a sudden strain, followed by local tenderness with swelling and pain at the base of the 5th metatarsal. Radiologic examination will

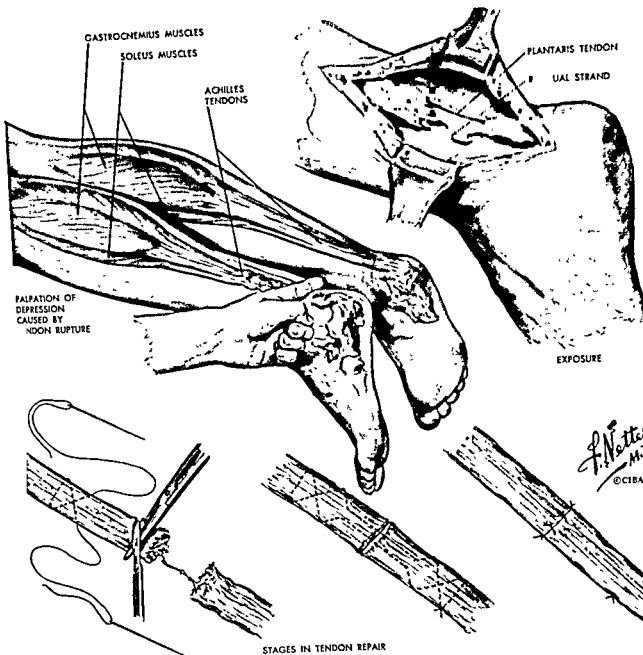


Plate 92 —Rupture of Achilles Tendon.

Courtesy Moseley, H. F. CIBA
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demonstrate the fracture and extent of separation.

Treatment.—When displacement is absent, immobilization by strapping suffices. When displacement is present, as indicated by a definite gap shown on the x-ray, a below-knee walking cast for 3-4 weeks gives symptomatic relief and promotes recovery.

Tenosynovitis

Two painful conditions of tendons should be mentioned. They are achillodynia and tenosynovitis of the tibialis anterior. They correspond to a similar disorder of the lower forearm in which the tendons of the abductor pollicis longus and the extensor pollicis brevis cross the radial extensors. The pathologic process is a tenosynovitis produced by such factors as overuse, pressure, or friction.

In *achillodynia*, the synovial membrane, which surrounds the tendo achillis, becomes inflamed. Local tenderness is found on pressure and there is pain with crepitus on dorsal and plantar flexion.

Relief is obtained by elevating the heel of the shoe $\frac{3}{16}$ – $\frac{5}{16}$ " (0.5–0.75 cm). A firm bandage is applied to the ankle and foot over two strips of felt placed one on each side of the tendon. Treatment with hot packs and diathermy is soothing. Counterirritants associated with the administration of salicylates 10 gr. and cortisone derivatives may hasten the recovery.

With *tenosynovitis of the tibialis anterior*, the patient complains of pain on the anterior aspect of the lower third of the leg. Examination will localize the discomfort to the anterior tibial compartment, where the tibialis anterior muscle lies against the anterior border of the tibia. On active dorsiflexion and inversion of the foot, the discomfort is aggravated. A fine crepitus over the tendon is often discernible during these movements.

Treatment.—This consists of firm bandaging of the foot and leg, together with the use of local counterirritants and preparations of cortisone and salicylates.

Fractures and Dislocations of the Ankle

These injuries of the ankle are often grouped together and called Pott's fractures

This corresponds to the designation of fractures and fracture-dislocations of the lower end of the radius as Colles' fractures. It must be remembered that this is a relic of the days before x-rays and that the many varieties of injuries so loosely designated require individual consideration if improvement in standards of treatment is to be obtained.

The classification of ankle injuries follows the writings of Ashhurst, who considered them on a mechanistic basis.

Mechanism of Fractures and Fracture-Dislocations.—The foot may be violently displaced on the leg by the following forces:

1. External rotation
2. Abduction
3. External rotation and abduction combined
4. Adduction
5. Vertical compression

Depending on the intensity and duration of the force, different degrees of fracture and dislocation occur. This is best indicated by diagrams taken from type x-rays, but it must be remembered that all gradations occur. Further, there is frequently an associated compression force when the body weight is transmitted in injuries caused by falls from a height.

Diagnosis.—Fractures without displacement are differentiated from sprains by x-ray examination.

Fractures and dislocations with deformity often present the type appearance expected from the responsible mechanism, e.g., the abducted foot with posterior displacement in the third degree abduction injury. Marked swelling from edema and hematoma may mask the bony landmarks. X-ray examination with anteroposterior, lateral, and sometimes bimalleolar views is required.

Treatment.—

Emergency Measures.—When first seen any gross displacement should be gently corrected to prevent compounding the fracture and to diminish circulatory embarrassment. A firm support for the foot, ankle, and lower leg is arranged, using an Elastocrepe bandage applied over absorbent cotton. A most useful form of splintage is the pillow splint available in any home. This is most satisfactory for transportation to hospital or if the patient must remain at home overnight. The limb is elevated and ice bags are applied. These measures will

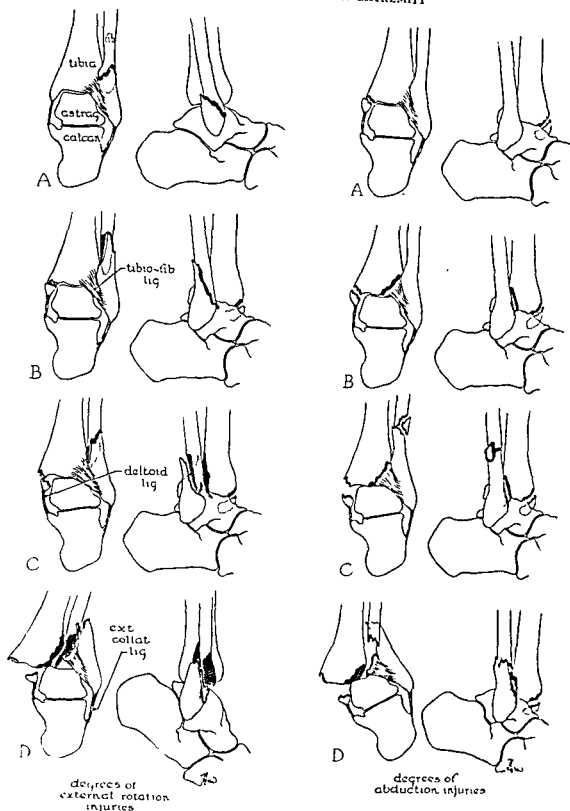


Fig. 677—A, B, C, and D represent stages of severity of the type fractures or fracture dislocations and are often designated first, second, third, and fourth degrees. Needless to say, every gradation exists. The important point is that instability of the mortise occurs from the second degree onward, necessitating most careful plaster splinting, the increasing use of internal fixation, and the avoidance of early weight bearing.

diminish the tendency to swelling which will delay the definitive treatment in these cases. The patient is reassured, and analgesics for pain with sedatives to ensure sleep are arranged.

Principles.—Fractures and fracture-dislocations require exact reduction because this is a weight-bearing joint and the mortise must be

properly restored. In the severe injuries with loss of joint stability, this reduction must be maintained until solid healing has occurred in both bony and ligamentous structures. It is best to avoid weight-bearing for as long as 6 weeks in these severe cases, and plaster casts must be changed as the swelling diminishes and careful molding of the cast around the

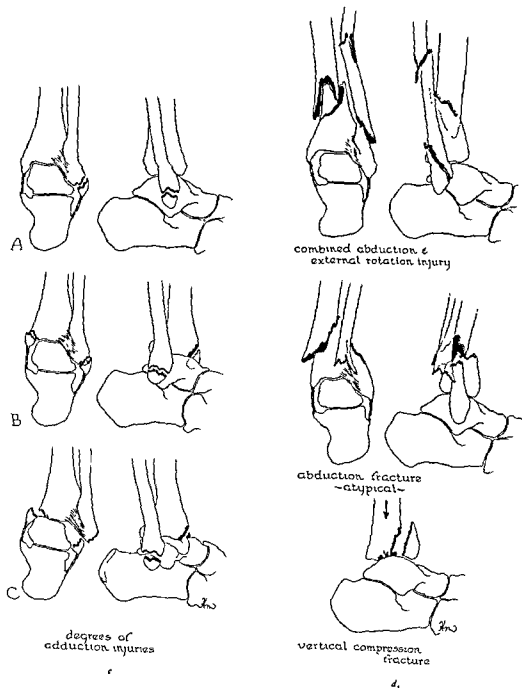


Fig 677, c and d.—Type fractures of ankle

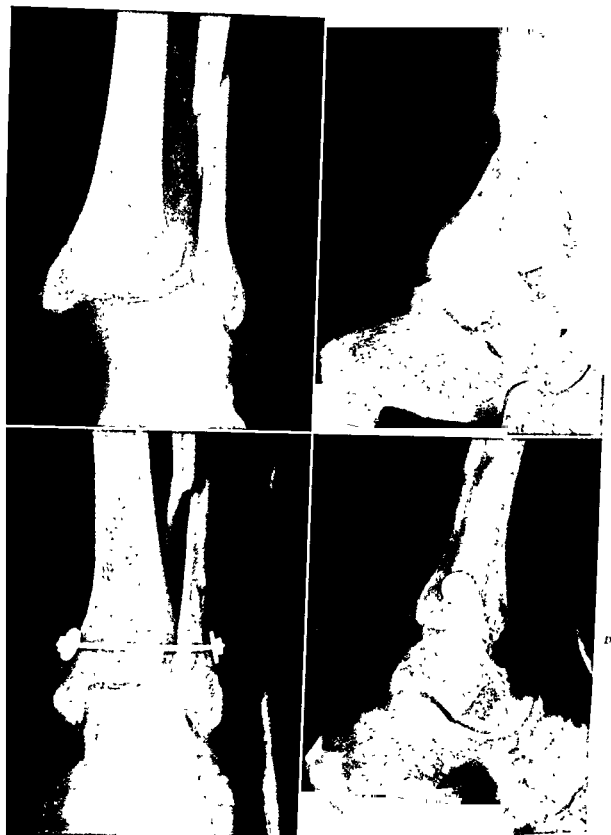


Fig. 678—Third degree fracture-dislocation produced by abduction

A. Anteroposterior view. Lateral displacement of talus with diastasis of tibiofibular joint and fracture of fibula. Internal collateral or deltoid ligament must also be avulsed (Dupuytren's fracture)

B. Lateral view. Posterior displacement of talus, together with fracture of posterior lip of tibial articular surface

C and *D.* Result achieved by operative reduction and internal fixation by bolt, washers, and nut

malleoli is required. Movements of all muscles under the cast are encouraged to diminish disuse atrophy. Elevation of the limb to prevent edema is important. When the cast is finally removed, support by Unna's paste bandage or elastic stocking is essential, and this should be combined with physiotherapy.

Reduction.—The earliest possible reduction of all major displacements is required. This is achieved by reversing the mechanism of the injury. Most posterior displacements are reduced by plantar flexing the foot, drawing downward and then forward as the foot is dorsiflexed. Firm compression of the malleoli by the palms of the hands corrects the widened mortise.

90 degrees (100 degrees in female patients with shortened Achilles' tendon), is the standard method. In undisplaced fractures and those without joint instability, immediate ambulation with weight-bearing is desirable.

If marked swelling is present, the application of a cast can be delayed and the edema reduced by elevation and massage. An alternative is to apply a cast for support, elevate the limb, and change at 7-10 days.

In severe fracture-dislocations, a mid-thigh cast with the knee in 20- to 30-degree flexion is applied; the patient is permitted to be ambulatory on crutches, but weight-bearing is avoided for 6 weeks. A change to a walking cast should be made at this time.

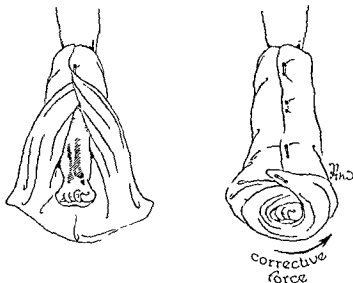


Fig. 679—Pillow splint (Gurd's method)

Fracture-separation of the internal malleolus is probably best treated by open operation and internal fixation by screw or suture. This is also true for displaced fractures of the anterior and posterior tibial articular margins. In the latter, the so-called posterior malleolus, the fracture line after manipulative reduction often persists as a "step" on the weight-bearing surface. Open operative correction is essential in these cases to prevent the development of traumatic arthritis. This is especially true if the fragment constitutes one third or more of the articular surface.

Fixation.—In those cases with a stable ankle mortise, a below-knee cast, with the foot at

Minor fractures require immobilization for 3-4 weeks. Major injuries may require plaster fixation for 8-12 weeks.

Most patients require re-education of the limb musculature and arch support on removal of the cast.

Complications.—

- 1 Traumatic arthritis
- 2 Post-traumatic edema with thickening of ankle
- 3 Recurrent dislocation of the ankle
- 4 Recurrent dislocation of peroneal tendons
- 5 Nonunion of internal malleolar fractures
- 6 Widening of mortise with inferior tibio-fibular diastasis

Calcaneus

Calcaneal fractures constitute the most disabling group of traumatic disorders involving the tarsal bones. The crush type fractures result in prolonged periods of disability, and Creer reports 20% incapacitated for work 3 years after the accident and still totally incapacitated at 7 years. Although fracture of the femoral neck is still regarded as the "unsolved fracture," the author would transfer the focus of attention to the crush fractures of the calcaneus. Understanding of the problems concerned and improvement in the functional recovery are evolving from attempts to analyze the individual types of fracture and the end results achieved by the different methods of treatment applied to the particular pattern of injury. Little progress could be expected while all types of fracture were treated by a routine approach and the end results recorded without correlation as to the type of fracture in question.

Mechanism of Injury.—The usual mechanism is a fall of varying distance onto a surface of varying consistency. Other variable factors are the patient's age, weight, and occupation. As can be readily understood the extent of calcaneal damage will vary considerably. The force may be distributed between the two feet, resulting in bilateral fractures of different degrees. Calcaneal fractures due to falls from heights are associated with compression fractures of the dorsolumbar vertebral bodies in approximately 10% of cases. Isolated fractures may be caused by direct violence or avulsion.

Classification —

Isolated Fractures —

Avulsion of tendo achillis insertion

Beak fracture above tendo achillis insertion

Fracture of sustentaculum tali by direct violence

Vertical fracture of medial tubercle by avulsion or direct violence

Fracture into calcaneocuboid joint by forced inversion

Crush Fractures —

Not involving subtalar joint

Involving subtalar joint

(1) Joint depression type

(2) Tongue fracture type

Clinical Picture.—The patient gives a history of a fall or direct violence to the heel with pain and inability to bear weight on this part.

Examination shows swelling due to edema and hemorrhage. There is acute tenderness on compression.

X-rays should be taken in the standard anteroposterior and lateral planes to include the ankle. Special axial views are required to demonstrate details of the type of fracture.

In the lateral x-ray, the tuber-joint angle should be estimated to see the degree of upward displacement of the posterior fragments. Attention should also be devoted to the degree of involvement of the subastragloid and midtarsal joints.

Fractures of the os calcis may be bilateral and are often associated with compression fractures of the dorsolumbar spine. Examination for this related injury should never be omitted.

Treatment.—The calcaneus is largely composed of an internal structure of cancellous bone with an outer cortical layer whose thickness varies in different parts. This shell, when grossly comminuted, is very much like the problem of Humpty Dumpty. Furthermore, the articular surfaces for the talus are usually deranged in the crush fractures so that a painful post-traumatic arthritis is a frequent sequel. Like cancellous bone elsewhere, e.g., vertebral bodies, a period of 3-4 months is required for healing, and compression by weight-bearing must be avoided to prevent deformity. The routine of incorporating the leg and foot of these patients in plaster casts with or without incorporated spikes or pins for 10-12 weeks is not without complications. Post-traumatic bone atrophy with osteoporosis, circulatory imbalance, atrophy of articular cartilage, and joint stiffness is the all too frequent complication. This is especially true for patients over 50 years of age. As a result of this fact many specialists are tending to treat calcaneal fractures of all patients over 50 as soft part injuries. This treatment includes elevation, protection from weight-bearing, compression bandages, and early massage and movements of all the joints of the ankle and foot. This method is also employed for most

ISOLATED FRACTURES



AVULSION OF TENDO ACHILLIS INSERTION



'BEAK' FRACTURE



FRACTURE OF
SUSTENTACULUM TALI



FRACTURE
OF MEDIAL
TUBERCLE



FRACTURE INTO
CALCANEO-CUBOID
JOINT



CRUSH FRACTURES

INVOLVING
SUBTALOID
JOINT

NOT INVOLVING SUBTALOID JOINT

JOINT
DEPRESSION
FRACTURE

TONGUE-TYPE
FRACTURE



1ST STAGE



MORE ADVANCED



JOINT DEPRESSION
FRACTURE WITH
FRACTURE OF
SUSTENTACULUM TALI



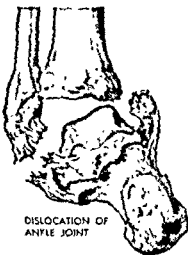
1ST STAGE



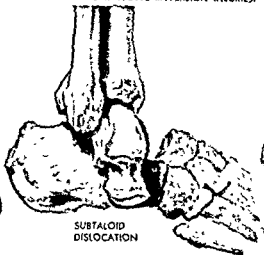
ADVANCED STAGE

L. Netter M.D.
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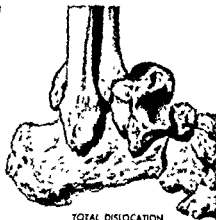
DISLOCATIONS (INVERSION INJURIES)



DISLOCATION OF
ANKLE JOINT



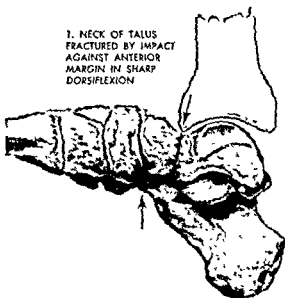
SUBTALOID
DISLOCATION



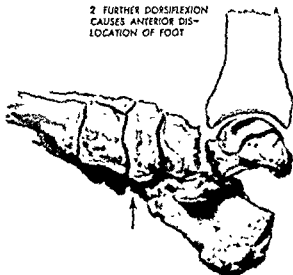
TOTAL DISLOCATION
OF TALUS (ANTERIOR)

FRACTURES (DORSIFLEXION INJURIES)

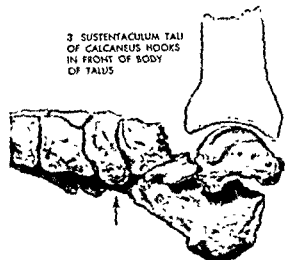
1. NECK OF TALUS
FRACTURED BY IMPACT
AGAINST ANTERIOR
MARGIN IN SHARP
DORSIFLEXION



2. FURTHER DORSIFLEXION
CAUSES ANTERIOR DIS-
LOCATION OF FOOT



3. SUSANTACULUM TALI
OF CALCANEUS HOOKS
IN FRONT OF BODY
OF TALUS



4. WHEN DORSIFLEXION FORCE
CEASES, FOOT PLANTAR FLEXES
AND SLIDES BACKWARD,
CARRYING FRACTURED
BODY OF TALUS OUT OF
TIBIO-FIBULAR MORTISE
AND TWISTING IT

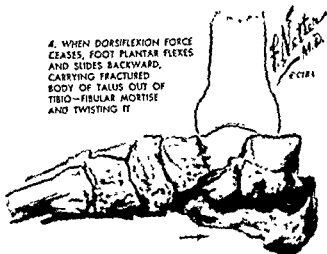


Plate 95.—Fractures and Dislocations of the Talus.

of the isolated fractures except those requiring open operation and internal fixation.

Radical measures are being restricted for the groups with an altered angle of incidence and those involving the subtalar joint in patients under 50 years of age, and this residue comprises 35-40% of any large series of cases.

Palmer and Essex-Lopresti distinguished two types of fracture involving the subtalar joint:

1. Joint depression type
2. Tongue fracture type

These are the two types of calcaneal fractures which so frequently result in total disability. The joint depression type is the more

to work within one year, which is a great advance. An alternate therapy is the use of subtalar or triple arthrodesis in these crush fractures performed 3-6 weeks after the initial injury. Salvage of some of the poor results of crush fractures is also possible, with similar arthrodeses performed 18 months or more after the initial injury. Although there is a definite tendency for continual improvement up to 3 or more years, the average case is stabilized and the result obtained within this period of 18 months.

Complications.—

1. Traumatic arthritis of the subtalar joint. This gives pain when walking, especially on

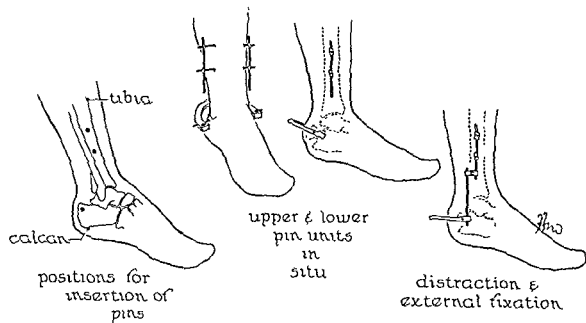


Fig 680—External skeletal fixation for fractured calcaneus

common, and in these cases the whole segment of the posterior joint surface is driven downward and backward. Open operative reduction and fixation by packing the resultant cavity beneath the articular surface with cancellous bone (Palmer) or retention by spike incorporated in plaster (Essex-Lopresti) are being used in these cases with some improvement in the results. In the tongue fracture type reduction by Bohler's two-pin method or Gissane's spike would appear to offer the best solution.

Series of these serious types are being reported, with 80% of the patients returning

irregular ground. If severe, arthrodesis is indicated.

2 Widening of the bone, causing impingement on the lateral malleolus. If symptoms are severe, arthrodesis of the ankle and subtalar joints is required.

3 Upward displacement of the posterior fragments resulting in relative elongation of the tendo achillis. Alteration in the appearance of the calf and ankle will be present with increased range of dorsiflexion.

4 Bony prominences in the sole of the foot which give pain from pressure.

Tarsus

The *talus* is the keystone of the longitudinal arch and constitutes the connecting bone between the leg and foot. Injuries of this bone are uncommon, but because of its essential function, the most efficient treatment is required to secure good results.

Classification.—Watson-Jones has classified injuries of this bone on the basis of the causative mechanism:

1. Inversion injuries*
 - a. Dislocation of the ankle
 - b. Subtalar dislocation
 - c. Total dislocation of the talus
2. Dorsiflexion injuries
 - a. Fracture of the neck of the talus without displacement
 - b. Fracture of the neck with subtalar dislocation
 - c. Fracture of the neck with subtalar dislocation and backward displacement of the body
3. Adduction and abduction injuries of the forefoot
 - a. Midtarsal dislocation with fracture of navicular
 - b. Tarsometatarsal dislocation with fracture of metatarsals

Diagnosis.—In cases of complete dislocation of the talus, diagnosis is easily made. The injury may be compound. Other cases present typical deformities. The exact post-traumatic anatomy is best assessed by careful x-ray examination.

Treatment.—Reduction should be carried out at the earliest moment. In complete dislocation of the talus, especially if compound, open operative reduction is best. The bone should not be removed even if devoid of ligamentous attachments. In dorsiflexion injuries, the ankle must be fixed in full plantar flexion to maintain reduction.

In severe injuries and dislocations, weight-bearing should be avoided, although the patient can be ambulatory on crutches. When aseptic necrosis of the talus is expected, weight-bearing is contraindicated for 4-6 months. The usual case may gradually increase weight-

bearing after 6 weeks. A below-knee cast gives adequate protection.

Other Tarsal Injuries

Direct violence may cause isolated fractures of other tarsal bones, of which the navicular is most commonly involved.

Treatment must be carried out to secure union in satisfactory position, and every effort is directed to preserve the arches of the foot and the muscular balance.

Metatarsal Fractures

The five metatarsals may be fractured at any point in their length. Fractures involving the base of the 5th and necks of the 2nd and 3rd metatarsals are most common.

Base of Fifth Metatarsal

Fracture in this situation is often called "dancer's fracture." The attachment of peroneus brevis is avulsed in sudden inversion of the forefoot. The base may also be fractured by direct violence. Strapping or plaster cast immobilization for 10-21 days suffices.

Shafts of Metatarsals

The shafts may be fractured obliquely by indirect torsion or transversely by direct violence.

Treatment.—If displacement is minimal, a walking cast, with sponge rubber molded to preserve the long arch and concavity of the foot, will prove satisfactory. Crutches should be used for 10-14 days. The cast is required for 4-6 weeks.

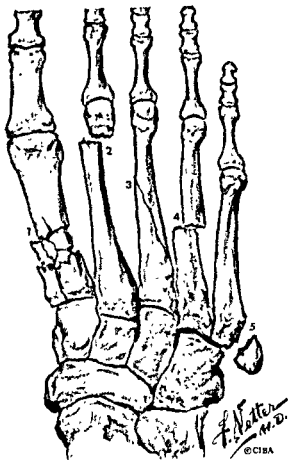
If displaced, traction through the corresponding phalanges should secure reduction. Care must be taken to avoid angulation into the sole of the foot. A metatarsal bar will be helpful when walking begins.

Necks of Metatarsals

The neck of metatarsal 2 or 3 may fracture from the stress of forced walking. This has been termed "march fracture."

Care must be taken to prevent excessive callus formation, which will give prolonged

*These correspond to dislocation at the three joints of the talus.



FRACTURES OF THE METATARSAL BONES
1 COMMINUTED FRACTURE, 2. FRACTURE
OF NECK, 3 OBLIQUE, 4 TRANSVERSE,
5 AVULSION OF TUBEROSITY OF 5TH
METATARSAL



CRUSH INJURY OF GREAT TOE



DORSAL DISLOCATION OF FIRST
METATARSO-PHALANGEAL JOINT

Plate 96—Fractures of Metatarsals; Crush Injury and Dislocation of Great Toe.

Courtesy Moseley, H. F.: CIBA
CLINICAL SYMPOSIA 7: 167, 1955

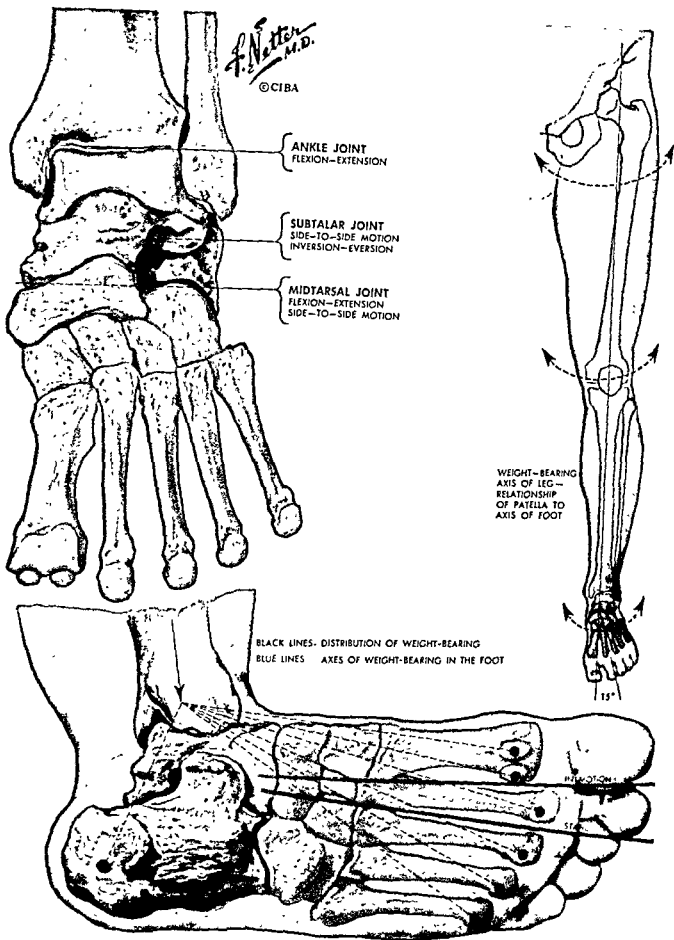


Plate 97.—Motion in the Foot and Distribution of Weight.

sability. This is obviated by early recognition and avoidance of unprotected weight-bearing; using a walking cast and crutches for the first 10-14 days.

Fractured Phalanges

Crushing injuries of the great toe are the most common. These result from direct injury from falling objects such as carried cases, furniture, etc. or by stubbing the toe. The injury is often compound.

Treatment consists of careful cleansing of the part, evacuation of subungual hematoma, and application of an easily removed splint. Elevation and hot baths when the wounds have closed diminish the swelling.

Ambulation should begin with crutches. A metatarsal bar is applied to the shoe, which has been cut out over the dorsum of the affected toe.

Dislocations of the Toes

Dorsal dislocation at the first metatarsophalangeal joint is the common dislocation. It is readily reduced by traction in flexion, with direct pressure. The reduction is stable, and protection from the pressure of weight-bearing is the essential point in treatment. The time required is determined by the disappearance of pain and tenderness. Crutches for 2-3 weeks are the usual requirement. A metatarsal bar should be applied to the shoe.

STATIC DISORDERS OF THE ANKLE AND FOOT

Posture of the Normal Foot

(Plate 97)

The long axis of the foot may be directed inward (in-toeing) up to 15 degrees or outward (out-toeing) up to 25 degrees, or forced up to 90 degrees, and the apparent average posture when standing is a position of out-toeing of 15 degrees. This angle is greatest when a person is stationary, less when walking, and least when running.

The direction of the long axis of the normally postured foot is determined by the rotation of the lower limb at the hip since the talus is firmly held in the ankle mortise. Thus

out-toeing presupposes external rotation and in-toeing, internal rotation at the hip joint. This interrelationship is important in our clinical examination when the foot presents the common static disorder of valgus deformity, and the direction of the patella is taken as our indicator of lower limb rotation.

Distribution of Body Weight

The body weight is transferred through the legs to the two feet, each foot receiving one-half the load. Measurements have shown that the weight is equally divided between the hindfoot and the forefoot as to a tripod. Analysis of the distribution through the forefoot discloses that the two sesamoids under the head of the 1st metatarsal and the heads of the 2nd, 3rd, 4th, and 5th metatarsals each receives an equal share. In this way the ball of the great toe sustains twice the pressure of that under each lateral metatarsal head. This ratio of weight borne by the hindfoot and forefoot is true only for the bare foot, and naturally in shoes the proportion carried by the forefoot increases as the level of the heel is raised.

When standing stationary, the *axis of balance* passes through the center of the heel and forward between the 2nd and 3rd metatarsals. When walking or running, the weight is transferred more medially and the *axis of leverage or propulsion* runs between the 1st and 2nd metatarsals.

Evolution of the Normal Foot

The human foot is a wonderful mechanism evolved in structure and function for stability and locomotion, our understanding of which can be greatly assisted by a brief reference to comparative anatomy.

When we compare the human foot with that of the anthropoid, we note that the human being has largely ceased to employ the foot for grasp, whereas the arboreal life necessitates brachiation. As a result, in the anthropoid, the 1st metatarsal segment is in varus and short, and its associated prehensile hallux is also short and mobile. In comparison, marked changes have occurred in the human foot. The varus position of the 1st metatarsal segment has largely disappeared. The great toe and its metatarsal have increased greatly in size and

become aligned in the long axis of the foot. With this change a great deal of the mobility has been lost, with consequent increase of the stability required for propulsion. The metatarsophalangeal joint of the 1st digit is now situated just distal to that of the 2nd digit.

Further changes include the great increase in the proportion of the tarsal segment, the proportion of the lateral four metatarsals remaining approximately constant while the lateral digital elements have been greatly reduced. The tarsal segment has lost mobility and has become more stable due to the altered structural arrangements of the bones and ligaments at the expense of the intrinsic musculature. The heel has been brought to the ground and an inner longitudinal arch formed to give elasticity to the gait.

The balance of the normal human foot therefore depends on two main factors: (1) the *structural stability* determined by the mechanical arrangements of the component bones and their articulations and associated ligaments and (b) the *postural stability* maintained by the activity of the intrinsic and extrinsic musculature.

Theories on Instability of the Foot (Plates 98 and 99)

Since the stability of the foot depends on the mechanical arrangement of its components and the balanced activity of its musculature, the explanation of static disorders will be found in derangements in these two aspects of the problem. However, with the tendency of the human mind to simplify, there have been two schools of thought, the one trying to explain most disorders by muscular imbalance, the other by structural defects. To the author it would seem that there has tended to be a fixation of thought on muscular imbalance rather like that found in the clouding of understanding of low back disorders by terms such as lumbago and sciatica and of shoulder disorders by terms like bursitis and neuritis. Just as the work of Schmorl on the intervertebral disc and that of Codman and Meyer on the supraspinatus and biceps tendons have brought light to these subjects, so the work of Dudley J. Morton, R. I. Harris, and many others is bringing understanding to the

subject at present under discussion. The search for an underlying demonstrable organic and structural basis for each type of foot disorder will be found to be the key to further advance in our comprehension. It is true that in certain cases of pes valgus the problem is that of muscular insufficiency as present in the overweight individual and in the patient after bed confinement or after plaster immobilization for injury, but by and large it will be found most helpful to regard the muscular imbalance as secondary to structural defect.

Structural Variations of the Human Foot

Probably one of the characteristics of the foot which most confuses the physician when evaluating its disorders is the variability of its size, shape, and functional qualities as noted in different individuals. Even its changes from birth to old age in the same person constitute a clinical study in themselves. In this connection certain points should be mentioned. The shape and size of the foot will tend to vary with the bodily habitus. A long narrow foot will be found in the tall thin man, whereas a shorter, broader foot will be present in the thick-set type. Yet every gradation of shape and size will be found in any general survey of patients.

In addition to this generalization on the structural variations, however, the student should look for the type of foot which occurs in approximately one third of the population, so well described by Dudley J. Morton, and which follows from the above remarks on the comparative anatomy of the foot taken largely from his writings. This investigation has stressed the structural type with the short, hypermobile, and often varus 1st metatarsal segment, associated with a short great toe and proximally placed metatarsophalangeal joint which might be regarded as an evolutionary throwback to more primitive ancestors. These cases present a structural defect which gives a lack of stability of the great toe element in propulsion and stance, with the result that the relatively longer 2nd metatarsal takes a disproportionate load. In time and with excessive use, *accentuated in the female sex by the wearing of high heels*, there is a hyper-

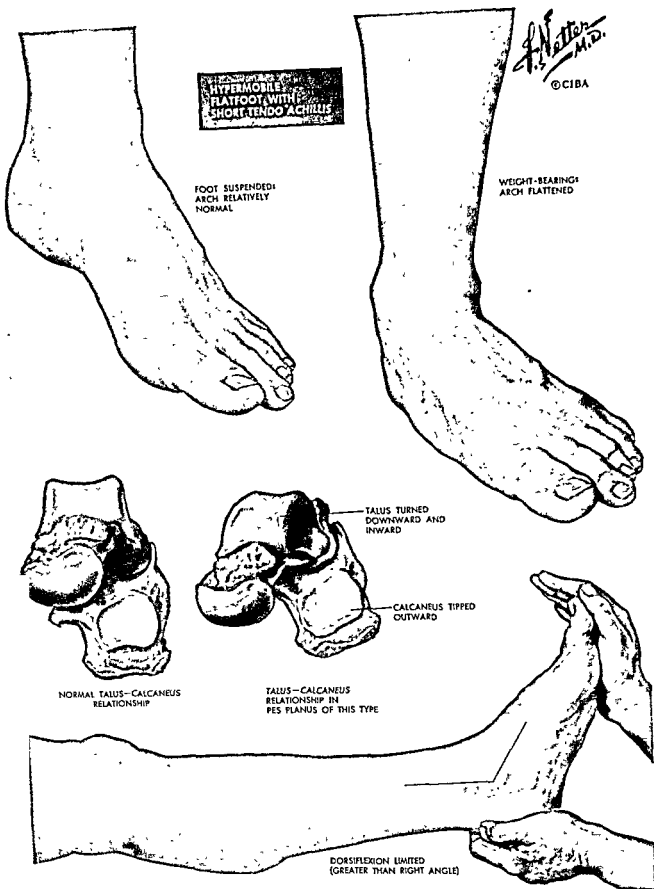


Plate 98.—The Hypermobile Flatfoot.



COMPLETE CALCaneo—TALAR BRIDGE



ALMOST COMPLETE BRIDGE (SYNDESMOSIS)



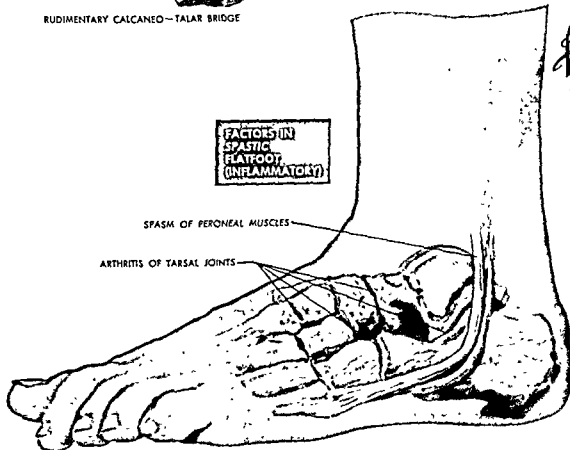
RUDIMENTARY CALCaneo—TALAR BRIDGE

CALCANEAL ANOMALIES
WHICH MAY CAUSE
RIGID OR SPASTIC
FLATFOOT



CALCaneo—NAVICULAR BRIDGE

FACTORS IN
SPASTIC
FLATFOOT
(INFLAMMATORY)



J. Natter
M.D.
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Plate 99.—The Spastic Flatfoot.

trophy of the 2nd metatarsal shaft, indicated by a widening of the bone and thickening of its cortex, an excessive strain on its articulation with the middle cuneiform causing synovitis followed by traumatic arthritis, and often premature degeneration of its articulation with the phalanx (Freiberg's disease). Calluses appear from the excessive intermittent pressure under the 2nd and often the 3rd metatarsal heads and, in the female sex, this is probably the most common static derangement of the forefoot. The excessive mobility of the 1st metatarsal segment and the distribution of weight largely through the 2nd and 3rd segments causes the foot to assume a valgus position, with muscular imbalance a secondary factor.

Following this trend of thought, a further addition to our understanding has come from the investigations of Harris and Beath. A foot survey was made of 3,619 Canadian recruits for World War II. All were males 18-35 years of age. In this investigation special attention was directed to the valgus foot, and they found that the most disabling type was that which they have designated "*the hypermobile flatfoot with short tendo achillis*." This disorder they believe is due to a lack of adequate calcaneal support for the head of the talus. This structural tarsal defect is associated with a shortened tendo achillis.

The clinical history of such cases reveals a foot disability of varying severity commencing in childhood. On examination one notes that on weight-bearing the foot assumes the valgus position of the heel and forefoot, whereas the shape and arch of the foot reform when the body weight is removed. If, with the knee held fully extended, the movement of the foot from plantar to dorsal flexion is tested, it is found that marked restriction in this movement at the ankle is present, stopping usually at 105 degrees and rarely reaching the right angle. However, at the point where ankle movement ceases, hypermobility and instability at the subtalar and midtarsal joints are noted, and further movement into dorsal flexion occurs as the whole foot moves into valgus around the talus. This type of pes valgus constituted 6% of the disabling disorders of the foot in their survey.

The second group presenting serious disability were those included in the designation "*peroneal spastic (rigid) flatfoot*" which accounted for 2%. These cases give a history dating back to adolescence, and in time secondary structural changes produced a rigid valgus foot incapable of sustaining prolonged use. In this type of pes valgus the deformity is largely at the subtalar joint.

Their original investigations have been continued, and they find that a certain percentage of these cases present congenital structural tarsal anomalies.

The first of these defects is the *calcaneotalar bridge* which may be osseous, cartilaginous, or fibrous and unites the posterior aspect of the sustentaculum tali with the talus. This abnormality, first noted at operation, interferes with the normal motion in the subtalar joint and results in an increasing valgus deformity of the heel and a secondary valgus or pronation deformity of the forefoot occurring at the midtarsal joint. In time, degeneration, as disclosed by osteophytic lipping, occurs in the talonavicular joint. The lateral x-ray of the tarsus disclosing this change should suggest the posterior oblique view of the tarsus, which will often reveal the full-blown osseous bridge. It is possible that some cases of talipes calcaneovalgus may be due to this structural defect.

A second abnormality, the *calcaneonavicular bar*, may be found on oblique lateral views of the foot. Again it may be osseous, cartilaginous, or fibrous in nature and in certain cases will lead to a rigid valgus foot presenting for diagnosis and treatment because of pain and deformity.

Accessory Navicular.—One further structural anomaly should be mentioned. An accessory navicular bone is present in 5% of people. It causes an undue prominence on the medial side of the foot and is often referred to as an accessory or secondary medial malleolus. Sometimes it is fused and the prominence is that of an abnormally large tuberosity of the navicular. Its clinical importance is that it is sometimes associated with intractable painful valgus feet in adolescents and also that its undue prominence subjects it to excessive friction and pressure.

These five groups of structural anomalies should be differentiated in cases presenting for examination for the so-called fallen or flattened longitudinal and metatarsal arches. In such cases muscular imbalance appears to be secondary to the structural abnormality.

Nomenclature (Plate 100)

Further discussion will be more readily followed if we now clearly define the various terms in common use.

The generic term *talipes* used in describing the deformities involving the ankle and foot is derived from the Latin words *talus* meaning ankle or ankle bone and *pes* meaning foot. Thus *talipes* should be used when the deformity involves both the ankle and foot, while *pes* is the generic term to be employed when the disorder is limited to the foot alone.

Movements at the ankle joint are chiefly plantar flexion and dorsiflexion. Because through evolution the horse has a foot in marked plantar flexion, the comparative anatomist has given the human foot deformity characterized by plantar flexion the name *equinus* (from *L. equus*, horse). The reverse deformity, characterized by dorsiflexion of the foot and prominence of the heel, is designated *calcaneus*.

The terminology of deformities involving the foot proper is best understood by considering the movements of the remainder of the tarsus and metatarsus as a unit in relation to the talus. A movement of this unit into inversion and adduction (supination) draws the foot toward the midline of the body, which is termed *varus* (*L.*, bent inward), whereas the opposite movement into eversion and abduction (pronation) results in a *valgus* (*L.*, bent outward) deformity.

When the foot is in valgus position, the longitudinal arch appears flattened and is often referred to as *flatfoot*, *pes planus*, or *pes valgus*. On the other hand, a varus deformity with an exaggerated depression (equinus) of the metatarsals increases the concavity of the arch and is termed *pes cavus*.

Combined deformities at the superior, inferior, and anterior articulations of the talus, i.e., of the ankle and foot, are best seen in

the types of congenital clubfoot and can be classified as follows:

Talipes	{	equino	{	varus
				valgus
	{	calcaneo	{	varus
				valgus

The significance of these terms will be more clearly understood as the various disorders are described in greater detail.

Examination

A careful history of the patient's complaints should be taken. General factors such as occupation, the necessity for prolonged standing or walking, carrying heavy weights, exposure to cold or wet environment, and, in the female patient especially, the type of footwear should be noted. Vascular problems demonstrable as ankle edema, varicose veins and their sequelae, and arterial insufficiency are important. The duration and degree of a diabetic derangement require accurate investigation.

The patient should be examined standing and walking with and without shoes, with limbs exposed from the lower thigh down. Deformity such as the valgus heel and forefoot is readily seen. On removal of the shoes and socks, the physician will quickly learn the patient's *care of his feet*. The *nails* will be noted as to method of pedicure, the presence of nutritional changes, ingrowth, deformity, or infection. The *clefts of the toes and soles* will be examined for fungous infection, plantar warts, or other dermatologic disorder. The position of corns and calluses indicates the *pressure points*. If foot-printing facilities are available, such provide valuable records of the shape and pressure points. The same information can be gained by noting the *texture of the skin*, whether soft or calloused, over the pressure areas. Manipulation of the foot will reveal the *mobility of its different parts*. In the peroneal spastic valgus foot, movement into inversion will initiate the spasm, and the rigidity of the valgus deformity will be appreciated. The *pulsations of the dorsalis pedis and posterior tibial arteries* should always be recorded, as also any *neurologic deficit* whether sensory or motor. With experience the student will learn to correlate the clinical history.

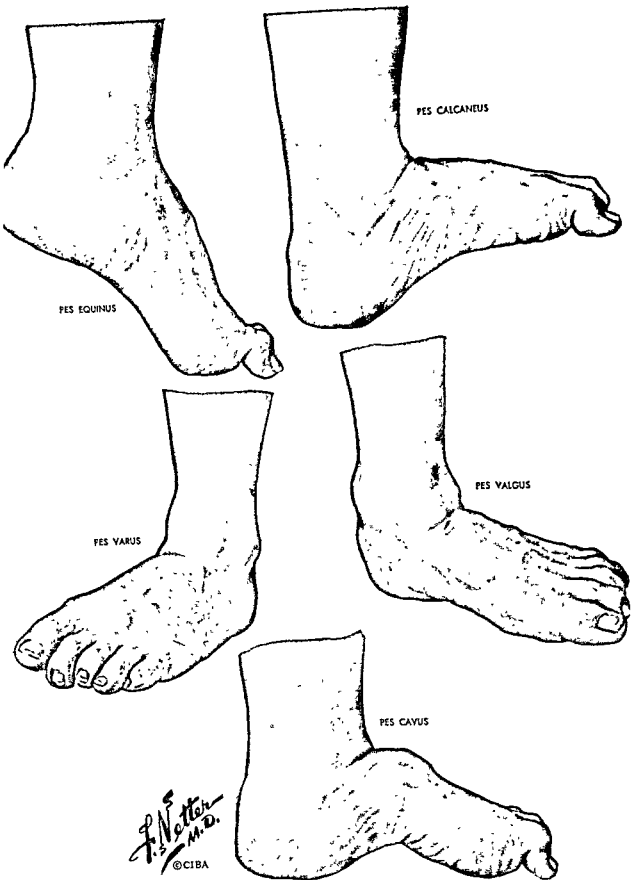


Plate 100.—Foot Deformities.

CONGENITAL
TALIPES EQUINOVARUS

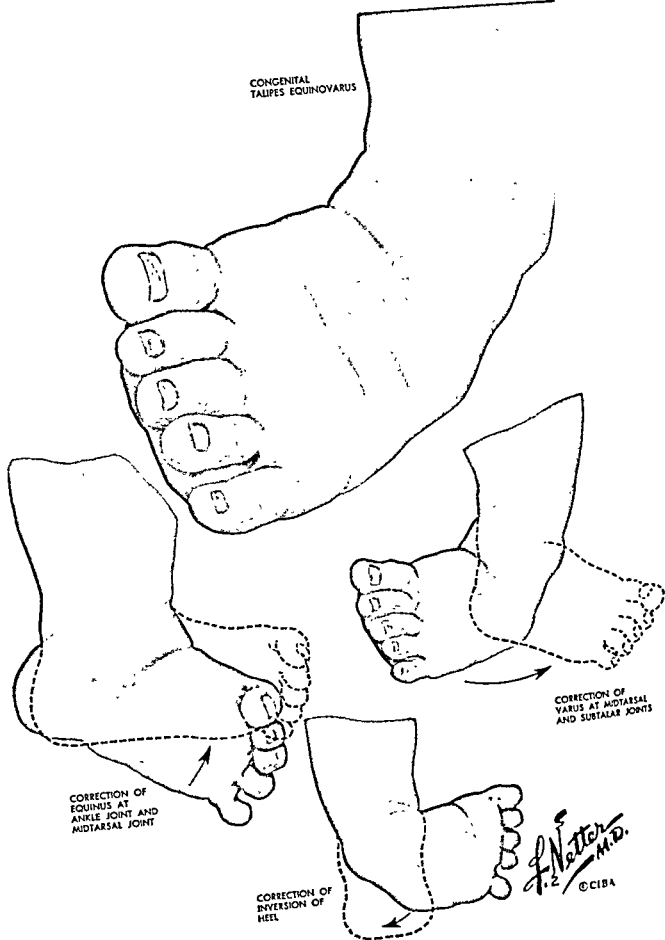


Plate 101.—Talipes Equinovarus.

Courtesy Moreley, H. F. CIBA
CLINICAL SYMPOSIA 9: 83, 1937.

symptoms, and signs in relation to the particular disorder presented by the patient.

Congenital Anomalies

The development of the ankle and foot may be arrested at any stage, and thus a multitude of variants occurs. Talipes, or clubfoot, and congenital structural anomalies already described are the most frequently encountered.

Congenital Talipes Equinovarus (Plate 101)

The word *talipes* is derived from the Latin words, *talus* and *pes*, and is the term used to cover all deformities of the foot centered on the talus. Congenital talipes equinovarus, or clubfoot, is the commonest congenital deformity. It may be unilateral or bilateral, it may occur as the only congenital abnormality or be associated with other stigmas such as cleft palate, spina bifida, arthrogryphosis multi-

plex, etc. There is a tendency for this defect to run in families.

Classification.—

SIMPLE TYPES

Talipes { equinus
 { calcaneus

Pes { varus
 { valgus

COMBINED TYPES

Talipes { equino { varus
 { valgus
 { calcaneo { varus
 { valgus

Etiology.—Various theories as to the causation of this deformity have been postulated. The view most commonly held is that it is due to the persistence of the feet in the plantar flexed and adducted position which is normally present up to the 3rd month of intrauterine life. In recent years experimental work on developing embryos of various animals and birds suggests that congenital defects are developmental arrests which may be due to chemical changes in the fetal environment during the "critical phase" of development. The work of Duraiswami is most interesting in

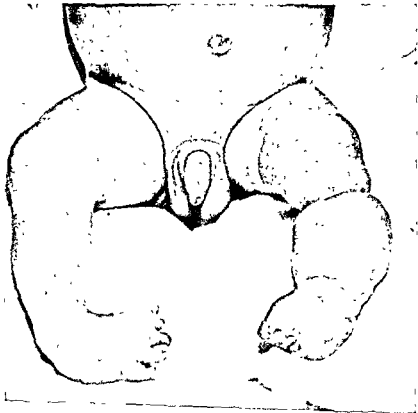


Fig 681.—Multiple congenital deformities of the lower limbs. On the right side, a talipes equinovarus is present together with failure in the differentiation of the great, second, and third toes. On the left side, there is a congenital constricting band just below the knee. The leg below this is of increased size, and there is a failure of differentiation of the toes.

this respect; he has been able to produce most of the typical congenital deformities in chickens by injecting various doses of insulin into the yolk of fertilized eggs. The critical phase of development is different for the various parts of the body. Thus, injection during the first 2 days of incubation caused defects in the vertebral column, on the 3rd day deformities of the feet, and on the 4th and 5th days deformities of the limbs and beak. A further interesting point shown in this research was that once the tendency to deformity had been induced, the deformities recurred in the next generation. From this research it would appear that dietary and hormonal influences during pregnancy may be the causative factors.

Pathologic Anatomy.—Analysis of the deformity shows that it consists of several parts (Plate 101):

1. The foot is plantar flexed. This equinus position takes place at the ankle joint where the talus is angulated plantarward and also at the midtarsal joint where the forefoot is angulated plantarward.

- 2 The varus deformity occurs mainly at the subtalar joint

- 3 An adduction deformity which is chiefly at the midtarsal joint, which draws the forefoot medially. In the infant this deformity is plastic, but as months and years pass, structural changes increase with alteration in the shapes of the bones, in the structure of the ligaments to fix the deformity, and with contractures of the muscles in the position of imbalance. Walking tends to aggravate the deformity. Pressure points occur from shoes and where weight-bearing is most marked. In the severe case the patient walks on the outer border of the foot

Diagnosis.—The clinical picture is so typical that the deformity is usually noted by the medical attendants at birth. When found, the presence of other defects should be suspected. The possibility of a nerve lesion associated with a spina bifida and paralytic type of talipes must be excluded.

Treatment.—The essential points in treatment consist of the earliest possible diagnosis and the immediate institution of corrective

measures. Some orthopedists begin treatment at birth; others wait until the child can be brought for treatment.

At this stage the dynamic splinting devised by Denis Browne is used. The feet are fixed on foot plates which are then attached to a crossbar which maintains external rotation and valgus. The movements of the limb stretch the shortened tissues while developing the muscle power. Full correction is obtained within a few weeks, but splinting is continued in order to prevent recurrence. When the child commences walking, outside wedges are used on the shoes. A similar type of night splint is applied when the child is resting. Clinical review of progress is continued at intervals until the age of 12 years.

The method of dynamic splinting has replaced repeated manipulation and plaster casts to hold correction which were previously in use.

In those patients who have not received the benefit of early diagnosis and treatment, and who present after the age of 2 years, repeated manipulation and plaster fixation can be employed. It is essential to avoid the use of excessive force. With repeated manipulations at intervals of 3 weeks and plaster fixation, resistant cases can be corrected, utilizing the plasticity secured by the osteoporosis of disuse.

In certain of these cases there is a fixed equinus deformity which requires correction by posterior capsulotomy of the ankle joint associated with elongation of tendo achillis by Z-plasty from the medial to lateral side. A further group will require a tibialis anticus transplant to the lateral side of the foot to correct the metatarsus varus deformity. This is usually carried out at 5-10 years of age.

After 11-12 years of age, cases presenting appreciable residual deformities are corrected by triple arthrodesis with suitable wedge resections.

Pes Cavus

This is one of the most disabling deformities of the foot. It is characterized by exaggeration of the long arch. The metatarsal heads, especially the 1st, are drawn proximally and project below the level of the calcaneus. The toes are clawed. In time, contracture of all the



Fig. 682—Twins presenting a marked degree of bilateral equinovarus, showing the result achieved after orthopedic treatment



Fig. 683—The Denis Browne dynamic splinting used on a case of clubfeet. Each foot is first carefully bandaged to a well padded metal plate and is then fixed, as illustrated, to a metal crossbar. The crossbar is bent so as to secure a valgus position of the foot, and the plate is fixed in external rotation. In this way the kicking movements stretch the shortened soft tissues while developing muscular power.

soft tissues occurs, and there is a shortening of the tendo achillis, giving an added equinus deformity.

The deformity can be analyzed into the following components:

1. Inversion at the subtalar joint
2. Possibly a calcaneus deformity
3. Dropping of forefoot (equinus) at mid-tarsal joint level
4. Possibly clawing of all toes

There are various etiologic factors resulting in this deformity:

1. Upper motor neuron lesion, e.g., Friedreich's ataxia
2. Lower motor neuron lesion, e.g., poliomyelitis
3. Congenital defects associated with spina bifida and cauda equina lesions
4. Muscular dystrophy of peroneal type
5. Vascular ischemia, e.g., following fractures of the upper end of tibia, giving Volkmann's type of contracture

The above group accounts for only about 20% of cases. The vast majority are of unknown cause and are classified as idiopathic.

Clinical Picture.—The patient may present any degree of severity of the deformity. In the young, considerable mobility may still persist, but, with years, fixed deformity of the bones and joints occurs, with marked soft tissue contractures. The metatarsophalangeal joints may be dorsally dislocated. Severe, painful, and disabling calluses may be present.

Treatment.—Therapeutic measures will be determined by the patient's age, the severity of the deformity and disability, and the underlying mechanism.

Conservative measures include the use of special orthopedic shoes, manipulation, wrenching, splinting, and physiotherapy. *Radical treatment* includes section and advancement of the plantar soft tissues from the calcaneus to the plantar (Steindler), capsulotomies of the metatarsophalangeal joints with flexor-extensor transplants or arthrodesis of all interphalangeal joints to correct the clawing of the toes, wedge tarsectomies in late cases and even below-knee amputations may be required.

Some Remarks on Flatfoot

It is probable that the concept of flattened arches has done more to confuse the diagnosis

and treatment of static disorders of the foot than anything else. For this reason this exposition has stressed the structural variations of the foot and the delineation of the clinical entities that are readily demonstrable by the practitioner.

Many individuals have a foot of normal structure, but presenting a low arch, which is completely adequate for their work load. It is only when function is impaired and symptoms develop that a clinical problem arises. This may be in the way of acute or chronic foot strain from excessive use. *Acute foot strain* was occasionally seen in the days before the early ambulatory treatment of the hospitalized medical or surgical patient. After prolonged confinement to bed, muscular insufficiency was present, and ligamentous strain gave rise to pain and disability. Such is also noted after prolonged plaster immobilization of the lower limb for traumatic or orthopedic disabilities, after rapid gain of weight, and when the patient is subjected to unaccustomed prolonged standing, walking, or carrying of heavy loads. The treatment of such disorder of the foot must be directed to rest from the unaccustomed weight load and to graduated development of muscular strength by active resistance exercises.

Similarly, certain normal feet may become involved due to systemic disease such as gonorrhea, rheumatoid arthritis, and specific fevers. Some cases of peroneal spastic flatfoot may result from tarsal arthritis due to an infective agent. Such problems must be dealt with on the basis of the systemic disease and the foot protected during its activity.

Apart from the above situations one should concentrate on recognizing the syndromes with definite structural anomalies, which have been carefully delineated above.

Treatment of Hypermobile Flatfoot with Shortened Tendo Achillis.—The clinical features of this disorder together with the variability of its severity as to deformity and symptoms have been previously mentioned.

The treatment will be determined by the age of the patient and the severity of the clinical symptoms. Harris recommends conservative measures for all patients up to late adolescence or young adult life. *Palliative therapy* includes raising the heel of the shoe or boot, additional

inside wedges to the heels and soles, Whitman's plates, and exercises. *Operative procedures* should be restricted to the severe cases and should not be performed before the age of 12 years. Arthrodesis of the talonavicular and subtalar joints through a medial incision is recommended.

Treatment of Peroneal Spastic (Rigid) Flatfoot.—Here again the changing concepts of the underlying mechanisms of this group of disorders will tend to modify the principles of treatment. We have seen that some of these cases presumably have an infective basis. Such patients are best treated by manipulative correction, under anesthesia if necessary, and

ence on will be found necessary. Resection of the bar and subtalar and talonavicular fusion with correction of the deformity are advocated.

Similarly, in the problem of the talocalcaneal bridge with severe symptoms, fusion of the talonavicular joint and the subtalar joint when the osseous bridge is incomplete should afford the best solution.

Disorders of the Hindfoot (Plate 102)

Pain in the Region of the Heel

Lesions of the Calcaneus.—This bone is subject to the various disorders affecting any



Fig. 684—Hypermobile flatfeet with short tendo achillis

rest in plaster in the corrected position for 6-8 weeks. If on removal of the cast the spasm of the peroneals and extensors returns, a further period in the cast should be tried. The patient is ambulatory on crutches but without weight-bearing.

In cases presenting the calcaneonavicular bar the severity of the symptoms and signs is also variable. Some patients with complete osseous bar are without symptoms. When the symptoms are severe and conservative measures have failed, operative measures from late adoles-

cence on will be found necessary. Resection of the bar and subtalar and talonavicular fusion with correction of the deformity are advocated.

Similarly, in the problem of the talocalcaneal bridge with severe symptoms, fusion of the talonavicular joint and the subtalar joint when the osseous bridge is incomplete should afford the best solution.

Apophysitis (Osgood's or Sever's Disease).—Osteochondritis affecting the epiphysis

of the calcaneus occurs in children in the age range 8-15 years. There is complaint of pain in the posterior part of the heel which may have followed a strain or blow. The discomfort runs a self-limiting course of 3-6 months.

The posterior surface of the calcaneus is tender and slightly swollen on examination. Radiology discloses the epiphysis to be of increased density, with appearance of fragmentation as seen in Perthes' disease of the hip.

Treatment consists of raising the heel of the shoe $\frac{3}{16}$ " If this does not suffice to relieve the discomfort, the foot is placed in 10- to 20-degree equinus in a below-knee walking cast for 4-6 weeks.

Subcutaneous Bursitis.—The friction and intermittent pressure of the shoe may irritate the subcutaneous bursa over the posterior prominence of the heel.

In the acute stage protection from friction is required. In the presence of infection, hot bathing, surgical dressing, and sometimes antibiotics are indicated. Recurrence is prevented by properly fitted shoes with a heel protector.

Retrocaneal Bursitis (Haglund's Disease).—In certain patients there is a prominence of the posterolateral angle of the calcaneus. This is especially well seen on dorsiflexion of the foot, and the soft tissues over the prominence may be reddened and the related bursa inflamed. Radiologic examination does not usually disclose any marked bony projection.

Treatment necessitates protection by heel pads. If the condition becomes a nuisance, resection of the underlying bony prominence can be performed.

Achillodynia (Tenosynovitis of the Achilles Tendon).—Inflammation of the synovial-like tissue around the tendo achillis frequently results from overuse or may be of a rheumatic or gouty nature (See page 1187).

Plantar Fasciitis (Calcaneal Spur).—Discomfort, tenderness on pressure, and pain on walking related to the anteromedial and middle-plantar aspects of the calcaneus in the middle-aged male patient constitute a common dis-

order. It is most frequently seen in overweight men with tendency to gout, osteoarthritis, and degenerative vascular disease. The onset may follow a bruise of the heel but is usually insidious and without known cause. The patient may find it almost unbearable to place the weight through the foot in the morning on rising and will massage the heel before attempting to do so.

Clinical examination localizes the tenderness at the attachment of the plantar fascia, especially on the medial side. In more severe cases the whole calcaneus and subtalar joint areas may be tender on pressure.

A lateral x-ray film may show a large spur and sometimes a calcified deposit as seen in the shoulder and elbow areas.

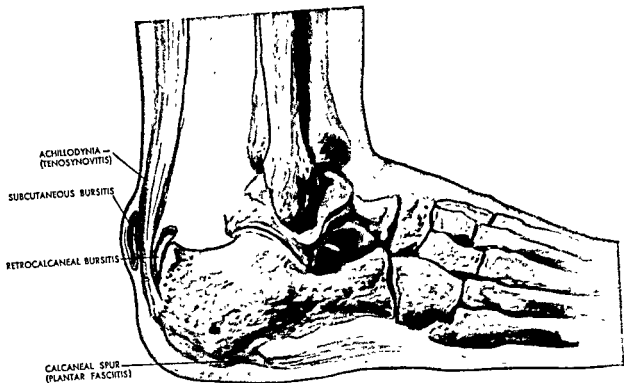
Treatment.—The disorder may respond rapidly to protection by wearing a heavy wool sock, applying a rubber pad with a hole cut out over the tender point, and using rubber heels on the shoes. In more refractory cases, deep kneading massage, diathermy, or ultrasonic therapy may help. Injection of procaine and hydrocortisone (25 mg) has sometimes produced dramatic results. Meticorten and salicylates by mouth have been the most useful drugs.

The symptoms and signs in no way appear to be related to the size of the bony spur, and removal of the spur should rarely be advised. Relief from such procedures probably results from section of the plantar fascia and the resulting hyperemic reaction.

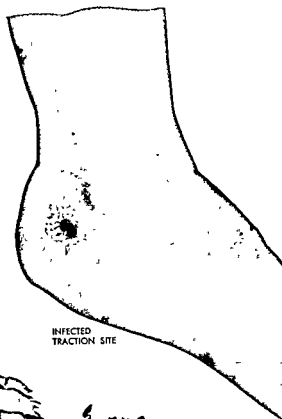
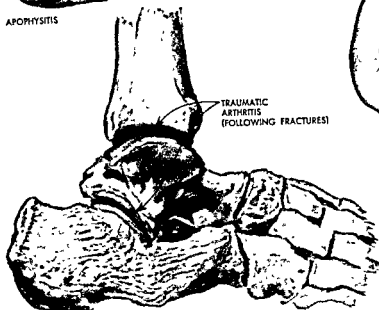
Köhler's Disease of the Tarsal Navicular

Pain and tenderness on the medial side of the foot is found in children in the age range of 8-15 years. This is due to an osteochondritic process involving the navicular bone. The area may be tender on pressure, and radiologic examination discloses the typical changes of increased density with areas of rarefaction to which the descriptive term *fragmentation* is given. The bone appears narrower than the normal as seen in the x-ray of the opposite foot.

The process runs a self-limited course and treatment is rarely required. A period in a plaster cast may be arranged if the complaint of pain is marked.



APOPHYSITIS



INFECTED
TRACTION SITE

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Plate 102.—Disorders of the Heel.

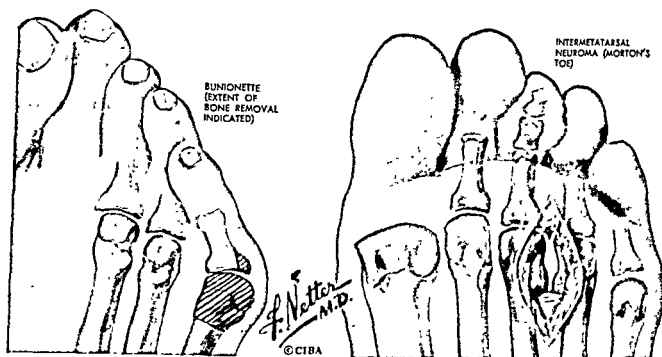
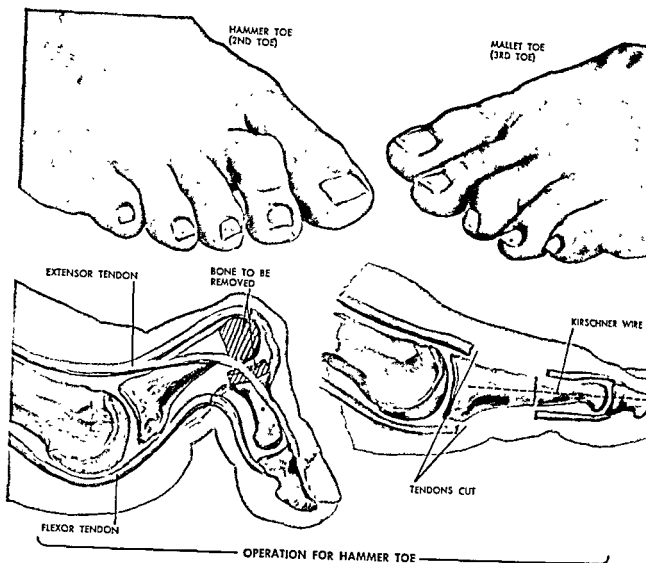


Plate 103.—Hammer toe, Bunionette, Morton's Toe.

Disorders of the Forefoot

Metatarsalgia (Fallen Metatarsal Arch)

Flattening of the transverse arch at the level of the ball of the foot, with resultant pain, designated *metatarsalgia*, is the most common complaint in the middle-aged female patient. Morton's explanation of this syndrome has already been given. Others explain the causation as follows.

Normally the muscular balance of the foot arranges that the weight is carried from the heel to the outer border of the foot, thence to

The patient complains of the increasing pain in the forefoot, often localized to the area of the callus, but frequently radiating up the muscles of the leg. The metatarsalgia is aggravated by wearing shoes, and the complaints are of increasing difficulty in securing a satisfactory fitting.

Treatment is directed to minimizing weight-bearing in shoes during the acute phase. A metatarsal pad is placed behind the metatarsal heads, with elastic strapping of the forefoot, or Morton's compensating insole is prescribed. The patient is given faradic foot baths



Fig 685—X-ray of Kohler's disease of left navicular with normal right navicular for comparison

the ball of the great toe, propulsion forward then being given by the plantar flexion of the proximal phalanx of the hallux. In dysfunction of the interosseous and lumbrical muscles the forefoot splays and the proximal phalanges of the four lateral toes are dorsiflexed. The 2nd, 3rd, and 4th metatarsal heads are depressed, and in time the soft tissues over the plantar aspect of these joints produce a convex surface which, with excessive use, becomes calloused and painful. High-heeled shoes aggravate the disorder.

to activate the small intrinsic muscles, the joints are mobilized by manipulation, and she is taught active exercises to plantar flex the proximal phalanges of the 2nd, 3rd, 4th, and 5th toes, together with rising on the toes with the foot inverted.

Osteochondritis of the Second Metatarsal Head

In certain cases, pain and discomfort are localized to the metatarsophalangeal joint of the 2nd toe. The syndrome begins in late

adolescence, and the patient may seek advice at any time thereafter. A callus may be present under this area.

Clinical examination usually reveals the Morton type foot with the long 2nd toe. The joint is tender on compression between the index and thumb, and plantar flexion of the digit produces pain.

Radiologic examination discloses changes in the metatarsal head, varying with the stage of the disease. The articular cartilage is first involved and the process goes on to a flattening of the head and gradual destruction of the

Syndrome of T. G. Morton's Toe (Plate 103)

In certain cases the metatarsalgia involves chiefly the lateral part of the forefoot. The pain is of a spasmodic type, burning in character and radiating up the adjacent sides of the 3rd and 4th toes. This pain is so severe that the patient often must remove the shoe on the street and massage the foot. A history of such incidents, with relief of pain by shoe removal, affords the clinical diagnosis. It is now known that the cause is a neuroma formed on the digital nerve just proximal to the head

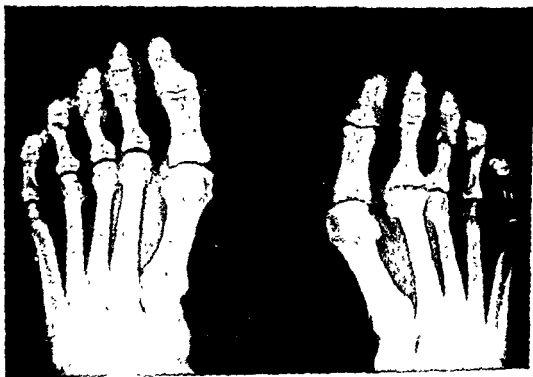


Fig. 686 --Anteroposterior view of forefoot, right and left sides. Morton's type foot with short varus first metatarsal. Note degenerative arthritis of right 2nd metatarsophalangeal joint (Freiberg's disease).

joint. There is usually a thickening of the metatarsal shaft, and many believe the etiologic explanation is a stress-strain hypertrophy of this metatarsal bone with premature degeneration of the joint.

Treatment may be reserved to conservative measures, employing Morton's compensating insole with attention to well-fitted Oxford shoe with Cuban heel. When the symptoms warrant surgical intervention, resection of the metatarsal head with $\frac{1}{4}$ " of the neck gives satisfactory results.

of the metatarsal. The etiologic factor is probably friction and pressure between the metatarsal heads and ligaments. Degenerative changes are also noted in the digital vessels.

This disorder is cured by resection of the neuroma through a dorsal or plantar incision.

Summary

In diagnosing disorders of the forefoot the student must differentiate between splaying of the forefoot with metatarsalgia due to excessive use of the Morton type foot, osteochondritis

drifts of the 2nd and occasionally of the 3rd or 4th metatarsal head, neuroma of the digital nerve producing the syndrome of Morton's toe, callus formation and plantar warts under the ball of the foot, and finally march fractures of the 2nd, 3rd, or 4th metatarsal neck.

Disorders of the Great Toe (Plate 104)

Hallux Valgus

This is the commonest disorder of orthopedic significance involving the hallux. The long axis of the great toe is deviated laterally and rotated internally in variable degrees, even

Anatomic Changes.—It is of fundamental importance in our understanding of the causation and especially of the treatment of hallux valgus to recognize that it is not an isolated derangement of the great toe but rather a part of a group of lesions involving the whole forefoot and finally the foot as a whole and the musculature of the leg.

Because of the forcing of the forefoot into the narrow wedge-shaped toe end of the shoe in walking, the great toe is deviated laterally and the remaining toes medially toward the long axis of the shoe. As the sole of the shoe is more rigid than the upper, this latter element stretches and the 1st and 5th metatarsals



Fig. 687.—Hallux valgus. Dorsal and plantar views of the forefoot, showing the deformity from the wearing of shoes. Note the flattening of the transverse arch and the crowding of the toes and the pressure callosities.

to the point of lying transversely on the dorsal surface of the other toes with the nail facing dorsally.

In some cases the etiologic factor is a congenital metatarsus primus varus, representing a throwback to the prehensile hallux. In most instances the deformity is acquired and is due to the mechanical factors resulting from improper shoes, such shoes are either too short, have too narrow a sole in the forefoot with a wedge-shaped toe-piece, or have too high heels. As these defects predominate in the female footwear, hallux valgus is 15-20 times more common in this sex

and toes are forced dorsally, while the 2nd, 3rd, and 4th metatarsals are displaced plantarward, giving the convex appearance to the ball of the forefoot with the usual designation, fallen or flattened metatarsal arch. Calluses under the 2nd and 3rd metatarsal heads follow in time. Further, the reverse of the hallux valgus deformity, namely, the *bunionette*, occurs in relation to the 5th toe.

As inadequate length of shoe exists, the toes must flex, and this flexion is accentuated by the muscular imbalance resulting from the high heels with shortening of the tendo achillis. Excessive dorsiflexion of the proximal

phalanx leads to clawing of the toes, and dislocation of the metatarsophalangeal joint of the 2nd toe is especially common.

Careful studies of the anatomic changes of the great toe have been made, and it has been found that the ligamentous change has been a gradual stretching of the ligament, binding the medial sesamoid to the 1st metatarsal head. As the great toe is displaced laterally, the sesamoids with the flexor digitorum longus remain fixed in relation to the 2nd metatarsal by the deep transverse ligaments. The dorsal and medial displacement of the 1st metatarsal opens up the first intermetatarsal space, and the sesamoids in relation to the metatarsophalangeal joint are seen to be abnormally lateral in position on the anteroposterior radiograph. The capsule is shortened on the lateral and lengthened on the medial aspect.

The lateral deviation of the 1st phalanx uncovers part of the articular cartilage of the metatarsal head. With irritation from the shoe, the angular prominence of the joint with the overlying bursa becomes inflamed, producing the characteristic *bunion*. There is no evidence that an actual proliferation of bone producing an exostosis occurs. The deformity, once initiated, is aggravated by the bowstring effect of the extensor hallucis longus and flexor hallucis longus. The corrective effect of the abductor hallucis is lost, with the plantar and lateral displacement caused by the rotation and deviation of its insertion on the proximal phalanx.

With the increasing structural changes the fitting of the shoe becomes more difficult, and there is a progressive insufficiency of the intrinsic musculature of the foot evidenced by generalized foot and leg pain.

Symptoms.—The female patient usually complains of pain in relation to the bunion and inability to carry on with her housework. Walking any distance is difficult. Suppuration may develop in relation to the bursa. Some are concerned about the cosmetic appearance, the difficulty of getting a properly fitted shoe, or the impossibility of wearing the style of shoe directed by fashion for social activities. In most cases the hallus valgus and bunion are part of a complex with as many facets for the orthopedic surgeon as facial rejuvenation presents for the plastic surgeon. It is often equally

difficult to satisfy the patients presenting these analogous problems.

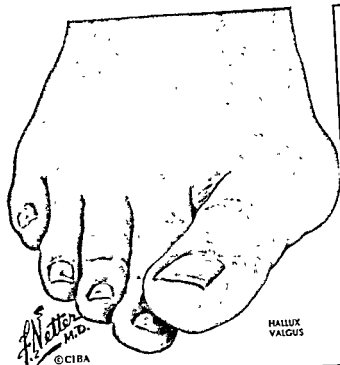
Treatment.—The problem of correcting the metatarsus varus and hallux valgus in the adolescent is most difficult. Conservative measures include the reduction of overweight, exercises, the use of loosely fitting socks, and the fitting of shoes of adequate length and width with straight medial edges and low heels. With progressive changes, soft tissue operations have been employed. The most popular of these is that of McBride, in which the prominence of bone on the medial aspect of the joint is resected and the capsular ligaments shortened; through a second incision the lateral sesamoid is removed and the adductor tendon shortened and resutured to the neck of the 1st metatarsal. If the interspace is still excessive, strong chromic catgut sutures are used to encircle the 1st and 2nd metatarsals to hold them in closer approximation.

When the metatarsus varus predominates, attempts have been made to arrest the epiphyseal growth on the lateral side of the base of the 1st metatarsal. In the older patients, wedge resection and arthrodesis of the joint between the 1st cuneiform and metatarsal base have been advocated (Lapidus). Joplin has utilized a tendon transplant of the long extensor tendon of the 5th toe across the plantar surface of the metatarsals and through the 1st metatarsal neck. The tendon is fixed to the capsular tissues of the 5th and 1st toes after compressing the forefoot.

Following are the three most popular procedures for the adult female patient:

- 1 Simple resection of the prominence of bone on the medial side of the joint with the thickened bursa
- 2 The Keller procedure
- 3 The Mayo procedure

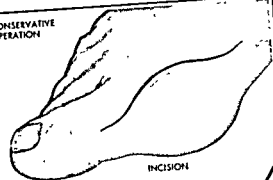
Before the patient is advised operative treatment, the surgeon should carefully evaluate the general and local factors. The patient's personality should be assessed, and an effort should be made to ascertain whether she will cooperate in postoperative exercises and in wearing sensible shoes. There is nothing more unsatisfactory than trying to satisfy the large, overweight female with out-sized deformed feet who for years has been crucifying her



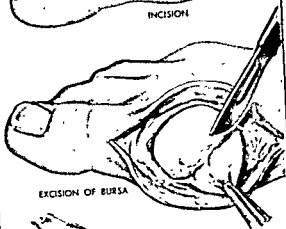
HALLUX VALGUS

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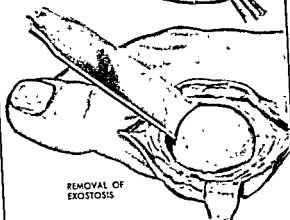
CONSERVATIVE OPERATION



INCISION

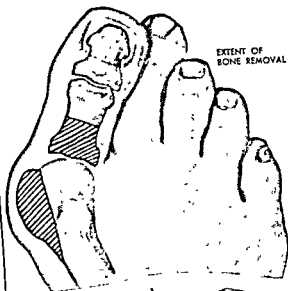


EXCISION OF BURSA

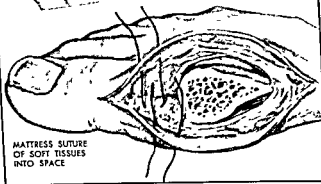


REMOVAL OF EXOSTOSIS

KELLER'S OPERATION



EXTENT OF BONE REMOVAL



MATTRESS SUTURE OF SOFT TISSUES INTO SPACE

MAYO'S OPERATION



Plate 104.—Operations for Hallux Valgus.

appendages in the shoes of fashion. A telecast of the problems of the shoe salesmen with such patients would be a valuable propaganda weapon for better foot health.

The surgeon must also decide whether the cosmetic factor is predominant in the patient's mind or whether pain and suffering due to structural changes necessitate relief.

In the selection of the procedure to be followed, the circulatory status of the limb must be evaluated. Operative measures must be avoided in any case of arterial insufficiency, and, generally speaking, conservative measures should be used in patients over 60 years of age. When relief from pain is necessary, the older patient should be restricted to resection of the prominence of bone and thickened soft tissues.

For the cases in which more radical measures are indicated, and after a period of conservative therapy to mobilize and strengthen the feet, the Keller and Mayo operations are best.

The *Keller procedure* consists of the removal of the prominence of the metatarsal head together with the proximal one half to two thirds of the proximal phalanx. The soft tissues are sutured to assist in maintaining the corrected position.

In the *Mayo operation*, the head with a short length of the neck of the metatarsal is removed together with the prominence of the base of the 1st phalanx.

In both procedures it is sometimes of value to lengthen the extensor hallucis longus tendon. Where necessary, associated problems such as hammertoes and corns are treated at the same time. If both feet are involved, it is best to shorten the great toes an equal length to give a symmetric appearance.

The postoperative treatment consists of maintenance by strapping or plaster cast in the corrected position for 10-14 days. Following this, a careful program of strengthening exercises should be given for several months. At first moccasin slippers only are permitted. As the operative areas become less sensitive, shoes of proper width and with a Cuban height heel can be worn for increasing periods. A toe post may afford a certain support and comfort in the early period of weight-bearing in shoes.

Satisfactory results can be expected in properly selected cases when the operative procedure is combined with adequate preoperative and postoperative care.

Hallux Rigidus

This disorder is characterized by degenerative arthritis of the metatarsophalangeal joint. It occurs chiefly in the male sex and begins in adolescence. An extra long great toe, stubbing, and hyperextension injuries are predisposing factors.

The onset may be sudden, and the pathologic process appears to be an acute traumatic synovitis. If the joint is not protected the process is progressive, and loss of the dorsal area of the articular cartilage on the base of the phalanx ensues. A characteristic gait and wearing of the shoes have been noted. The gait develops to protect the sensitive joint, and the body weight is borne on the lateral aspect of the heel, the ball of the 5th toe, and directly to the proximal phalanx of the great toe instead of to the ball of the great toe. This produces a wearing of the outer side of the shoe heel, the outer side of the sole, and the area of the sole under the pulp of the great toe which is used for propulsion. There is an outward bulge of the upper on the outer side of the heel and forefoot, with convex deformity of the sole of the shoe from side to side.

Restriction of joint movements develops at first in dorsiflexion and finally in all directions. The joint in time takes up a position with some plantar flexion.

Clinical Picture.—The patient presents himself because of pain in one or both great toe joints. When only one is involved it is usually the one more utilized in propulsion.

In the acute phase the joint is swollen and tender and these features are most marked on the dorsal aspect. In time a thickened bursa may develop over this surface and is called a *dorsal bunion*. Movements, especially dorsal flexion, are painful and limited.

In the chronic stage there is a fixed plantar flexion of 10-15 degrees, and the range of plantar flexion is restricted. X-rays show little change in the early case, but the later changes include loss of joint space and osteophytic proliferation at the joint periphery.

Treatment.—The acute phase is best treated by rest to the joint. Ambulation on crutches is recommended. When the acute pain has subsided, a metatarsal type bar placed at the level of the metatarsophalangeal joints, instead of behind this line as for metatarsalgia, provides a rocker action protection. A shoe of adequate length and width must be worn.

The chronic painful stage can be treated by the Keller procedure or by arthrodesis of the joint. Both operations relieve the pain and permit satisfactory function.

Sesamoids

The two sesamoids under the great toe are important anatomic structures of the ball of the foot.

Congenital variations include *bipartite sesamoids* which must be differentiated from fracture. The history of a direct injury followed by local swelling, bruising, and tenderness will assist the clinician. X-rays should be taken of both feet. Congenital lesions are usually bilateral, and the line of division is usually smooth when compared with the irregular fissuring found in fractures.

Irritation of the sesamoids can occur from contusion or intermittent pressure in feet with a poor soft tissue covering. The best treatment of *sesamoiditis* is by pads arranged to diminish the pressure on the sensitive bones. Excision is rarely necessary.

Gout

The great toe is the site of election for this metabolic derangement, and patients are seen presenting the clinical picture of the acute, subacute, and chronic stages. Gout is included here because of its frequency and also to remind the physician to consider it as an etiologic factor in painful disorders of the great toe joint.

A history of recurrent attacks of pain related to this area with complete freedom from discomfort in the intervening periods is characteristic of the early stages.

Excruciating pain, reddening of the medial and dorsal aspects of the joint spreading over the dorsum of the foot, and extreme discomfort on walking are additional signs.

In the chronic stages, radiographic evidence is available in the bitten-out appearance of the head of the 1st metatarsal. Tophi in ears and involvement of other joints may be noted. (See Fig. 565.)

Treatment.—The acute attack is best treated by rest from work and weight-bearing. The foot is elevated and cooling lotions applied. *Heat aggravates whereas cold relieves the pain.* A light diet with the addition of vitamin capsules is prescribed, and fluids are restricted. Colchicine is specific and 0.5 mg. is given hourly (usually 8-10 doses) until diarrhea occurs. *Mistura comphorae composita* (opiate) is given to diminish the intense discomfort. Intravenous injection of 3 mg. colchicine is dramatic. Butazolidin 800-1000 mg. daily is also valuable.

The chronic case must be advised a diet low in fat and red meat. Alcoholic beverages are prohibited. Salicylates and a maintenance dose of colchicine are beneficial. Moderation in drinking, exercise, and sexual activities will be found the best way of life.

Disorders of the Toes Other Than the Hallux

Clawing of the toes is the most common disorder. It should be remembered that muscular balance of the toes is the same as for the fingers, and that clawing of the toes is a result of imbalance of the short intrinsic musculature.

The extensor digitorum longus supplements the action of the extensor digitorum brevis acting chiefly on the proximal phalanges of the toes, producing dorsiflexion. The flexor digitorum longus and flexor digitorum brevis act on the distal phalanges, producing flexion. The interossei and lumbricals insert into the dorsal expansion and produce flexion of the proximal phalanges and extension of the distal phalanges as in the fingers.

The usual claw deformity of these toes consists of hyperextension or dorsiflexion of the proximal phalanx and flexion of the distal phalanges. This position is produced by overactivity of the long and short extensors, reciprocal inhibition of the flexors, and weakness of the interossei and lumbricals. The high heel element of the female shoe is

obvious cause of such a deformity. Calluses develop on the prominences of the interphalangeal joints from the intermittent pressure of the shoe.

Hammer toe and Mallet Toe
(Plate 103)

When the clawing is confined to one toe, the designations above are used. This most frequently involves the 2nd toe. *Hammer toe*

consists of marked dorsiflexion of the proximal phalanx with plantar flexion of the 2nd phalanx. The distal phalanx may be hyperextended, neutral, or hyperflexed. The condition may be familial and unilateral or bilateral. When the deformity consists only of a marked plantar flexion of the distal phalanx, the name *mallet toe* is given.

Such deformities may be symptomless, or the patient may seek assistance because of pain

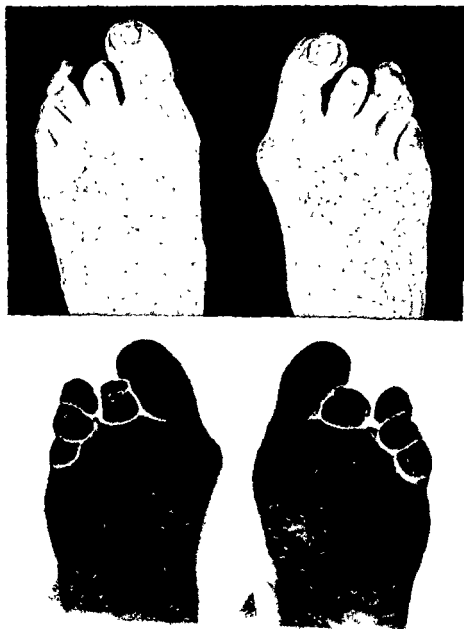


Fig 688—Bilateral hammer toes involving the 2nd toe. Note also the pressure points over the interphalangeal joints.

from callus or corn formation, pressure on the nail, or for cosmetic reasons.

In children, strapping may be of some help, but supervision for many months by the parents is essential to produce lasting results.

When treatment is desirable in the adult, arthrodesis of the proximal phalangeal joint after correction of the deformities by tenotomy and capsulotomy gives excellent results. The straight position of the toe is maintained for 4 weeks by a fine Kirschner wire, which is then removed under local anesthetic as an office procedure.

Bunionette

The *bunionette* for the 5th metatarsophalangeal joint is the counterpart of the *bunion* for the great toe. It consists of a bursal enlargement over the angular projection at this joint.

Conservative measures of treatment comprise correction of faulty shoes and relief from pressure by application of protective pads.

When the deformity is cosmetically unsightly and pain is severe, removal of the prominence of the head of the metatarsal and phalangeal base may be considered.

Overlapping Fifth Toe

(Plate 105)

This is a congenital lesion involving the 5th toe which shows a tendency for adduction of the digit at the metatarsophalangeal joint. This may develop into a transverse dorsal displacement of the toe, interfering with the proper fit of the shoe.

Correction in the very young can be obtained by the Budin sling. However, this often fails, and operative correction by extensor tenotomy, capsulotomy, and plication of the lateral capsule can be used. Some adult patients prefer excision of the proximal phalanx or amputation of the digit, especially when associated with a persistent soft corn.

Disorders of the Nails

The toenails present a considerable number of problems to the practitioner and advice is frequently sought because of their improper

care. The soft, irregularly shaped nails of the aged disclose the nutritional changes occurring in arterial insufficiency. Gangrene of the limb can develop from misdirected attempts to pare such nails or from neglect of associated infection.

Ingrowing Toenails

(Plate 106)

This common disorder most frequently involves the nail of the great toe. Tight shoes, shrunken socks, and improper paring of the corners are the common etiologic factors. In some patients malformations of the nail resulting from trauma or of a congenital origin constitute the predisposing cause.

Plate 106 shows the proper way in which the nail should be pared. The corner on each side should project beyond the flesh. If the corner is cut off, the flesh is gradually pushed in by the pressure of the other toes, the socks, or the shoe. With growth, the nail is too wide for the space, and ulceration or cutting of the flesh by the sharp corner occurs. Pain from this pressure of flesh against the sharp nail edge develops, and it is only a matter of time before infection ensues. Thus it can be seen that the designation *ingrowing toenail* is a misnomer. We are really dealing with ingrowth of the flesh. A vicious cycle is established, and the patient again cuts the corner of the nail to relieve the pain.

Usually by the time the patient consults the physician infection is present and a granulomatous mass projects in relation to the nail edge. Treatment at this stage consists of rest from shoes, socks, and weight-bearing, hot soaks, and the administration of antibiotics in cases of spreading infection. When the inflammation has subsided, a wedge of absorbent cotton is inserted under the corner of the nail to relieve the direct cutting of the flesh. It is sometimes possible with a well-intentioned patient to clear the trouble and arrange for adequate care of the nails in the future.

Frequently the condition is so severe that removal of the nail under general anesthesia is necessary. The infiltration of the infected great toe with local anesthetic should be avoided, as it may lead to spread of the infection and even to gangrene in the older patient. When the

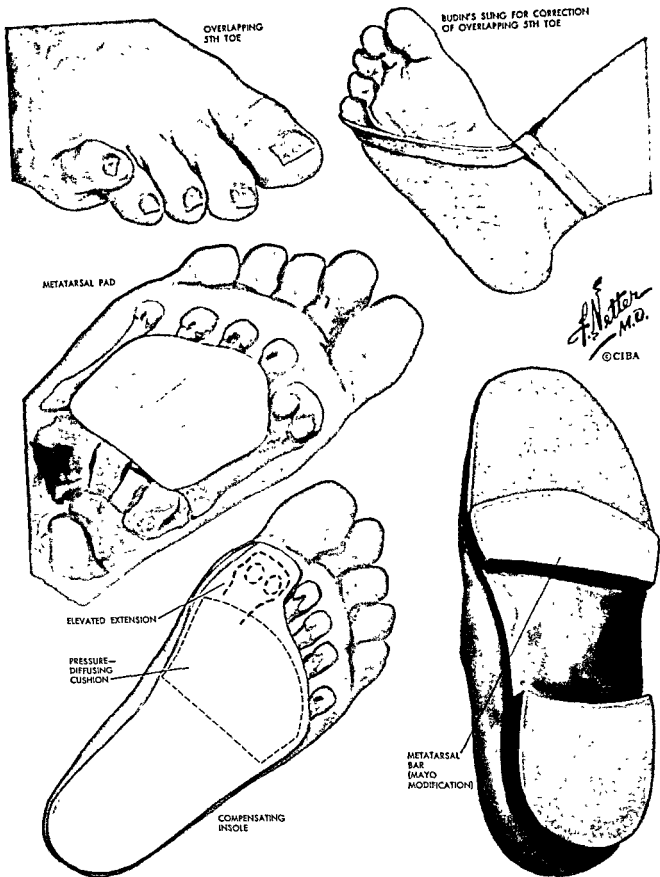
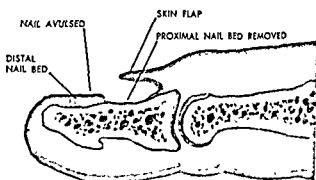
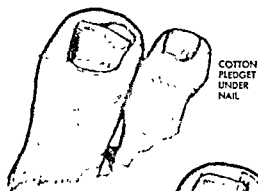
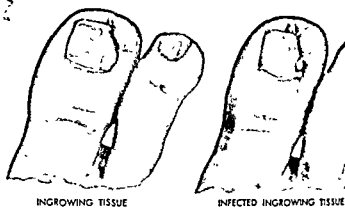
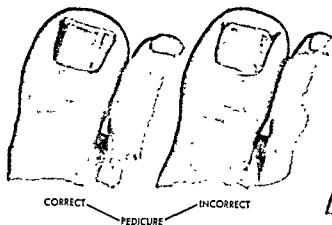
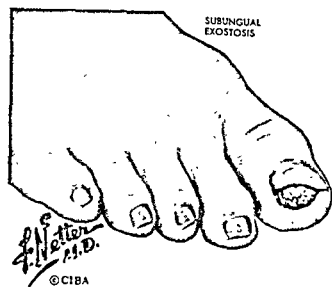


Plate 105.—Overlapping 5th Toe, Metatarsal Pads and Bar.



REMOVAL OF ONE
THIRD OF NAIL BED

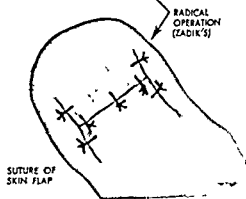
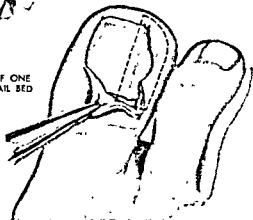


Plate 106.—Disorders of the Nails.

Courtesy Moseley, H. F.: CIBA
CLINICAL SYMPOSIA 9: 83, 1957.

infection has been cleared, advice as to the proper care of the nails must be given, and supervision is required in many cases to see that it is properly followed.

In neglected and recurrent cases, especially when malformation of the nail is a predisposing factor, removal of part of the nail with its nail bed is indicated. Sometimes it is better to remove the whole nail with its bed together with part of the phalanx, and this results in a permanent cure. All remnants of the proximal corners of the nail bed must be secured to prevent regrowth leading to a horny projection through the scar.

In the early ambulatory phase of these various painful conditions a metatarsal bar on the shoe should be worn.

Oonychogryphosis

This is an overgrowth of the nail, so called because of its resemblance to a ram's horn. Different etiologic factors such as irritation of the nail bed by direct trauma, intermittent pressure, and infection may be cited as possible causes.

Treatment.—The size may be controlled by regular attention with an electric burr. Attempts to cut with standard clippers are usually painful to the patient and often unsuccessful. Radical measures include the operation of complete removal of the nail and nail bed as performed for the ingrowing toenail.

Subungual Exostosis

The distal phalanx of the great toe is the occasional site for the development of an exostosis. The bony proliferation projects from the dorsal surface under the nail, causing pain and deformity.

Treatment.—The exostosis is best exposed through a fish-mouth incision, with complete removal of the bony growth including its base.

Common Dermatologic Disorders

Hyperidrosis, or excessive sweating, may involve the feet. It occurs in the overweight individual with chronic foot strain, especially in the summer months. In other individuals it appears to be a vasomotor disorder. At times the

action of bacteria on the moist detritus gives an offensive odor to which the name *bromidrosis* is given. The feet are wet and clammy, and there are often an associated fungous infection and plantar warts.

Treatment should be directed to correct any mechanical dysfunction. The patient should be advised to wash the feet carefully morning and evening in hot soapy water. The parts must be thoroughly dried and any detritus removed. The skin should then be swabbed with rubbing alcohol.

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newed until diminution of the sweating occurs; those with nylon mesh uppers give good aeration. In resistant cases dehydrating solutions such as Burow's (1/20 aluminum acetate) or formaldehyde 6 drams to a quart of water can be used for foot baths. Pro-Banthine has been helpful in certain cases. X-ray therapy is dangerous and should be avoided unless used by experts.

Epidermophytosis, or athlete's foot, is a fungus infection and is frequently associated with hyperidrosis. It is almost universal in persons frequenting the public swimming pools and athletic clubs. The characteristic sodden and cracked white skin between the toes is well known, but the more extensive skin involvement on the soles, the contact spread to the anal and eyelid areas, and the id reaction on the hands are often overlooked. Secondary infection by streptococci and staphylococci may be a serious complication.

Treatment.—Proper foot care with careful washing, drying, and powdering, together with the use of clean socks and shoes, is essential at all times. In public places the use of disposable slippers and antiseptic foot baths should be utilized.

Many agents have been used for eliminating the fungus. When uncomplicated by infection, the unsaturated fatty acids such as propionic and undecylenic acids (Desonex) have been useful. Drying powders such as titanium tannate (Metanium) can be used by day. Exposure to salt water and sunshine is most efficacious.

When infected, antifungicidal remedies should not be used. The infection, when severe

of a narrow shoe. Pressure of the medial surface against a prominent base of the proximal phalanx of the 4th toe or from the deformity of an overlapping 5th toe is the usual cause of the *soft corn* in the 4th interspace.

Treatment of the Hard Corn.—Protection from intermittent pressure by properly adjusted footwear and a protective pad are the primary measures. Careful paring and enucleation of the corn by a chiropodist will give relief. The application of a corn plaster can be used to soften the corn before removal.

Treatment of the Soft Corn.—The mechanical derangement producing the pressure must be removed. When this consists of overlapping toes, prominence of the base of the 4th or 5th phalanx, or incorrect shoes, such must first be corrected. The excess detritus is removed and the toes kept apart and ventilated with lamb's wool. Sweating can be diminished by dusting with titanium tannate (Metanium) powder. Cauterization of the corn may be performed, using electrocoagulation or 50-60% silver nitrate solution. Removal may be secured by one or more applications. Care must be taken to prevent too severe a reaction from treatment, which may result in a deep ulcer with superimposed infection.

Radical Treatment for Hard and Soft Corns.—When the 5th toe is seriously involved with both hard and soft corns, resection of the proximal phalanx (fillet operation) is the most successful procedure and is preferable to amputation.

Treatment of Calluses.—Calluses can be kept in check by regular paring, but cure can only be achieved by the use of mechanical supports or orthopedic correction of deformities. Salicylic acid (40-80%) plasters can be used to soften the callus before paring. A moleskin adhesive covering of the callus is a worth-while palliative protection.

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Title	Running Time	Sound or Silent	Procureable From
Surgical Approaches to the Hip Joint (By use of animated diagrams and actual dissection, approaches to the hip joint from the anterolateral, lateral, and posterolateral aspects are shown) (1951) (By LeRoy C Abbott, M.D., Donald B Lucas, M.D., Paul A Gregorieff, M.D., J Robert Close, M.D., and J B de C M Saunders, M.B., San Francisco)	36 min	Sound Color	Central Office Film Library, Veterans Administration Vermont Ave. and H St., N.W. Washington 25, D C
Supplementary Fixation in Medullary Nailing of the Femur			Veterans Administration, Medical Dept., under direction of Dr. Dana M Street, Memphis, Tenn
Surgical Approaches to the Knee Joint (1951) (By LeRoy C Abbott, M.D., Paul A Gregorieff, M.D., Donald B Lucas, M.D., and J B de C M Saunders, M.B., San Francisco)	36 min	Sound Color	Central Office Film Library, Veterans Administration Vermont Ave. and H St., N.W. Washington 25, D C

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<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Tears of the Medial Meniscus (1917) (By H M Coleman, M.D., Toronto)	21 min	Silent Color	Dept. Medical Illustration Sunnybrook Hospital Toronto, Ont.
The Early Repair of Ruptured Knee Ligaments (1918) (By H M Coleman, M.D., Toronto)	16 min	Silent Color	Dept. Medical Illustration Sunnybrook Hospital Toronto, Ont.
Surgical Approaches to the Ankle Joint (The anatomy and surgical approaches to the dorsolateral and medial aspects of the ankle joint are shown by animated diagrams and dissection) (1952) (By LeRoy C. Abbott, M.D., Robert D. Ray, M.D., Philip D. Wilson, M.D., John J. Callahan, M.D., and J B de C. M. Saunders, M.B., San Francisco)	33 min	Sound Color	Central Office Film Library, Veterans Administration Vermont Ave and H St., N.W. Washington 25, D. C.
Surgical Approaches to Joints of the Foot (By use of animated diagrams and actual dissection, various approaches are shown to anterior and posterior aspects of the joints of the foot) (1951) (By LeRoy C. Abbott, M.D., Robert D. Ray, M.D., J. Robert Close, M.D., and J B de C. M. Saunders, M.B., San Francisco)	33 min	Sound Color	Central Office Film Library, Veterans Administration Vermont Ave and H St., N.W. Washington 25, D. C.
Operative Correction of Metatarsus Varus Primus in Hallux Valgus deformity (1936)	23 min	Silent Color	Paul W. Lapidus, M.D. 1133 Park Ave New York 28, N. Y.
Care of the Feet (1934) (in collaboration with Dudley J. Morton, M.D., New York)	10 min	Sound Color	Encyclopaedia Britannica Films, Inc Wilmette, Ill
Triple Arthrodesis of the Foot (1948) (By Guy A. Caldwell, M.D., New Orleans)	16 min	Silent Color	Alton Oschner, M.D. Medical Foundation 3503 Prytania St New Orleans 15, La
Plantar Neuroma (1950)	19 min.	Silent Color	Lyon K. Loomis, M.D. 116 S. Jefferson Parkway New Orleans, La
Massive Tibial and Iliac Bone Grafts for Non-union of the Tibia (1947) (By Edwin F. Cave, M.D., and Carter R. Rowe, M.D., Boston)	25 min	Silent Color	American Cyanamid Co Surgical Products Division Danbury, Conn.
Rupture of the Quadriceps Tendon Pathology and Surgical Repair (1956)	15 min	Sound Color	Carlo S. Scuderi, M.D. 104 S. Michigan Ave Chicago 3, Ill.
Surgical Treatment of Fresh Injuries to Major Ligaments of Knee (1952) (By Don H. O'Donoghue, Oklahoma)	36 min	Color Silent	American Academy of Orthopaedic Surgeons 116 S. Michigan Ave Chicago 3, Ill.

Chapter 41

Infections of Bone

H Fred Moseley, D.M.

Osteomyelitis is the term used to designate the inflammatory process in bone. When the process is confined to the epiphysis, it is called epiphysitis, and when the inflammation is localized to the surface of the bone beneath the periosteum, it is named periostitis. The inflammatory process may vary in extent and severity and is classified as acute or chronic in nature.

Bacteria may metastasize to bone in the course of a bacteremia or septicemia associated with a systemic infection, resulting in the disease *acute hematogenous osteomyelitis*. In other cases organisms may be directly introduced as a result of trauma which causes open or compound fractures. If the contamination is not adequately removed by surgical débridement, infection of the bone results, and this type is called *primary osteomyelitis*. When bacterial infection extends from a neighboring soft tissue inflammatory process such as an abscess or ulcer to involve bone, the type of lesion is termed *secondary osteomyelitis*. The characteristic reaction of bone to the inflammatory process is determined by the rigid nature of osseous tissue and by the arrangement of the blood supply.

The Structure of a Long Bone.—Osseous tissue consists of an organic matrix upon which calcium salts are precipitated to give it a rigid character. A typical long bone comprises a compact bony cylinder called the *diaphysis* which contains the *medullary cavity*. At each end there is an *epiphysis* of cancellous bone covered by articular cartilage which enters into

the formation of the neighboring articulation. Between the epiphysis and diaphysis in the maturing individual lies the area of maximum growth called the *epiphyseal plate*. The diaphyseal side of this epiphyseal plate receives the richest blood supply. In this area the nutrient vessels end in large capillary loops, and bacteria carried by the blood stream tend to stagnate there. Besides, in the young patient, twisting strains find in this area the weakest point in the bone-joint structure as is well known from epiphyseal fracture separations. This area of the bone so important in growth, injuries, and infections is named the *metaphysis*.

The Blood Supply of a Long Bone.—The work of Lexer remains the classic study in this field and the arrangement can be understood by reference to the blood supply of the tibia. The main nutrient artery enters on the posterior surface of the upper third of the tibia and divides into an ascending and a descending branch which proceed to the respective epiphyseal plates. The branches of the artery pass longitudinally in the medullary canal and in the compact bone through the longitudinal or *Haversian* canals which contain, in addition, the nerves and lymphatics. Besides the arterial circulation to the diaphysis, a second supply derived from the periosteal circulation enters the compact bone along the *Volkman's canals* which run tangentially to the surface. At each end of the bone, arteries enter the epiphysis and the metaphysis separately from the rich

arterial anastomosis around the joint. Because of the arrangement of the vessels in rigid canals, any inflammatory process in bone tends to raise the tension around the vessels and soon causes occlusion and thrombosis. The inflammatory processes within the bone soon release their tension by spread along Volkmann's canals to the subperiosteal area. This extension by stripping off the periosteum interrupts the periosteal circulation and again leads to interference with the nutrition of bone, producing local death of tissues.

a *sequestrum*. While this process of separation and extrusion is taking place, the periosteum, which has been raised from the bone by the inflammatory process, proliferates to give strength to the bone as a whole and produces a cylinder of new bone around the damaged area. This new cylinder of repair bone is called the *involucrum*. The area of sequestration sooner or later communicates with the exterior by *cloacae* traversing this involucrum, and it is through these sinuses that nature attempts to extrude the sequestrum.

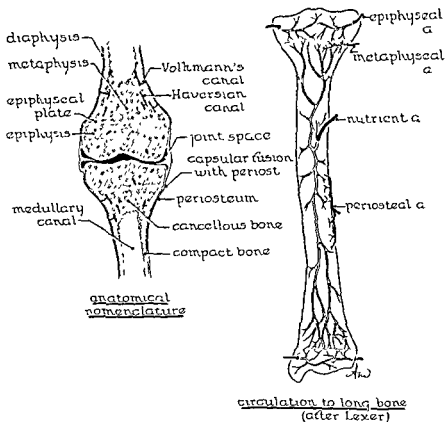


Fig 690.—The circulation in a long bone

The Reaction of Bone to Infection.—The main effect of the inflammatory process on bone is the interference with its nutrition by occlusion and thrombosis of the blood supply. Bacterial toxins also play their part in destroying the living bone cells or osteoblasts. This local destruction of bone is called *necrosis*. If an area of bone has undergone necrosis, the adjacent living tissue reacts to separate and extrude the dead portion. When the area of necrosed bone has been separated, it is called

in flat bones such as the skull and pelvis, the reaction to the infection differs from that in long bones and corresponds more to the cellular death of tissue seen in soft tissue ulceration. This process in bone is called *caries*. Most studies of the progress of infection in bone are made by repeated radiologic examinations. During the early inflammatory process, decalcification due to the intense hyperemia causes a loss in the normal density of the x-ray shadow. This change in bone density and radiologic

appearance is referred to as *osteoporosis*. The increased density that follows ischemia of bone and repair with scarring is termed *osteosclerosis*.

ACUTE HEMATOGENOUS OSTEOMYELITIS

Before the advent of penicillin and the other antibiotic agents, few infective processes afforded the surgeon greater difficulty in treatment than acute hematogenous osteomyelitis. In the fulminating type, death from the septicemia and generalized pyemic abscesses throughout the body was of frequent occurrence. In cases of lesser systemic severity, the extensive necrosis of bone with subsequent sequestration, sinuses, recurrent abscesses, and adjacent joint ankylosis often left the patient a cripple for life. Today, with early diagnosis and appropriate antibiotic therapy, the systemic and local processes can be rapidly controlled, and a dramatic improvement in the mortality and morbidity rates has resulted.

Bacteriology.—The vast majority of cases of acute osteomyelitis (90%) is caused by *Staphylococcus pyogenes*. A relatively small number is due to *Streptococcus pyogenes* or *Diplococcus pneumoniae*, and rare individual cases may be caused by other microbes. In acute hematogenous osteomyelitis the causative organism can be isolated from the blood stream or the local lesion in pure cultures. In primary or secondary osteomyelitis mixed infections may be found and have to be treated accordingly. *Streptococcus pyogenes* and *D. pneumoniae* infections are more prone to involve the epiphysis and the neighboring joint.

Pathogenesis.—The most common source of staphylococcal osteomyelitis is an infection of the skin, furuncle, or carbuncle, etc. From the cutaneous lesion, invasion of the blood stream occurs, with or without clinical manifestations, and the organisms are carried to the metaphysis of the bone. The most common site involved is the region of the knee, including the upper end of the tibia and lower end of the femur. The ankle is second in frequency, and the metastasis involves the lower end of the tibia or the lower end of the fibula. The upper end of the humerus and upper end of the femur are also common sites, but it must

be remembered that any metaphyseal area may be involved. Re-entry of bacteria from the osseous lesion into the circulation may in turn cause septicemia or further metastatic foci in other bones or organs.

Various factors predispose to the development of this disease. Among these the age of the patient is most important. The age period marking the maximum incidence is 8-12



Fig. 691.—Museum specimen of chronic osteomyelitis of the tibia showing involucrum, sequestrum, and cloacae.

years, but all ages from the first week of life until epiphyseal closure around 20 years may be affected. Boys are more commonly afflicted than girls, and this sex incidence lends support to the theory that juxtaepiphyseal strain caused by an injury is a predisposing factor. As previously mentioned the area of the metaphysis is the weak link in the bone joint structure in the growing patient, and strains may produce a hematoma affording a nidus for circulating bacteria. Lowered resistance and climatic influences are often mentioned as contributing factors. Cases of osteomyelitis are more common in the late winter months in the north temperate climates and in the poorer classes of the population.

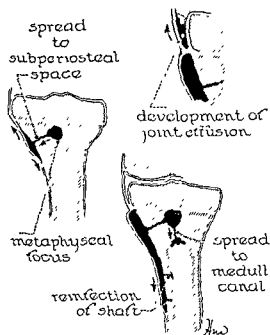


Fig. 692.—The spread of osteomyelitis in a long bone

The Pathologic Spread in Bone.—The accepted ideas on the spread of the infection from the fixation point in the metaphysis follow the observations of Starr and are summarized in Fig. 692. As the tension rises from the suppurative process, the inflammation extends into the cancellous tissue and down the medullary canal. The next extension is outward to the subperiosteal space along the Volkmann canals. The epiphyseal plate affords a strong

barrier to the passage of bacteria into the epiphysis and thus into the joint. Once the subperiosteal space is involved, the tension again rises and the periosteum is stripped off the bone and the shaft infected via Volkmann's canals. Because of the close relation of the subperiosteal abscess to the synovial reflexion and joint capsule, an effusion into the joint may occur, and in time a pyogenic arthritis develops in the neglected cases.

In certain cases the embolism may affect the metaphyseal area only; in other cases the embolus may block either the main ascending or the main descending branch of the nutrient artery, or the entire vessel may be blocked in the initial stage. These different sites of embolism determine the pattern of the inflammatory process and have been emphasized by Wilensky.

As the bone is rendered ischemic by the thrombophlebitis and also by the cellular death caused by bacterial toxins, the process of sequestration begins. In the neglected cases in the preantibiotic era the separation was completed in the average case in 6-8 weeks.

Clinical Types of Acute Hematogenous Osteomyelitis.—There are three main clinical types of this disease determined by the severity of the systemic and local process:

1. The fulminating type in which septicemia and pyemia predominate and the bone lesion is of secondary importance.
2. The type where the blood stream invasion and osteomyelitic process are practically silent. The bacteremia passes unnoticed and the osteomyelitis becomes clinically apparent with the development of a subacute or chronic Brodie's abscess.
3. The common type in which a septicemia of varying severity exists for several days and the osteomyelitic process predominates.

Clinical Picture.—The clinical picture will vary, depending on the intensity of the septicemic and osteomyelitic processes.

In the *fulminating type* the young patient is desperately ill and frequently delirious and irrational. The temperature ranges above 104° F., the pulse is 120-160, and the leukocyte count 25,000 or more. The patient is often dehydrated. Complaints may be of fleeting pains

from one bone or joint to another. The general symptoms and signs far overshadow any local signs in bone which may not be discovered. A blood culture is usually positive if taken just before the peak of the temperature, and the prognosis can often be judged by the number of colonies cultured and the number of days the septicemia persists. Before the advent of penicillin the majority of these patients died from generalized pyemic abscesses in brain, lung, kidney, and other organs.

the symptoms and signs will be both general and local.

The general features will be determined by the intensity and duration of the septicemia. The patient will be ill with a raised septic type of temperature, rapid pulse rate, and leukocytosis. Repeated blood cultures in the early stages will be positive and enable the isolation of the organism and study of the antibiotic sensitivity. As previously stated 90% or more are due to the staphylococcus.



Fig. 693 --X-rays showing early changes of acute hematogenous osteomyelitis

A, Primary lesion shown 10 days after the onset of disease

B, Several weeks after evacuation of subperiosteal abscess with primary closure and antibiotic therapy. Note decalcification and periosteal thickening

In the *silent type* with the development of a Brodie's abscess, the general symptoms and signs are negligible. The patient complains of an increasing pain or aching at the extremity of a long bone with some limitation of function of the neighboring joint. In the lower limb a lump or a tender enlargement of the bone may be noted.

In the *common type* of case with a septicemia of several days' duration and an acute inflammatory process in one of the long bones,

The local symptoms are those of a circumscribed acute infection. The patient complains of an increasing intense and throbbing pain situated deep in the limb which will be localized by the examiner on palpating the area of maximum tenderness. The area may be swollen and reddened. If the process is of several days' duration, a subperiosteal abscess with fluctuation may be present.

Radiologic examination in the early stages is generally negative, but an area of rarefaction

near the epiphyseal line will be apparent after 7 or more days.

Differential Diagnosis.—The history of a sudden illness with acute pain in one of the limbs in a young active child or adolescent should suggest an acute hematogenous osteomyelitis. The diagnosis will be assisted if there has been a recent skin or upper respiratory infection. In the infant an infected umbilicus should be looked for. Acute osteomyelitis must be differentiated from acute rheumatism. In *acute rheumatism* the pain, tenderness, and swelling around a joint usually appear at once, whereas in acute osteomyelitis the pain generally precedes the swelling of the joint by 2-3 days. Besides, in acute rheumatism it is customary to have the pains in several joints and often fleeting from one to another. Acute osteomyelitis is characterized by exquisite tenderness of the bone, and the young patient recoils from the examiner. It is best to regard any acute process at the end of a long bone in the child or adolescent as acute hematogenous osteomyelitis and treat promptly as such by appropriate antibiotic therapy. If the process is *rheumatic in character*, the response to antibiotics will not be satisfactory but will usually be dramatic when salicylates are administered.

In *poliomyelitis* the young patient may complain and give signs of pain and loss of function in one limb, but characteristically the patient is not so ill nor the local signs so marked as in acute osteomyelitis. Neck rigidity will be present, and lumbar puncture will reveal increased cells in the cerebrospinal fluid.

The differentiation from a *sprain or fracture* may be required, but in these cases the systemic signs of an infection are lacking. However, the association of juxtaepiphyseal injuries with osteomyelitis must be remembered. Radiologic examination often discloses minor degrees of epiphyseal injuries with flakes of bone torn off with the periosteum or fragments of the metaphysis fractured.

Treatment.—The treatment of acute hematogenous osteomyelitis had undergone great changes by the time the chemotherapeutic and antibiotic drugs had arrived. More and more, surgeons were devoting their attention to supportive general treatment directed toward

the septicemia and treating the local process in bone with rest to the part and masterly inactivity from an operative point of view. The results of radical removal of cortical bone and exposure of the medullary canal had not been satisfactory and many surgeons were content to open the periosteum and place fine drill holes into the metaphyseal area to lessen the tension and afford drainage. The use of the antibiotic drugs has made the treatment of acute osteomyelitis practically a medical problem.

The principles underlying the modern treatment may be summarized as follows:

1. The patient should be treated in bed and general supportive measures given. Small transfusions are valuable in the early and late stages.

2. Blood cultures should be taken to isolate the specific organism and to study its antibiotic sensitivity.

3. While awaiting the results of blood culture, penicillin in massive doses and streptomycin should be given. In the fulminating type, intravenous penicillin should be administered. If the organism does not respond promptly to this regimen, the most suitable antibiotic determined by the sensitivity should be tried in place of penicillin.

4. The bone involved should be placed at rest in a plaster-of-Paris splint with immobilization of the adjacent joints. Should the patient be seen late and a subperiosteal abscess be present, bacteriologic study of the aspirated pus should be made. The subperiosteal area may be irrigated and local penicillin, 300,000 units, instilled. Similarly, if an effusion into the joint is present, it should be aspirated and the fluid cultured. If bacteria are present, penicillin should be instilled.

It may be mentioned that there are those today who believe that the local treatment should include the incision of the periosteum and washing out of the subperiosteal abscess and primary closure of the wound. Others prefer to add to this multiple drill holes into the metaphyseal area to diminish the tension. More and more, the cases are being diagnosed and treated at an earlier stage. It can almost be stated that if prompt treatment by the appropriate antibiotic is given within 5 days of the

onset, the general and local processes can be controlled. Indeed, according to Trueta, if penicillin treatment at the level of one million units a day is started within 48 hours of the onset of the disease, it appears to prevent altogether the bone changes usually detectable by roentgenographic examination. After this period there is a greater incidence of bone necrosis leading to sequestration.

CHRONIC OSTEOMYELITIS

Patients presenting chronic osteomyelitis include the end results of three groups of cases in which the acute primary infection has not been eradicated and the bacteria have become organized in the scarred and devascularized bone. The radiologic examination shows osteosclerosis and probably sequestration

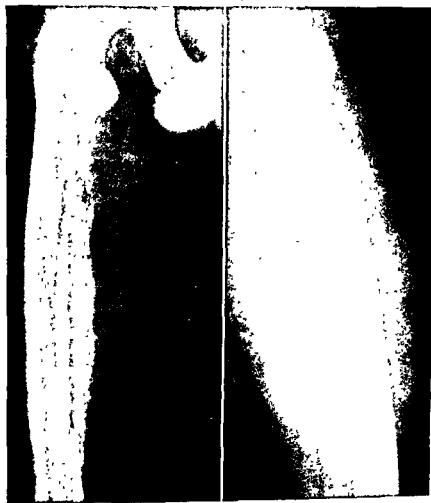


Fig. 694—X-ray showing late changes of chronic osteomyelitis

Complications.—In the fulminating cases, toxemia and pyemia with multiple metastatic abscesses in the organs and serous cavities lead to death.

In the cases which are neglected, sequestration with the development of recurrent abscesses and sinuses are the clinical features of one type of chronic osteomyelitis. Such cases so frequent in the pre-antibiotic era should soon become uncommon.

The three groups of cases:

1. Late cases of acute hematogenous osteomyelitis

2. *Open fractures*, including those due to war wounds with presence of foreign bodies such as shell fragments; *closed fractures*, infected at operation by penicillin resistant staphylococci, as a result of the increasing use of open reduction and internal fixation for such cases



Fig 695—Tuberculous abscess of upper tibia with involvement of the knee joint, treated by resection of the diseased tissues, packing of the cavity with cancellous bone chips, and compression arthrodesis of the knee

A and B, Preoperative appearances, anterior and lateral views
C and D, Postoperative appearances 3 months later

3. Chronically infected bone from spread from adjacent soft tissue infections

The cases of chronic osteomyelitis following each of these primary types of infection are becoming less frequently seen because of the prompt and efficient antibiotic therapy in the early stages, but during World War II countless numbers of Group 2 were seen and great advances made in their treatment. The time has come when the severe general reaction to such chronic infections with weight loss, secondary anemia, lowered plasma proteins, and amyloid disease is rarely seen.

The principles underlying the local treatment of the infected bone are based on the fact that the bacteria are protected from antibiotic drugs by the scarred and devascularized bone. It is therefore necessary to remove such bone, scarred and adherent skin and soft tissue, together with any sequestra and foreign bodies that may be present. This is done after the general health has been improved by appropriate transfusions and nutritional and vitamin therapy as is required, and under the protection of antibiotics begun 3-5 days preoperatively. A careful bacteriologic study of the wounds is made prior to operation, and the nature of the mixed infection is determined. Where possible the local condition is improved by topical application of antibiotics prior to operation.

Once the dead bone and scarred tissues have been removed, the wound is closed with a petrolatum pack for 7-10 days. In some cases the surgical procedure results in the saucerization of a long bone, in others an exteriorization of a cavity. At the end of this period if a clean granulating surface is provided, the area is covered with a split-thickness skin graft. In certain cases where a satisfactory vascular bed is present, primary skin grafting is possible. Small sequestra may separate through such a covering, but when the skin graft has finally closed the area, consideration may be given to filling defects by various procedures, which include the use of cancellous bone chips to fill cavities and the transfer of muscle, fat, and skin flaps to improve the cosmetic appearance.

By such means and with the protection of systemic and local antibiotic therapy, many

limbs previously amputated and others presenting recurrent abscess and sinus formation can now be cleared of their chronic osteomyelitis.

SPECIFIC TYPES OF OSTEOMYELITIS

Typhoid osteomyelitis is now a rare condition due to the public health measures controlling the water and milk supplies. It is mentioned to remind the student that in typhoid cases involvement of bones such as the tibia, rib, sternum, and vertebrae may occur in the 2nd or 3rd week of the disease or even years after the disease has subsided. In these cases the abscesses, if incised, will discharge pure cultures of typhoid bacilli and can cause contamination of other patients.

Tuberculous osteomyelitis is also today of decreasing significance. Usually the initial lesion is in the juxta-articular area and involves the corresponding joint. This has been discussed in the general section on joints and in the section Pott's disease in Disorders of the Vertebral Column.

Occasionally, tuberculous infection is a cause of a chronic abscess in bone and is a differential diagnosis in cases of Brodie's abscess. Rarely, one sees an expansile swelling in the small bones of the fingers due to this cause, which is called *tuberculous dactylitis*.

Syphilitic infection of bone is also a relatively rare condition from the surgical standpoint. Efficient and early treatment of the initial lesion in the free clinics has caused the disappearance of such cases.

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Chapter 42

Tumors of Bones and Joints

J. Gordon Petrie, M.D.

Tumors of the bone may be benign or malignant. In addition metastatic tumors of bones arise from malignant growths in other parts of the body.

GENERAL CONSIDERATIONS

The *benign bone tumors* grow slowly, do not metastasize, and rarely show abnormal blood findings.

The *primary malignant bone tumors* vary in rate of growth, do metastasize, and frequently show blood changes to help in differentiation.

The *metastatic tumors* to bone are most frequently from carcinoma of the breast, prostate, kidney, thyroid, and lung.

The exact cause of bone tumors is unknown. Trauma is usually considered an important factor in the development of certain bone tumors.

The patient suspected of a bone tumor should have a careful history, a complete general examination, a hemogram, urinalysis, blood serologic and chemical determinations, a roentgenogram and a biopsy.

Laboratory Examination.—Certain types of bone tumors produce changes in the blood and

TABLE 37
CLASSIFICATION OF PRIMARY BONE TUMORS

TYPE	BENIGN	MALIGNANT
1. Osteogenic series	a. Exostosis, osteoma b. Osteoid osteoma	Osteogenic sarcoma a. Medullary and subperiosteal b. Telangiectatic c. Sclerosing Periosteal sarcoma Fibrosarcoma a. Medullary b. Periosteal Capsular and periosteal sarcoma
2. Chondroma series	Chondroma	Chondrosarcoma
3. Giant	"	"
4. Angio	"	"
5. Reticuloendothelial series	Solitary myeloma	a. Reticulum cell sarcoma b. Ewing's sarcoma (endothelioma) c. Multiple myeloma d. Myelosarcoma
6. Fatty tissue series	Lipoma	Liposarcoma

urine which may help in their differentiation. *Alkaline phosphatase* is secreted by proliferating cartilage cells and osteoblasts and excreted by the liver, so there is an increase of alkaline phosphatase in the blood with liver impairment, and also during the active phase of bone growth. This is found also in the healing of fractures and in certain bone tumors such as osteogenic sarcoma and in patients with bone metastases especially from the prostate.

Acid phosphatase is an enzyme found in large amounts in hypertrophy or in carcinoma of the prostate gland. When the carcinomatous tumor has ruptured the capsule of the gland

amputation is carried out on a patient with any bone tumor. The wound after surgical biopsy must always be closed and never packed or drained.

BENIGN TUMORS

Osteoma

Osteomas or exostoses frequently arise from a bony surface. Those arising from the center of the bone are known as enostoses. An osteoma may be compact or cancellous. The former frequently originate from the periosteum of membranous bones, e.g., skull or face.



Fig 696—Lipoma of bone. A very rare benign tumor of bone that responded completely to curettage and packing with bone chips.

and metastasized to the soft parts or to the bones, there is, in about 70% of cases, an increase of acid phosphatase in the circulating blood.

The *Bence Jones protein* may be found in the urine in 60% of cases of multiple myeloma. Bone marrow biopsy from the sternum is positive in 95% of cases of multiple myeloma.

In all bone tumors where there is any doubt as to diagnosis, a biopsy should be done. A biopsy must always be performed before an

Treatment by excision is indicated when such a tumor causes pressure on nerves or deformity. A large group of exostoses originate at the ends of long bones and are often multiple, hereditary, and associated with other abnormalities.

Osteoid Osteoma

This tumor, described by Jaffe in 1935, occurs most frequently in patients 10-25 years of age. In general, long bones are affected. There is slight swelling and localized tender-

TABLE 38

CHARACTERISTICS OF PRIMARY BONE TUMORS

TYPE	INCIDENCE OF ALL BONE TUMORS	AGE (YR.)	SEX	TYPES OF BONES AFFECTED	ORDER OF FREQUENCY	SITES OF ORIGIN IN BONE	METASTASES	X-RAY APPEARANCES	HISTOLOGY
1 Osteogenic sarcoma	50%	10-20	$\frac{M}{F} = \frac{4}{3}$	Long bones any bone	Femur Tibia Humerus Pelvis Fibula Scapula	Ends (meta- physis)	Lungs and other bones	Rarefaction Sclerosis New bone formation Codman's sun-ray spicules	Spindle cells Pleomorphic cells Bone cartilage Osteoid
2 Ewing's tumor	12.5%	5-15	$\frac{M}{F} = \frac{2}{1}$	Long bones (short bones)	Tibia Fibula Humerus Femur Jaw Small bones	Center (di- aphysis)	Skull Lymph glands Lungs Other viscera Subcutaneous tissues	Sclerosis at cortex and medulla "Onion peel" New bone Patchy rarefaction	Round polyhedral cells Very scanty intercellu- lar stroma
3. Multiple myeloma	3%	40-60	$\frac{M}{F} = \frac{2}{1}$	Flat bones	Ribs Vertebrae Sternum Skull Pelvis Clavicle	Center (me- dulla)	Spleen Liver	Bone destruction No compensatory new bone formation	Round or oval plasma cells (giant cells)
4. Parosteal sarcoma	15%	25-45	$\frac{M}{F} = \frac{1}{1}$	Long bone Flat bone Skull	Femur Tibia Pelvis Scapula Skull	Center (di- aphysis)	Lungs	Erosion from outside Osteolysis Irregular calcification in soft tissue	Spindle cells Oat cells in bundles Intercellular collagen- ous material
5. Osteoclas- toma, be- nign	20%	16-30	$\frac{M}{F} = \frac{1}{1}$	Long bone	Tibia Femur Radius	Ends (epiphy- sis)	None	Expansion Thinning at cortex Trabeculation No new bone forma- tion	Multinucleated giant cells (15-50 nuclei) Round cells Spindle cells (stroma) Vascular spaces

TABLE 39
CLASSIFICATION OF BONE ENLARGEMENT

UNDER 20		OVER 20	
OSTEOLYTIC	OSTEOBLASTIC	OSTEOLYTIC	OSTEOBLASTIC
<i>A. Single Lesions</i>			
Benign bone cyst	Exostoses or osteochondroma	Benign chondroma	Periosteal fibrosarcoma
Osteogenic sarcoma	Ewing's tumor	Giant cell tumor (osteoclastoma)	
Myxosarcoma	Periosteal osteogenic sarcoma	Bone cyst	
Pyogenic bone lesions	Garré's sclerosing osteitis		
<i>B Multiple Lesions</i>			
Metastases	Rickets	Metastatic carcinoma of thyroid or kidney	Paget's disease
Sympatheticoblastoma		Multiple myeloma	Metastatic carcinoma of prostate or breast
Osteomyelitis			



Fig. 697—Bilateral multiple osteochondromas of lower end of femur, and upper end of tibia

ness but no heat or redness. The roentgenogram shows a zone of sclerosis, in which is an area of rarefaction and a nidus. Either sex may have the tumor. The differential diagnosis includes Brodie's abscess, Garré's sclerosing osteomyelitis, eosinophilic granuloma, Paget's disease, and fibrous dysplasia. The differentiation may be made by the history, the x-ray, and at times the biopsy. The treatment is surgical excision of the nidus; if removal is incomplete, the tumor continues to grow and

to cause symptoms. Histologically, one sees cellular proliferation (osteoblasts), patches of osteoid tissue, and areas of calcified atypical bone.

Osteochondroma or Cartilaginous Exostosis

This common tumor occurs chiefly between 10-30 years of age and is most frequently found in the region of the knee joint. The symptoms are usually associated with interference of function of the joint or tendons,

Surgical excision should be carried out. These tumors are often encapsulated and can be readily shelled out. Degenerative changes occur so that myxomatous degeneration, cyst formation, calcification, and ossification are frequently seen. Geschickter and Copeland state that malignant transformation occurs in 7% of cases. The growth then becomes much softer, the regularity of cell arrangement is lost, and invasion occurs.

Chondroma

This tumor occurs centrally (enchondroma) in the small bones of the hands or feet or projects from the surface of the bone (exenchondroma). The centrally occurring chondromas cause expansion of the bone and are best treated by curettage and by filling the defect with bone chips.



Fig. 698—Chondroma (enchondroma) of phalanx

Giant Cell Tumor (Osteoclastoma)

This tumor occurs between 15-30 years of age. The lesion is locally destructive and tends to recur but does not metastasize. The commoner locations are the lower end of the femur, the upper end of the tibia, and the lower end of the radius. They may also occur in the jaw, pelvis, tendon sheaths, bursae, and capsules of joints. The tumor arises in the epiphyseal area of the bone as a soft, dark red, hemorrhagic mass, which is frequently lobulated and causes expansion. Periosteal new bone does not occur unless a pathologic fracture has been present.

Appearance of the Tumor.—The tumor is cystic, hemorrhagic, and traversed by bony trabeculae. The cortex is extremely thin. On microscopic section it shows three types of cells:

1. Small round cells which are numerous
2. Large multinucleated (15-20 nuclei) giant cells
3. Spindle cells

The stroma shows spindle cells and also vascular spaces.

The treatment of choice is curettage and insertion of bone chips unless the tumor occurs in a bone where resection is possible. Curettage must be complete, as recurrences follow those incompletely treated. If a recurrence follows, then a thorough curettage should again be done and the cavity filled with bone chips, or, if possible, excision of the tumor and replacement of the defect with bone grafts and bone chips.

Röntgen therapy should be used only on inaccessible tumors or when excision would lead to mutilation or functional impairment.

Some persons doubt that there is such a tumor as a malignant giant cell tumor.

In the differential diagnosis of giant cell tumor of bone the following should be considered: nonosteogenic fibroma, osteogenic sarcoma, simple (unicameral) bone cyst, aneurysmal bone cyst, benign chondroblastoma, fibrous dysplasia, and the bone lesions of hyperparathyroidism.

Nonosteogenic fibromas are often discovered incidentally on x-rays of older children. Microscopically the predominant cells are fibroblasts.



Fig 699—Giant cell tumor of lower end of tibia, before and after operative excision, and filling of cavity with cancellous bone chips

The *simple (unicameral) cyst* has a wall of fibrous tissue which may contain numerous benign giant cells. The cystic spaces contain gas or fluid unless a fracture is present, when they may contain blood.

The *aneurysmal bone cyst* has spaces filled with unclotted blood and portions of solid tissue containing many giant cells.

The *benign chondroblastoma* shows chondroid ground substance which may show degeneration and later calcification.

Fibrous dysplasia may show giant cells but will also present irregular bony trabeculae or areas of osteoid tissue.

Hyperparathyroidism shows fibrous replacement of bone in which many giant cells can be found. There are a consistently elevated serum calcium, a decreased serum phosphorus, and an increased blood phosphatase. The excretion of calcium in the urine is also increased.

Hemangioma

This rare tumor occurs usually in the spine or in the skull. In the roentgenogram of the vertebra the appearance is honeycombed and may show collapse and wedging of the body. It must be distinguished from an ordinary compression fracture. This tumor responds to roentgen therapy. Occasionally it occurs in a long bone.

Solitary Cyst of Bone

Solitary cysts of bone occur typically in the metaphysis and adjacent diaphysis of long bones before the related epiphysis has closed and thus usually before the age of 20 years. The x-ray appearances are similar to those of giant cell tumor, but this usually occurs after the age of 20 and involves the epiphysis itself. Some authorities believe that the two conditions are closely interrelated.

The solitary cyst may be simple or multilocular. The exact causation is unknown, but the cyst may arise as a result of a metaphyseal hemorrhage or as the result of an attenuated central bone abscess.

The patient seeks help most frequently for a fracture which has occurred through the cyst. The common sites of involvement are the upper end of the humerus and upper end of the femur.

Treatment.—A considerable proportion (c. 50%) are cured by the healing process after the pathologic fracture. If the cystic changes persist or extend after fracture or are found on x-ray examination for pain, operative treatment is indicated. This consists of removal of an adequate area of compact bone to expose the cavity, thorough curettage of the wall, and cauterization with saturated solution of zinc chloride followed by packing with cancellous bone chips. Protection of the area by immobilization in plaster is required until the x-rays show consolidation. X-ray treatment is best avoided because of the possibility of damage to the epiphysal plate.

Eosinophilic Granuloma

There are a number of lesions of the skeletal system which are believed to have reticuloendothelial elements in them, namely, *Gaucher's disease*, *xanthomatosis*, *eosinophilic granuloma*, and *Letterer-Siwe disease*. The etiology remains unknown.

Eosinophilic granuloma affects chiefly older children and adults. The granuloma may be of the soft parts or of the bone. The disease is insidious with possibly a low-grade fever and some pain. Pathologic fracture may occur. Diagnosis depends on biopsy. Excision with bone replacement or radiation is the recommended treatment.

Osteitis Deformans (Paget's Disease)

This is a generalized disease showing a thick and enlarged skull, bowing of the legs, and changes in other portions of the skeleton. It affects adults only. The disease does not shorten life significantly unless sarcomatous changes in one of the involved bones occur.

The affected bones show enlargement with changes in contour. The bone and marrow show areas of degeneration and areas of attempted repair.

The serum calcium and phosphorus are normal, and the serum phosphatase is consistently elevated and appears to parallel the skeletal involvement and activity as shown by roentgenographic examination.

Polyostotic Fibrous Dysplasia

This is a rare disorder showing a regressive metaplasia of bone into fibrous tissue. In those affected, precocious puberty is frequent in females, but the sexual development of males is normal. The disease begins in childhood of unknown cause. Some of the cases show cutaneous nevi. The progress of the disease may be slow or rapid, and pathologic fractures may be produced.

The entire skeleton should be examined roentgenographically, and if doubt in diagnosis exists, biopsy is done. There is no cure for the basic disorder. Pathologic fractures are to be treated with braces or splints, and bone graft replacements should be considered.

PRIMARY MALIGNANT TUMORS OF BONE

These are found chiefly in the younger age groups. They are relatively rare in comparison with other malignant growths and can present many difficulties in diagnosis to the roentgenologist, pathologist, and surgeon. Successful treatment of bone sarcoma, as in other types of malignant lesions, depends upon early diagnosis, the type of tumor, and to some extent on its location.

Osteogenic Sarcoma

This is the most malignant and most common bone tumor. Mayo Clinic statistics show that in 75% of cases, osteogenic sarcoma oc-



Fig. 700



Fig. 701

Fig. 700—Paget's disease involving the tibia in which sarcomatous degeneration has occurred at the upper end. Note the oval area of rarefaction with destruction of the anterior cortical bone.

Fig. 701—Fibrous dysplasia of the radius treated by resection and replacement by bone graft.

curs about the knee. Most cases occur between 10-20 years of age and slightly more in males than in females. The metaphysis of the lower end of the femur or that of the upper end of the tibia is usually involved, other sites being upper end of humerus, upper end of fibula, pelvis, and scapula.

Symptoms and Signs.—Pain, at first intermittent and then continuous, is the chief symptom. It may be accompanied by a limp, and later swelling may be seen. The diagnosis must take into consideration many factors,

The alkaline phosphatase is increased in osteogenic sarcoma. Osteogenic sarcoma metastasizes by the blood stream to the lungs and to other bones.

Histology.—The microscopic picture is most variable in this lesion. Spindle cells predominate, especially in the more malignant types, and occasional giant cells are present. The intercellular substance may consist of mixtures of cartilaginous, myxomatous, osteoid, osseous, fibrous, and hyaline tissue.



Fig. 702.—Osteogenic sarcoma involving the upper shaft of femur, showing the radiating spicules of bone and raised periosteum in anteroposterior and lateral views

history, physical examination, roentgenogram of tumor and of chest, laboratory examination, surgical biopsy, and previous treatment. Microscopic examination of the tissue is the most valuable aid, but this may also be difficult if the tumor has been previously irradiated or if infection has occurred from a previous biopsy. Laboratory examinations should include urinalysis, hemogram, and blood Wassermann

X-ray Features.—These vary somewhat, depending upon whether the tumor is osteolytic or osteoblastic. Early, the x-rays show an area of bone destruction in the subcortical zone extending outward. Strands of new bone formation are seen with raising of the periosteum but no expansion of the cortex. The new bone is frequently laid down as radiating spicules at right angles to the shaft.

Treatment.—Amputation is usually the preferred treatment. Partial amputation or resection has been considered for some cartilaginous tumors in the distal end of an extremity. Osteogenic sarcoma in children may be radio-sensitive, and x-ray may be used as a pre-operative treatment. X-rays of the lungs should always be taken before surgical removal is undertaken.

Site of Amputation.—One must decide whether to do the amputation through the bone affected above the tumor or through the bone proximal to the bone affected. When amputation is through the bone affected above the tumor, a section should be made from the end of the bone removed, and if malignancy is present, amputation above the joint or disarticulation is indicated. If pulmonary metas-



Fig 703—Fibrosarcoma of the lower femur. This lesion appears to be arising from the medullary portion of the bone and is usually of low-grade malignancy. This patient had symptoms of pain and dysfunction for more than a year and was treated by amputation. Microscopically the picture was of malignant fibroblasts with some pleomorphism.

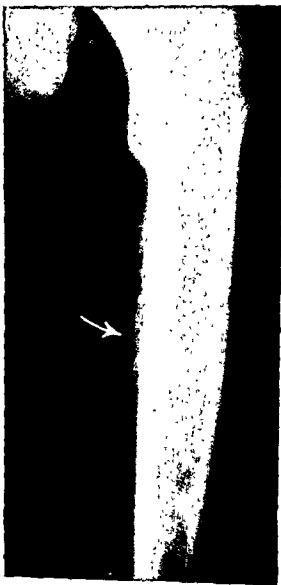


Fig 704—Ewing's endothelioma of the femur.

tases are already present, no surgery should be done, unless for palliative reasons, e.g., to remove a useless leg or for pain.

Chondrosarcoma

This tumor appears in adolescence and early adult life. It occurs chiefly at the lower end of the femur and upper end of the tibia. The tumor consists of cartilage, connective tissue, and bone derived from malignant tumor cells.

Treatment.—Biopsy should first be done. If the diagnosis is confirmed, early amputation is necessary.

Endothelial Myeloma (Ewing's Sarcoma)

This tumor is found in children and in young adults. The shafts of long bones are most commonly involved, but occasionally the jaw and small bones are also affected.

Fever is a frequent sign of Ewing's tumor and not of other bone tumors. The differentiation between this tumor and osteomyelitis has always been difficult but is more so if anti-



Fig. 705—Reticulum cell sarcoma of pelvis and femur. This bears considerable resemblance to Ewing's tumor, although in general it occurs at a slightly older age and is extremely malignant and very radiosensitive. It can usually be differentiated only microscopically.

A secondary type of chondrosarcoma may arise from a pre-existing osteoma, chondroma, or Paget's disease. Biopsy for confirmation should be performed. If microscopy discovers sarcoma, amputation is justifiable in the absence of pulmonary metastases.

biotic or preliminary x-ray therapy has already been given. The sedimentation rate may be raised in these cases, but the leukocyte count is not usually as high as in cases of osteomyelitis.

The diagnosis is to be made by biopsy and microscopic examination.

The structure of the tumor is semisolid and of a grayish white color with areas of hemorrhage. The tumor extends along the medullary canal as well as through the haversian canals to the surface of the bone. The inner layers of bone are destroyed while new layers of bone are deposited on the outside, described as "onion layers" in the x-ray picture.

The common sites are the shafts of the long bones (tibia, humerus, femur, fibula). The os calcis is the most frequently involved small bone.

Treatment.—This is one of the most radiosensitive of all tumors, and x-ray therapy should always be given after the diagnosis has been established. This usually results in diminution of the tumor, but secondary deposits are common. Amputation of the affected limb following a course of irradiation is generally preferred. This should be done through the bone proximal to the affected bone, if possible. Resection of certain bones, e.g., fibula, ulna, or scapula, is possible if the tumor is present. The metastases of Ewing's tumors are



Fig 706—Multiple myeloma of pelvis and upper femur

Histology.—Small round and polyhedral cells are arranged in solid masses. The intercellular stroma is scanty. The picture may be obscured by degenerative changes or previous irradiation.

X-ray Features.—Central rarefaction of a patchy type with sclerosis of the cortex and "onion peel" new bone are characteristic.

also radiosensitive and are frequently found in other bones (skull, sternum, vertebrae). Pulmonary metastases are common.

Multiple Myeloma (Plasma Cell Myeloma)

This tumor is being diagnosed with increasing frequency though formerly it was considered a rare disease. It occurs chiefly in

adults 40-60 years of age and is almost twice as common in males as in females. The origin of the tumor is from the hematopoietic cells of the bone marrow and involves chiefly the spine, pelvis, skull, ribs, and proximal long bones in multiple locations.

Multiple myeloma usually manifests itself by pain, especially in the back and thorax, by weight loss, and by pathologic fractures which are common.

X-ray Features.—There is bone destruction without compensatory new bone formation. Early cases may show only a diffuse osteoporosis, later the trabeculae and cortex are destroyed and appear as multiple clear punched-out areas.

Diagnosis.—(1) Bone marrow biopsy of the sternum or of the actual lesion shows typical round or oval cells with eccentrically placed nuclei, arranged diffusely without intercellular substance. (2) Bence Jones protein is present in the urine in about 50% of the cases. (3) Hyperproteinemia, usually of the globulin fraction, is present in approximately 60% of the cases. (4) Uric acid may also be elevated in some cases and is probably a reflection of the nucleoprotein catabolism of the myeloma cells.

Treatment.—An occasional solitary lesion has been excised, with complete relief, but it is more usual for the solitary lesion to show multiple involvement at a later date. The main treatment is palliative and includes deep x-ray therapy or urethane for the relief of pain, together with general supportive measures. The disease is usually fatal in 2-3 years.

SECONDARY MALIGNANT TUMORS OF BONE

Metastatic deposits of carcinoma occur in bone from primary carcinoma in any tissue but are chiefly from carcinoma of the breast, prostate, thyroid, kidney, and bronchus. The spine, humerus, and femur are most frequently involved. Pathologic fractures are common in the metastases from the breast, thyroid, and kidneys but rare in those from the prostate.

X-ray Features.—The bone appears mottled due to the localized destruction. There is no periosteal reaction.

Treatment.—The prognosis is hopeless, but pain can be relieved and prolongation of life secured by irradiation. Pathologic fractures are to be immobilized for the relief of pain, and the fracture sometimes unites.



Fig 707—Secondary carcinoma involving the upper shaft of femur, a metastasis from carcinoma of the breast.

TUMORS OF JOINTS

These may arise from the structures within the joint or secondarily invade the joint when the primary neoplasm arises in the periarticular tissues.

The benign tumors are *lipoma*, *fibroma*, *hemangioma*, *xanthoma*, and *endothelioma*.

Wide resection of the tumor is indicated. When complete synovectomy is required, motion is usually restricted by 25-50%. If the hemangiomatous lesion is diffuse, roentgen therapy is indicated.

Synovium is the most common malignant tumor. It is a disease of young adults and is believed to arise from a specially differentiated

type of mesodermal cell. Microscopic examination shows fibrous septa that enclose masses of spindle-shaped or polyhedral cells. The cells may line irregularly shaped spaces. Metastasis usually occurs by the blood stream. The diagnosis is made by biopsy and is followed immediately by a high amputation. The prognosis is poor, especially in the older age group.

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Chapter 43

Disorders of the Vertebral Column

Albert A. Butler, MD.

ANATOMIC CONSIDERATIONS

The vertebral column consists of 33 vertebrae; they are named in regions. There are 7 cervical, 12 thoracic, and 5 lumbar vertebrae which are movable. There are 5 sacral, fused in the adult to form the sacrum, and 4 coccygeal fused to form the coccyx. The total number of vertebrae is almost constant, but regional variations frequently occur. As one would expect, we often see transitional vertebrae which have some of the characteristics of two regions. Thoracic vertebrae show most of the common characteristics, but each region has its special form. The 1st and 2nd cervical are entirely individual. The usual parts of a vertebra are the body, the pedicles, the superior and inferior vertebral notches, the laminae, the vertebral arch, the vertebral foramen which is bounded by the vertebral arch and the back of the body, the spinous process, and the transverse processes, the superior and inferior articular processes which project upward and downward from the junction of the pedicle and lamina and have smooth surfaces for articulation with the contiguous vertebrae. In the vertebral column the vertebral foramina form the vertebral canal. The vertebral notches with the articular processes, bodies and discs, form a series of intervertebral foramina for the emergence of spinal nerves and vessels.

Cervical Vertebrae

(See Plate 107.)

Cervical vertebrae are small and show three foramina—the large vertebral foramen in the middle and small ones in each transverse process. The pedicles are short, the laminae are long, the spinous process is short and bifid, and the transverse process, enclosing a foramen, ends in an anterior and posterior tubercle. The 1st cervical vertebra, or *atlas*, has no body and no spinous process. It is formed by a pair of lateral masses united by an anterior and a posterior arch. There is an anterior tubercle for the attachment of ligaments, and the transverse processes are large for the attachment of the muscles that rotate the head. The 2nd cervical vertebra, or *axis*, is characterized by the odontoid process which articulates with the anterior arch of the atlas. The spinous process is strong and broad and bifid, whereas the transverse process is small and not bifid.

Thoracic Vertebrae

Thoracic vertebrae have facets on the bodies for articulation with the heads of ribs and most have facets on the transverse processes for the tubercles of ribs. The bodies are large, the laminae are broad, each overlapping the lamina below, the vertebral foramen is circular, the spine is long and slender, and the transverse processes are thick.

Lumbar Vertebrae

(See Plate 108)

Lumbar vertebrae show a large body, the pedicles are short and strong, the laminae are thick, and the vertebral foramen is large. The spinous process is almost horizontal, and the transverse processes are long and slender. The articular processes are large, the superior ones being concave, facing medially and the inferior, convex, facing laterally.

Sacral Vertebrae

The sacral vertebrae, 5 in number, are fused to form the sacrum, which is triangular in shape. The base is directed upward and forward, and its projecting anterior part is called the promontory. Behind the fused bodies the vertebral foramen leads into the sacral canal. The fused lateral masses form the ala of the sacrum, spreading laterally from the body. The apex is formed by the small lower surface of the body of the 5th sacral vertebra.

Coccygeal Vertebrae

The coccygeal vertebrae vary in number from 3-5 and are fused to form the coccyx, which is triangular in shape, with its base proximal and its apex distal. It consists of the rudimentary bodies of the vertebrae, more often fused than not but commonly irregular in shape.

Ossification of Vertebrae

The typical vertebra has three primary centers. One appears in each body of the lower thoracic region at about the 10th week, and two centers appear in the bodies superior and inferior to this region until present in all bodies by the 20th week, except the lower sacral ones which appear at the 30th week and the coccygeal ones which appear after birth. Two appear at the bases of the superior articular processes in the upper cervical region about the 7th week. This process extends downward, reaching the sacrum about the 20th week. Ossification extends from these two centers into the vertebral arch, processes, and lateral parts of the body. The central and lateral parts of the body begin to unite about the 3rd year

in the neck, and the process is complete in all regions by the 7th year. Laminae ossification is completed after birth, first in the lumbar region, extending upward and downward, becoming complete in the cervical about the 15th month and in the sacral about the 10th year. After union of the laminae, ossification extends into the spinous processes.

Five secondary centers appear at puberty and unite with the rest of the bones by the 25th year. There is one for the tip of the spinous process, one at the end of each transverse process, and an annular one on the upper and lower surface of the body.

Joints of Vertebral Column

The bodies of movable vertebrae are united by fibrocartilaginous discs, aided by longitudinal ligaments. In the cervical region there are also capsules of small synovial joints between their lateral parts. The articular processes are united by capsules of synovial joints. The vertebral arches are united by the intertransverse, interspinous, and supraspinous ligaments and ligamenta flava. The intervertebral discs are attached firmly to the articular cartilage which is seen on the contiguous surfaces of the bodies. The disc forms a dense annulus fibrosus in the periphery and softens toward the center, forming the nucleus pulposus. In the cervical region they do not reach the posterolateral edges of the bodies, due to the presence of small synovial joints. The anterior longitudinal ligament is thick and strong, extending from the anterior arch of the atlas to the sacrum, attaching to the fronts of bodies and to the intervertebral discs. The posterior longitudinal ligament lies in the anterior wall of the vertebral canal, from the axis to the sacrum, attaching to the backs of the bodies and to the intervertebral discs. The ligamenta flava are strong elastic sheets that connect the laminae of contiguous vertebrae, laterally blending with the synovial joint capsule. The interspinous ligaments are attached to adjacent spines from base to tip, and the supraspinous ligaments form a long continuous band connecting the tips of the spinous processes along the whole length of the vertebral column. This is thickened in the neck to

form the ligamentum nuchae, of importance because of its occipital and muscular connections.

The Vertebral Column as a Whole

The vertebral column may be considered as a central post for support of the body with a complex arrangement of its component parts, supported by strong ligaments and powerful muscles. There are four normal curves, two with their convexities forward in the cervical and lumbar regions and two with their convexities backward in the thoracic and sacral regions. These are smooth curves with a moderate variability within the range of normal, beyond which they are considered pathological, named *lordosis* and *kyphosis*, respectively. The posterior aspect of the vertebral column should present a straight vertical line, or just a trace of right thoracic curve being considered within the normal limit. Any appreciable curve is considered pathological, named *scoliosis*, and designated right or left with its region, according to the side of the convexity. There are, of course, variations with age. The child does not assume the normal curves until he is standing and walking. In old age there is a tendency for the spine to assume a smooth posterior curve throughout. Movements of the spine are carried out by a complex interaction of the enveloping musculature. Flexibility is provided by the compressibility of the intervertebral discs and the gliding action of the synovial joints. The greatest stability is in the erect posture, by virtue of the interlocking articular processes. In the cervical region anteroposterior movement is freely carried out. Lateral flexion is only possible by a combination of torsion and rotation of the vertebrae. In the thoracic region anteroposterior movement is greatly limited by the vertical position of the articular processes. Lateral flexion is much freer, and there is quite a good range of rotation. In the lumbar region flexion is free, whereas true rotation is negligible. The apparent movement is a combination of flexion and circumduction. Alterations from the normal range of appearance and function range from postural defects to traumatic and diseased processes.

Applied Anatomy

Dehydration of Nucleus Pulposus.—At an early age fibrocartilage invades the nucleus pulposus and there is an associated loss in water content with consequent reduction of its resilience. This causes an irregular distribution of stress, which also occurs when the nucleus protrudes through the hyaline cartilage plate or through the annulus fibrosus. The anterior portions of the bodies have less support than is found posteriorly, because of the presence of pedicles, and therefore show more involvement.

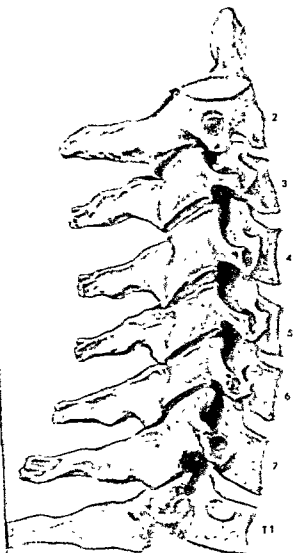
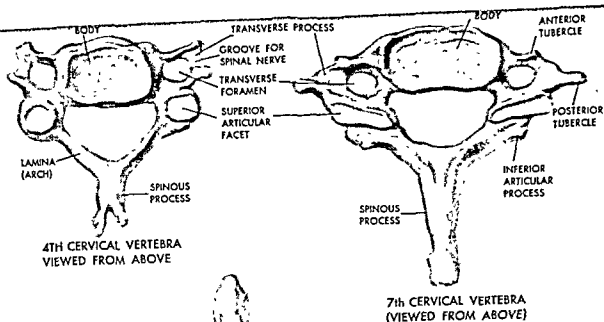
Adolescence.—Increased pressure anteriorly causes retardation of growth, fragmentation of the epiphyseal ring, and wedging of the vertebrae. This is called *Scheuermann's disease*.

Adults.—Reduced efficiency of the discs results in reactive new bone formation with peripheral osteophytosis especially marked laterally. Osteophytes are evidence of disc degeneration but do not necessarily produce symptoms. In senility, the discs may ossify peripherally, fusing the bodies, with wedging of the vertebrae as seen in senile kyphosis. If the vertebrae are atrophic and the discs relatively normal, the discs may expand symmetrically as seen in radiologic studies of senile osteoporosis.

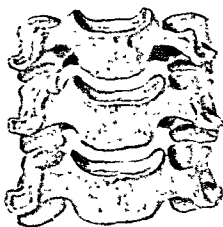
Hyaline Cartilage Plates.—Damage to the hyaline plates may permit the nucleus to enter into the bone of the vertebral body, which reacts by producing a ring of sclerosis around the intruded nuclear material. This may be demonstrated by x-ray, and such a lesion is called a *Schmorl's node*.

Adults.—Damage to the hyaline plate permits the nucleus to enter the bone, but it is not walled off as occurs in adolescence. Instead, trabecular absorption takes place and collapse follows. This is seen when the x-rays at the time of injury are normal but subsequently show collapse and wedging of the vertebral body. This traumatic disorder is known as *Kummell's disease*.

Annulus Fibrosus.—Injury to the annulus permits early or late extrusion of the nucleus and causes symptoms characteristic for the region involved. In the cervical region it is common to find radiation with occipital head aches and interscapular, shoulder, and arm



THE 2ND TO 7TH CERVICAL VERTEBRAE
VIEWED FROM THE RIGHT SIDE



THE 3RD, 4TH and 5TH
CERVICAL VERTEBRAE
VIEWED FROM IN FRONT

F. Netter M.D.

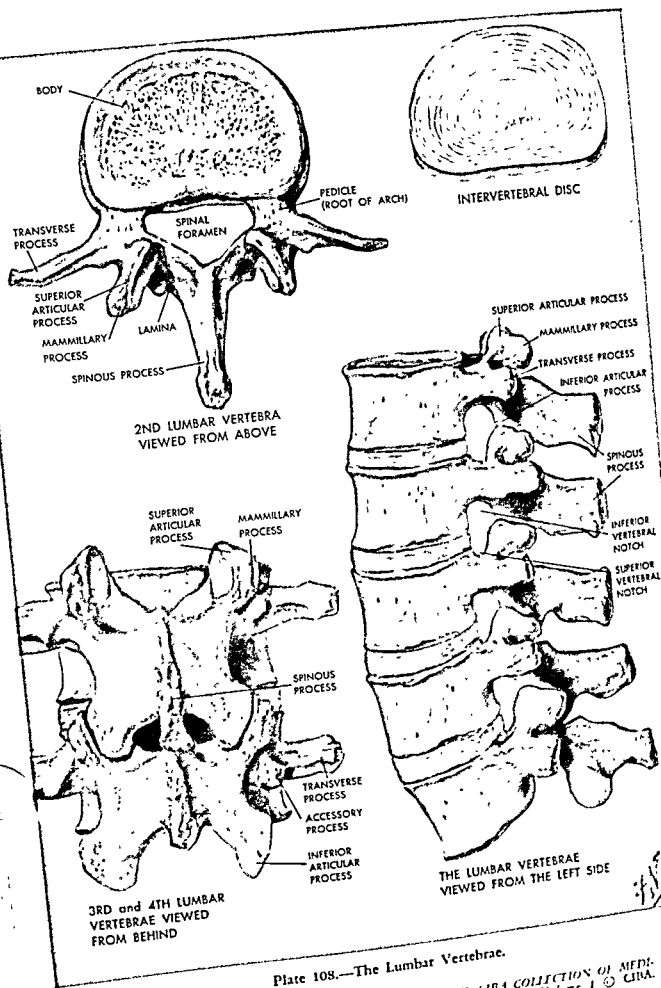


Plate 108.—The Lumbar Vertebrae.

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pain in the *thoracolumbar* region it may give rise to radiating pain simulating visceral disease. In the *lumbosacral* region it is the commonest cause of sciatica

BACK PAIN

Back pain is a common symptom in a multitude of disorders, and careful assessment is always necessary. There is a considerable divergence between severity of pain and objective findings. Often there are severe symptoms in the early stages of a lesion when objective findings are elusive, or again there may be gross objective signs of disorders which are well compensated for and therefore do not give rise to much pain. The former are frequently labelled psychosomatic disorders when in fact there is a definite organic lesion which could be recognized on more careful examination. Positive x-ray findings are often of great value, but it must be remembered that the vertebral column is largely composed of cancellous bone, and reactive changes must be extreme before a radiologic diagnosis can be made.

A complete medical history is important. This must be correlated with a detailed description of the spinal symptoms from time of onset throughout periods of remission and exacerbation, their relationship to bed rest, sitting, walking, lifting, straining, twisting, and other activities, effect of the weather, the menstrual cycle, and relationship to other illnesses.

Physical examination should be carried out with the patient undressed or with a minimum of underclothing. A complete routine should be followed, noting positive and negative findings. This examination should elicit body type, muscular development, general posture, deformities, location of tenderness or pain and its radiation, all movements of the spine and its extremities, with reflex, motor, and sensory testing, and with abdominal and rectal examination. The patient is examined standing, sitting, lying supine, and lying prone.

In scoliosis, flexion accentuates a structural curve but causes a postural one to disappear. In Pott's disease localized tenderness, spinal rigidity, and an angular kyphosis may be noted. Hyperextension tests the hip and sacroiliac and

lumbosacral joints, being positive according to location of pain. Rotation or lateral compression of the pelvis strains the sacroiliac joint and may indicate a disorder there. Straight leg raising is limited in sciatica, and the pain is further aggravated by dorsiflexion of the foot (Lasègue's sign). Short hamstrings cause a painless limitation of straight leg raising, and dorsiflexion of the foot is not painful. Joint crepitus is most easily elicited with forced rotation in the cervical region and may indicate osteoarthritis in the facet articulations. Clicking of the hip or sacroiliac or lumbosacral joint may be a normal variant or of clinical significance.

It is usually necessary to supplement the examination by radiologic studies of the region clinically indicated. Hemograms, blood chemistry, and other tests may be required, depending on one's provisional diagnosis.

Classification of spinal disorders is an aid to diagnosis and should be both anatomic and etiologic.

1 Developmental or Congenital Defects

- Hemivertebrae
- Synostosis
- Spina bifida occulta
- Spondylolysis
- Spondylolisthesis
- Kissing spines
- Transitional vertebrae
 - (a) Cervical rib
 - (b) Sacralized 5th lumbar vertebra
 - (c) Lumbalized 1st sacral vertebra
- Congenital torticollis

2 Postural Defects

- Lumbar lordosis
- Sway back
- Round back
- Flat back
- Round shoulders
- Postural scoliosis

3 Traumatic Disorders of the Vertebral Column

- Myofascial injuries
- Lumbosacral and sacroiliac strains
- Intervertebral disc protrusions
- Intervertebral disc traumatic degeneration
- Fractures and dislocations

4 Static Disorders of the Vertebral Column

- Fibrositis
- Osteoarthritis of the spine
 - (a) Hypertrophic spondylitis
 - (b) Hypertrophic spondyloarthritis
- Ankylosing spondylitis
- Coccydynia
- Adolescent kyphosis
- Senile kyphosis

Senile osteoporosis
 Vertebral osteochondritis
 Structural scoliosis

5 *Infectious Disorders of the Vertebral Column*

Tuberculosis of the spine
 Osteomyelitis
 Gonorrheal arthritis

Chapter 11, Neurosurgery.) Radiologic studies have shown that about 30% of all spines show some variation from the normal due to faulty development. Some of these anomalies are incompatible with life and others are of little or no consequence.



Fig. 708.—Multiple congenital anomalies showing hemivertebra at L1

Syphilitic spine
 Typhoid spine
 Undulant fever
 Actinomycosis
 Blastomycosis
 Echinococcus cysts

6 *Neoplastic Disorders of the Vertebral Column*

DEVELOPMENTAL OR CONGENITAL DEFECTS

The most frequent congenital anomaly of the vertebral column is spina bifida. (See

Hemivertebrae

The segments of cartilaginous vertebrae in the embryo may fail to unite, and two centers of ossification appear instead of one. Different rates of growth in the two segments may give rise to a congenital scoliosis; persistence of the separation between the two segments may be followed by an anterior spina bifida. Development in one segment only produces a true

hemivertebra, resulting in a sharp lateral curvature of the spine.

Synostosis

If two adjacent vertebral bodies fuse during their development, a synostosis or congenital block vertebra results. The Klippel-Feil syndrome is a synostosis of lower cervical vertebrae, associated with spina bifida, producing a short and rigid neck. Occipitalization is a synostosis of the atlas with the occiput. Sacralization is a fusion of the 5th lumbar with the sacrum.

be of no consequence, but approximately 20% of them do show some pes cavus deformity or other neuromuscular anomaly of the lower extremities. Often there is limitation of straight leg raising, due to shortening in the hamstring muscles, and often there is an equinus of the foot or at least a limitation of dorsiflexion, due to shortening of the calf muscles.

Spondylolysis

This is a defect in the neural arch, occurring between the facet articulations. With this defect in the pars interarticularis there is only soft



Fig. 709—Synostosis of lower cervical vertebrae

Spina Bifida Occulta

Spina bifida occulta as opposed to the true spina bifida shows little or no neurologic involvement. There is usually a defect in the neural arch of several vertebrae, the commonest location being in the lumbosacral region. Brailsford gives an incidence of 6% in the 5th lumbar and of 11% in the first two sacral segments. These defects are commonly stated to

tissue stabilization at this level of the spine. Displacement is accompanied by soft tissue separation at the defect, as well as at the intervertebral disc, and associated ligaments. It may produce a traction lesion by the nerve roots being dragged against the vertebral notch. This displacement is known as spondylolisthesis and is commonest in the 5th lumbar vertebra.

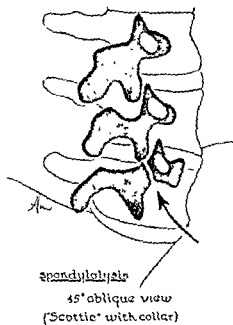


Fig. 710.—Spondylolysis showing defect in pars interarticularis

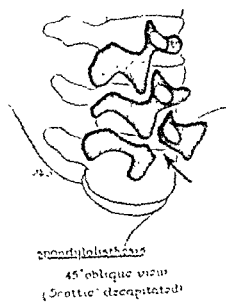


Fig. 711.—Spondylolisthesis showing displacement at the pars interarticularis

Spondylolisthesis

In spondylolisthesis 90% of the defects occur at the 5th lumbar, most of the others involve the 4th lumbar; they are rarely seen elsewhere. In some the displacement occurs soon after birth. Freiberg believes that most of the displacements occur about 12 years of age. Undoubtedly, many do not occur until there is some unusual stress on this segment of the spine. This might follow heavy weight lifting,

of slipping occurring during certain movements. The most extreme spondylolisthesis seen showed lumbar 3, 4, and 5 lying in front of the sacrum. There was no evidence of neurologic involvement; in fact, the patient refused treatment once his curiosity was satisfied as to why his ribs were resting on the pelvis. It is usual, however, for the displacement to be less than a complete dislocation. Traumatic spondylolisthesis, as a major hyperextension injury

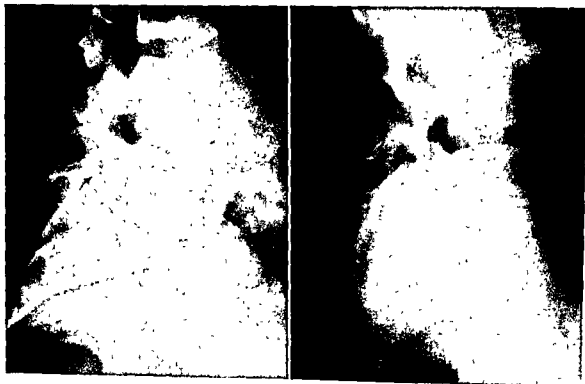


Fig 712—Spondylolisthesis showing forward displacement at L5-S1

or some illness with muscular impairment, or obesity with loss of muscular power after giving up athletic activities. Adaptive changes in the shape of the vertebral body would serve to indicate that the displacement had occurred during the growth period. Both spondylolysis and spondylolisthesis commonly give rise to the symptoms that we associate with ligamentous strain. It is not unusual for them to be entirely symptomless by virtue of adaptive changes in the ligaments and compensation by powerful muscle control. Most of the displacements are rigid in their position of deformity, but occasionally a good deal of instability can be demonstrated, and the patient may be conscious

producing a fracture-dislocation along the same path as the usual congenital defect, is a rare occurrence.

Clinical Features—The majority of patients do not complain of symptoms until well into adult life. It is often difficult to determine just when the displacement did occur. Usually they give a history of some fairly recent injury, to which they attribute their trouble. They are often incredulous when told that it is congenital in origin. Pain is not constant but is associated with certain movements, particularly stooping and lifting. Usually there are no neurologic changes, but occasionally a typical

sciatic syndrome is present. When the displacement is appreciable, there is a sharp depression above the 5th lumbar spinous process which is increased with hyperextension. The lumbar region may appear shortened and the sacral region appear lengthened. If there is marked displacement in a pregnant woman, it may constitute an obstruction to normal delivery. The condition is most clearly seen in oblique x-rays, although if there is significant displacement, it is well seen in the lateral view.

Treatment.—Many of these patients are free of symptoms, due to the natural compensation of powerful musculature. It is not surprising, therefore, that many of those who do have symptoms are relieved by exercises which develop the erector spinae. Strain also is reduced by development of the hip extensors, enabling them to diminish the pelvic inclination and flatten the lumbar lordosis. These measures may be aided by the use of a lumbosacral corset or brace, which reduces the painful movements. If pain is severe, or if freedom from restrictive measures is desired, or if there is increasing displacement, spinal fusion is indicated. Reduction of the displacement may be obtained, when it has occurred recently, by the method described by Watson-Jones. This consists of vertical traction on the flexed hips with the shoulders resting on a table, so that the pelvis is drawn forward against the body weight, if necessary supplemented by the downward traction of a sling around the abdomen. If correction is obtained, a plaster spica is applied and posterior spinal fusion carried out through a window in the cast. In the presence of a rigid deformity, operative reduction is a formidable procedure and is not worth while. Spinal fusion should be carried out in the position of deformity. Anterior fusion of the 5th lumbar body to the sacrum by an abdominal approach appears mechanically preferable, but the mortality rate has been excessive. Posterior spinal fusion is considered the method of choice. The area of fusion is subjected to a good deal of shear. Therefore the method used should produce a massive area of fusion, and it should be protected until it is mature. This reduces the very real risk of pseudarthrosis. The complete Hibbs procedure, with excision of the interarticular joints, and the loose vertebral arch and the fibrous masses over the nerve roots, sup-

plemented by a massive "H"-shaped iliac graft, seem to fulfill these requirements.

Kissing Spines

This is a developmental variation in which there is contact between deformed spinous processes, giving rise to pain, and usually limitation of extension of the spine. An x-ray of the spine which gives good detail in the vertebral body region does not show the spinous processes so well and requires close inspection. There may be a false joint or condensation of bone at the points of contact. A similar condition is seen as a complication of spinal fusion. If the surgeon fails to preserve the soft tissue between the spinous processes at the limits of his fusion, then the graft or subsequent callus may impinge on the adjacent spinous process with spinal movements and give rise to pain of the same type. A cure of both conditions is simply to excise the offending bony protuberance and suturable soft tissue between them.

Sacralized Transverse Process of the 5th Lumbar Vertebra

This is a congenital variation from the normal, in which there is enlargement of one or both transverse processes. It (or they) articulates with the sacrum or sacrum and ilium. The term transitional vertebra is used to indicate that it has some characteristics of both lumbar and sacral segments. Often the articulation is structurally poor and suffers traumatic or degenerative change readily. This will produce pain on the same side. Commonly, however, pain is on the opposite side. This is probably due to strain on the weaker side. With movement to the side of the bony buttress there may be leverage against the ligaments on the opposite side. This would produce recurrent ligamentous injury. The 4th lumbar nerve passes over the prominent transverse process. With spinal movement may give rise to a traction lesion. If the enlarged transverse process is just short of articulating with the ilium, it may, with spinal movement, give rise to interfering soft tissue and give rise to traumatic inflammatory changes. It could also produce an adventitious bursa, with recurrent bursitis.

Cases have been described in which osteophytes from an arthritis in the false joint have encroached on the intervertebral foramen, affecting the emerging nerve root. The counterpart of this syndrome occurs with lumbarization of the 1st sacral segment. One must recognize that this anomaly may be present but the symptoms are due to one of the other numerous causes of low back pain. For instance, the intervertebral disc immediately above is rendered more vulnerable and may be the lesion requiring treatment. Thorough investigation is essential.

3. The maintenance of powerful musculature by daily remedial exercises is designed to prevent recurrences.

Operative.—Indicated if adequate conservative treatment fails or if heavy use of the back is necessary.

1. *Transversectomy* is indicated if one is certain this anomaly accounts for the symptom complex. It allows early mobilization of the spine with a short convalescence.

2. *Spinal fusion* is often preferable as it strengthens the spine. It will also cure the



Fig. 713—Sacralized transverse process of 5th lumbar vertebra

Treatment.—

Conservative—

1. *Bed rest* is indicated at the start of an acute exacerbation of symptoms.

2. *Limitation of painful movement* is achieved with a Harris brace or a plaster corset.

3. *Novocain infiltration* is often of diagnostic and therapeutic value.

4. *Deep heat, massage, and exercises* are used as the acute phase subsides.

symptoms of almost any low back disorder associated with this condition.

Cervical Rib and Scalenus Anticus Syndrome

(See Chapter 11, Neurosurgery, and Chapter 33, Peripheral Vascular Diseases.)

Congenital Torticollis

There are many causes for *wryneck*—hysterical, habitual, ocular, bony, muscular, and nerve lesions. It may be associated with con-

genital, paralytic, traumatic, or inflammatory changes

The congenital variety is due to a retardation in lengthening of the sternomastoid muscle. The disturbance is probably due to interference with the arterial blood supply—analogous to a Volkmann contracture.

Clinical Picture.—Soon after birth a firm lump may be felt in the sternomastoid. This disappears spontaneously, but an observant mother may notice that there is some restriction of neck movements. Gradually a deformity develops due to the related shortening of the muscle. There is a flexion of the head to the

Conservative treatment consists of passive stretchings. The mother is trained in the technique and carries it out three times daily. Clinical review is essential at frequent intervals. It should be discontinued unless effective.

Operative treatment is indicated with the appearance of any appreciable deformity. The sooner it is done the better. It consists of an open division of the sternal and clavicular heads of the sternomastoid muscle. Postoperative splintage in the overcorrected position is used till soft tissue healing is consolidated. This is followed by postural exercises and passive stretchings. Review at suitable intervals up to



Fig. 714—Congenital torticollis

side of the lesion and a rotation of the face to the opposite side. If the condition is not treated, asymmetry of the face develops. There is ocular adjustment to the position of deformity. This becomes a problem in late corrections. There may be secondary structural changes, giving rise to an upper thoracic scoliosis and elevation of the shoulder. These changes constitute a very ugly deformity. Double torticollis and posterior torticollis are rare. The latter is due to similar changes in the trapezius and other posterior muscles.

Treatment.—It appears that some infants develop the lump in the sternomastoid muscle and go on to complete and spontaneous recovery. If, however, any of the other signs develop, it is imperative to commence early treatment.

adolescence is advisable. Any recurrence should be prevented or corrected before it develops into a real disability.

POSTURAL DEFECTS

Postural defects are those which cannot be attributed to any traumatic or disease process and which can be corrected by voluntary efforts. They are often associated with postural defects elsewhere in the body. Form and function are interrelated, so that during the growth period a postural defect may be converted into a structural one. Also, conversely, during the growth period minor structural defects may be corrected by the development of a good posture. While in the adult the plasticity of bone is not entirely lost, these changes be-

come negligible. The natural tendency, nevertheless, to compensate for a structural defect can be enhanced by the development of powerful musculature. The maintenance of a poor posture is probably a function of the central nervous system. A habit produces a conditioned reflex, which maintains the altered muscle length as if it were normal. It is common to have weak muscles associated with a faulty posture, but this is not necessarily so. The conscious effort to maintain a correct posture until it becomes a conditioned reflex is probably the most important feature in the treatment

the hip flexors and decreased by the hip extensors, any alteration affecting the entire posture of the spine.

Lumbar Lordosis

Lumbar lordosis is a deviation from normal posture, in which the pelvis is tilted forward. In order to maintain the line of weight-bearing, it is necessary to have an increased concavity in the lumbar region. There is perhaps a little increase in the thoracic kyphosis, but this is not a prominent feature. The whole appearance is

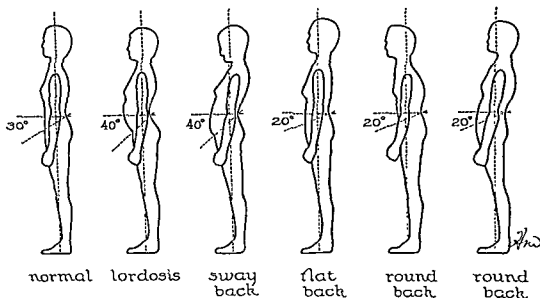


Fig 715—Types of postural defects

It is apparent that any alteration in the weight-bearing joints may affect spinal posture. It may be a valgus foot with external rotation of the limb and tilting of the pelvis and therefore of the spine; or it may be primarily in the hip, pelvis, or the spinal column. There are various methods of measuring pelvic inclination, but the range of normal variation is wide enough to depend on careful observation. An instrument called a *pelvic inclinometer* permits a reading of pelvic inclination from the upper part of the symphysis pubis to the level of the posterior superior spines of the ilium. The instrument has two blades to be placed centrally at these levels and an attached protractor with a plumb line to give the reading. The inclination of the pelvis is increased by contraction of

altered by correcting the pelvic tilt, e.g., tightening the gluteal and abdominal muscles.

Sway-Back

Sway-back is a combination of forward tilting of the pelvis and a dorsolumbar kyphosis. In this disorder, instead of correcting the line of weight-bearing by increasing the lumbar concavity, the entire spine is bent backward, rather markedly at the lumbosacral level. The pelvis projects forward, with the body and the legs sloping backward.

Round Back

Round back is characterized by a decreased pelvic inclination in combination with a dorso-

lumbar kyphosis. With this, the line of gravity may be corrected by the trunk being held forward, producing obliteration of the lumbar curve. It may also be corrected by bringing the pelvis forward and angulating the trunk backward at the lumbosacral level, producing an appearance somewhat similar to the sway-back posture.



Fig. 716—Fangue posture (sway back).

Flat Back

The flat back is produced by the pelvic inclination being reduced and is balanced by a reduction in the lumbar curve. This type of variation from the normal is less noticeable than the others, but it does give rise to trouble in later life and should be corrected.

Round Shoulders

This is a mixed type of postural defect which the scapular muscles sag, allow shoulders to fall downward and forward further aggravated by some increase thoracic curve and forward inclination head and neck

Postural Scoliosis

This is a functional lateral curvature can be fully corrected by voluntary effort is in the same category as other postural defects and does not give rise to serious structural changes. There is usually a smooth transition of the lumbar and dorsal regions, with no rotation of the vertebrae and no secondary changes in the thoracic cage. The curvature appears when the spine is flexed. It soon disappears when sitting and can be corrected by muscular efforts when standing. It is intermittent and associated with fatigue. It is distinguished from the lateral curvature which occurs with a short extremity. Variations in leg length up to $\frac{1}{4}$ " are of no consequence and do not require correction. Children who show a postural scoliosis will usually correct it spontaneously with growth and muscular development. It is, however, wise to check on the child's general health and to teach postural exercises.

Treatment of Postural Defects.—On the mechanism of a postural defect is understood then the exercises necessary to correct easily determined. Advantage is taken of reciprocal innervation of antagonistic muscle groups. If an extensor group of muscles is contracted in carrying out a movement, the group relaxes sufficiently to permit the movement but contracts sufficiently to control the joint. If the extension is against resistance control of the joint is obtained between two forces, and the reflex inhibition of flexors is much more profound. If, then, we wish to correct the increased pelvic inclination of a lumbar lordosis or a sway-back defect, we should develop the gluteal muscles and relax the hip flexors. Gluteal resistance exercises are therefore prescribed. If an exercise is carried out from a correct postural position and returns to this position, and is repeated

frequently enough, the normal postural reflex is re-established. It is not sufficient to simply build up muscular power by general exercises. It is important then to utilize the normal posture in all the daily activities, until it is finally maintained without conscious effort.

TRAUMATIC DISORDERS OF THE VERTEBRAL COLUMN

The complex arrangement of the vertebral column is susceptible to a wide range of injuries: muscular, ligamentous, discoid, cartilaginous, and bony, as well as neurologic. Pain arising in any of these structures may be referred to the skin distribution of the segmental innervation of the part concerned. It is accompanied by tenderness and muscular rigidity, and it is well to bear in mind the false localization and reflex muscular effects which may occur from the involvement of deep-lying somatic structures and also of certain viscera. Sciatica, for instance, may arise reflexly from a gluteal muscle lesion, a lumbosacral ligamentous lesion, or osteoarthritic changes in the facet articulations, as well as from direct pressure on the nerve root by protrusion of an intervertebral disc.

Myofascial Injuries

These injuries may produce a sciatic scoliosis with lumbosacral tenderness, occasionally tenderness along the course of the sciatic nerve, limitation of spinal movement (most marked limitation being of flexion), and limitation of straight leg raising. The attachments of the injured muscles are particularly tender, and there may be moderate tenderness over all of the muscle groups. The symptoms are relieved by heat and rest and are aggravated by activity. Novocain infiltration of the points of maximum tenderness is useful both as a test and for relief of symptoms. It is considered confirmatory when insertion of the needle aggravates both the local and referred pain, and the injection of the local anesthetic relieves both of these and is followed by the restoration of straight leg raising. Occasionally one sees systemic reactions, characterized by giddiness, rapid pulse, sweating, and a drop in blood pressure. If

severe, these symptoms may be relieved by the injection of 1 ml. of 1/1,000 Adrenalin. The antihistamine drugs are ineffective. Also, there may be a delayed painful reaction as the effect of the local anesthetic disappears, so that for a while the pain is far worse than the original one. This may be relieved by the use of analgesics. Treatment should have as its object the healing of damaged tissues with the prevention of extensive adhesions. Therefore, in the acute phase, rest and support of the part to relieve pain are indicated. Depending on the severity of symptoms, this may be achieved by strapping, a tight corset, a brace, or plaster jacket. Bed rest in the early stages is often advisable. Radiant heat and light massage are soothing in their effects. Short-wave diathermy and heavy massage will aggravate the condition, although they are often useful with active exercises as the acute phase subsides.

Chronic strain or improperly treated acute injuries may result in myofascial adhesions. They give rise to recurrent low back pain and sciatica, often with acute exacerbations. In some of these there is contracture of the gluteal fascia below the posterior superior spine. Roberts and Heyman have advised stripping of the posterior superior spine. In others, the contracture is more anterior in the tensor fascia lata and the iliotibial band. This may be demonstrated by the Ober test, which consists of having the patient lie on his good side and flexing the hips to flatten the lumbar spine. The affected leg is flexed to a right angle at the knee while the hip is steadied and then is abducted and extended, bringing the thigh in line with the trunk. The abduction contracture of the iliotibial band can then be felt as it maintains the limb in the abducted position. Ober has recommended division of the iliotibial band and fascia lata with the intermuscular septa for relief of this condition. When the original symptoms were due to involvement of the erector spinae muscles and there is no abduction contracture of the hip, manipulation of the spine is often recommended. Due, however, to the danger of displacing an intervertebral disc, this is considered a dangerous procedure. It also appears that in the vast majority of these conditions, an equally satisfactory result can be obtained by conservative measures. These consist of Novo-

DISORDERS OF THE VERTEBRAL COLUMN

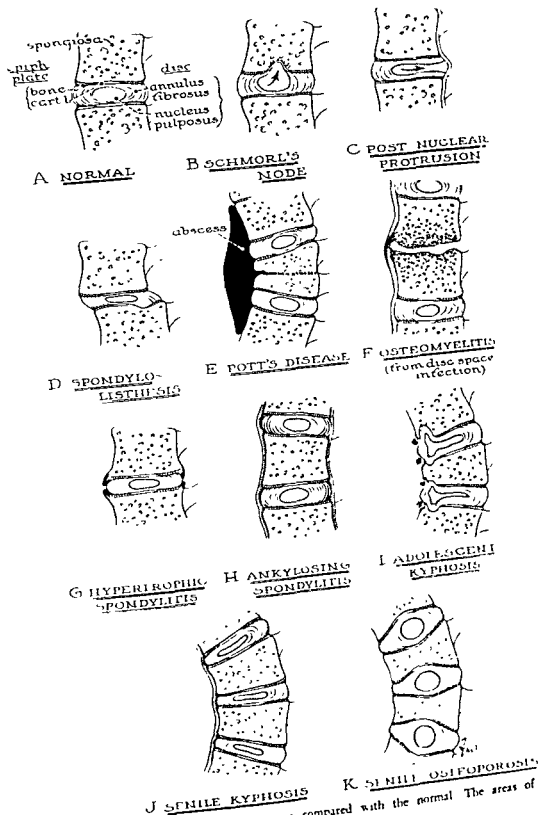


Fig 717—Varieties of disc involvement as compared with the normal. The areas of lighter stippling connote osteoporosis.

cain infiltrations, massage, short-wave diathermy, and spinal exercises, sometimes supplemented by the use of a support.

Lumbosacral and Sacroiliac Strains

The ligaments supporting the lumbosacral and sacroiliac articulations are so powerful that it must be indeed rare to have an actual tearing of these structures. Ligaments, however, do not stand up well to continuous strain, and with muscular insufficiency and postural defects, they frequently give rise to symptoms. There is protective muscle spasm, with pain both local and referred, which may be present in varying degrees according to the severity of the strain. Usually there is relief of symptoms with bed rest, which continues into the morning, but the symptoms recur with the onset of muscular fatigue. A minor injury will often produce an acute exacerbation of the symptoms. Testing the joints while symptoms are present will produce an exacerbation, but often if examined in a period of remission, localization cannot be determined. The history of mechanism of injury may be of value, flexion injury being likely to affect the lumbosacral articulation, and twisting strains the sacroiliac joint. The latter is very powerful except during pregnancy, when, due to the hormone *relaxin*, the pelvic ligaments are relaxed. The sacroiliac joint may be tested by having the patient lie on his side and compressing the iliac crests, also lying on the back and forcing down on both anterior superior spines; or lying on the back, hyperextending one hip over the examining table. If positive, each of these tests reproduces pain and tenderness at the sacroiliac level. With the patient lying prone, hyperextension tests both joints, being considered positive according to the localization of the pain. Pain with flexion is often well localized in lumbosacral strains. Rotation tests are not very useful but, if positive, point to the sacroiliac joint. The vast majority of so-called sacroiliac strains are shown by these tests to be lumbosacral ones. Treatment is the same as described for myofascial injuries, except that Novocain infiltration is much less useful, and very occasionally for persistent cases a fusion of the affected joint may be required.

Intervertebral Disc Protrusions

There are two common regions for intervertebral disc protrusions to give rise to symptoms. The lower lumbar L4-5 and L5-S1 account for probably 90% of disc protrusions. The lower cervical C5-6 and C6-7 account for most of the others, the former being twice as frequent. There are many more symptomless disc protrusions than ones that give rise to trouble. Myelograms suggest a proportion of 4:1, but anatomic dissections suggest it is very much higher. It is obvious that a "proved disc" is not proof that it causes the symptoms. All other causes for the symptom complex must be considered in the clinical appraisal. There are many vertebral disorders in which some discoid anomaly is associated. These are described elsewhere in this section—see:

- 1 Spondylolisthesis
- 2 Pott's disease
- 3 Osteomyelitis of the spine
- 4 Hypertrophic spondylitis
- 5 Ankylosing spondylitis
- 6 Adolescent kyphosis
- 7 Senile kyphosis
- 8 Senile osteoporosis

The modern glib diagnosis of "disc" for almost every vertebral column disorder has, therefore, a grain of truth in it. It is, however, a retrograde development. The term should be reserved for the clear-cut symptom complex associated with extrusion of the nucleus pulposus or herniation of the annulus fibrosus into the vertebral canal, causing cord or nerve root irritation. The rarer nuclear prolapse or sequestered disc is likely to give rise to intractable nerve pain or cord symptoms and usually requires operative measures. The commoner, herniation of the annulus, is usually reversible, gives rise to intermittent symptoms, and responds to a variety of treatments.

Cervical Discs

If the protrusion is central and large enough, it produces a cord lesion resembling a cord tumor. Operative removal is imperative in order to prevent permanent damage. Fortunately these midline massive extrusions of the nucleus are rare.

Commonly the protrusion is lateral, and involvement of the nerve root is variable and usually reversible.

Clinical Features.—Some history of trauma, recent or remote, can usually be established. One would expect extrusion of the nucleus if symptoms were persistent from the time of injury. The vast majority of cases show a considerable time interval between the injury and the onset of symptoms. There is a predilection to the middle-age group, so that degeneration undoubtedly plays an important part. It is a traumatic degenerative change in a disc which has probably lost some of its resilience. The fluid content of an intervertebral disc decreases with age, and this dehydration is appreciable at about 40 years of age.

process at the involved level helps localization. The affected dermatome can be mapped out if there is objective sensory change.

A disc protrusion C5-6 is likely to produce sensory changes in the thumb and index finger. There may be loss in power of elbow flexion. The biceps jerk is diminished or absent.

If the involvement is at C6-7, the middle and ring fingers may show paresthesia. There will be loss of power of extension in the elbow, and the triceps reflex is diminished or absent.

X-ray examination may be normal in a recent involvement. When the condition has been present long enough for adaptive changes to occur, we expect to see narrowing of the intervertebral space. Often if films are taken in flexion and extension, an alteration in the

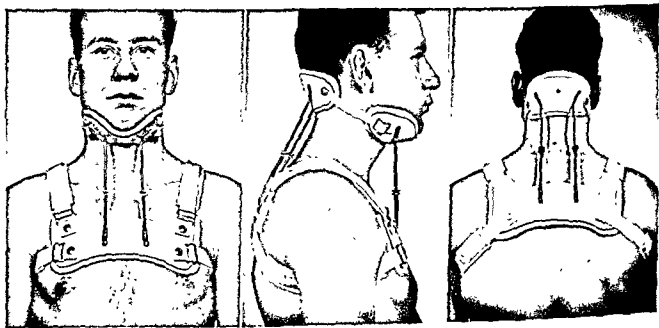


Fig. 718—Cervical brace shown in anterior, lateral, and posterior views

Limitation of movement due to pain is seen early. There is referral of pain according to the segment involved. Jarring of the spine in any way is likely to aggravate symptoms. Maneuvers which increase cerebrospinal fluid pressure produce a sharp stab of pain, e.g., coughing, sneezing, and straining. There are troublesome subjective symptoms of numbness, pain, and coldness, before sensory involvement can be demonstrated. Muscular weakness may be detected by comparison of power with the unaffected limb. Tenderness of the spinous

curve can be detected. There is loss of mobility at the level of a disc lesion. Further progression will result in peripheral osteophytosis, and if this projects posteriorly, it is direct evidence of encroachment on the vertebral canal.

Treatment.—

Conservative.—It is generally recognized that a cervical disc protrusion responds well to conservative measures. These are as follows:

1. Short-wave diathermy and massage to relieve the secondary muscle spasm and pain

2 Analgesics and sedatives for the same reason

3. Head traction to reduce the herniation of the annulus; this may be by a small weight for a long period or a larger weight for short periods. A usual prescription is 20 pounds for 20 minutes, three times weekly for 3-4 weeks. Occasionally a 5-pound weight is used almost continuously for a week, in bed, and succeeds where the first has failed. A well-fitted head halter with ropes, pulleys, and weights is used.

4 Cervical splintage, this may be useful to limit painful movement, and there is a variety of methods: (a) plaster "doll's collar," (b) molded leather collar, (c) plastic mold collar, (d) high felt collar, etc.

dous interest in the subject since the early work of Mixter and Barr in 1934. Prior to that time the condition was not commonly recognized. It is probably now being diagnosed too frequently on insufficient evidence. The whole problem of low back pain must be understood in order to place the component parts in a proper perspective.

The lumbar discs at the lower two segments are subject to a good deal of stress, just as are the other structures at this level. When there is a nucleus pulposus extrusion, symptoms of nerve root irritation are usually severe and persistent. Bulging of the annulus fibrosus, which viewed from the vertebral canal aspect would give a washboard appearance, is often of much less importance. The former condition

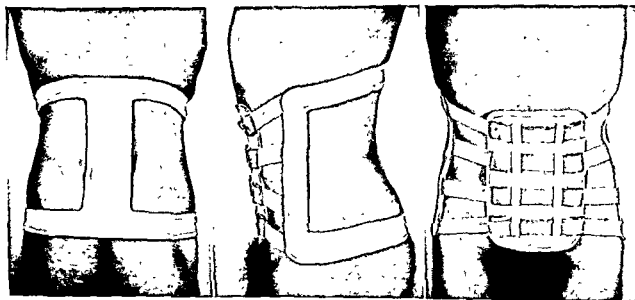


Fig. 719—Harris brace shown in posterior, lateral, and anterior views. Especially used for lumbosacral lesions.

These measures should clear up symptoms in 90% of cases. Permanent relief is obtained by developing powerful cervical musculature and avoiding trauma to the region.

Operative—Operative treatment is as described for lumbar discs. The cord must be carefully protected and a careful technique is required.

Lumbar Discs

The involvement of lumbar discs is currently believed to be the commonest cause of low back pain. Certainly there has been a tremen-

ous interest in the subject since the early work of Mixter and Barr in 1934. Prior to that time the condition was not commonly recognized. It is probably now being diagnosed too frequently on insufficient evidence. The whole problem of low back pain must be understood in order to place the component parts in a proper perspective.

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Clinical Features.—Symptoms fall into two main categories: those of gradual onset, which appear compatible with a degenerative lesion, and those of sudden onset, which suggest a traumatic lesion. Obviously, there are many variable factors and combinations of modes of onset. If there is a central protrusion, one finds bilateral involvement of the lower extremities, and the lesion may be at a higher level than expected. As localization clinically is

based on the level of nerve root emergence from the vertebral canal, a central protrusion may affect it a little higher up. The more common lateral disc protrusion is more accurately localized by the dermatome involvement. This is one of the many causes of the symptom complex referred to as *lumbago* or *sciatica*. It is probable that some cases with severe subjective complaints of this nature are minimal disc protrusions, but it is a speculative diagnosis which should not be made. Other more inclusive terms are more in keeping with the

and flexion of hip and knee on the affected side. Straight leg raising is limited on both sides but much more on the affected side. Lasègue's test is positive. It consists of flexing the knee and bringing the hip to a right angle. Then as the knee is extended, pain is produced short of full extension. If at this point the foot is dorsiflexed, there is further aggravation of the pain.

There is tenderness of the regional spinous processes, often maximal at the level of the lesion.

Hypesthesia or paresthesia should be mapped out and aids localization according to the dermatome pattern.

Muscle weakness is a common finding, particularly extensor hallucis longus in L5-S1 protrusions. The knee jerk is diminished or absent in L3-4 disc lesions, the ankle jerk in L4-5 and L5-S1 protrusions. This, however, is not entirely reliable. Due to variation in innervation, there is an error in localization in about 20% of individuals. Myelograms are also only accurate in about 80% of cases. However, when myelographic studies are correlated with the clinical findings, errors in localization are reduced to around 5%.

Rectal examination is done to exclude intrapelvic neoplasm as a possible cause for sciatica.

X-rays are of considerable value, both for confirmatory and exclusion evidence. There may be no changes in a recent protrusion, but later there is reduction of the interspace, perhaps loss of mobility in bending films, and possibly posterior osteophyte encroachment on the vertebral canal. Anteroposterior, lateral, and oblique films, at least, should be taken to help rule out congenital, infectious, and static disorders.

Myelogram is a special type of x-ray study in which 3-9 ml of Pantopaque are injected into the vertebral canal by ordinary lumbar puncture. By changing positions of the tilting table on which the patient lies, this can be gravitated as desired to outline the vertebral canal. Any encroachment by a disc protrusion is recognized by a filling defect. If the findings are compatible with the clinical picture, it is considered significant. Symptomless protrusions thus demonstrated are disregarded. The test is restricted to cases in which operative treatment is contemplated.

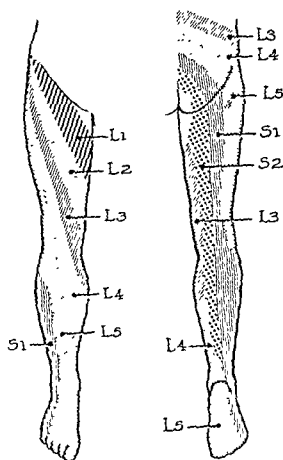


Fig 720 -The dermatomes of the leg (After Keegan)

probabilities and less likely to lead to misinterpretation. If there are motor, reflex, or sensory changes, then the probability of a protrusion is greater.

With *lumbago*, regardless of its cause, there is spasm of the erector spinae, flattening of the lumbar lordosis, and painful limitation of movement. In *sciatica* there is a scoliosis due to unequal muscle spasm in the lumbar muscles

Treatment.—

Conservative.—

1. Short-wave diathermy, massage, and flexion exercises often suffice to clear up mild attacks.

2. Analgesics, sedatives, and local anesthetic infiltration of points of maximal tenderness aid the symptomatic relief.

3. Bed rest may offer a good deal of relief. Fracture boards under the mattress may give some firm support to the part. More often the

5. A Harris brace is useful to control chronic or recurrent attacks by limiting painful movement. It should be fitted with reduced lumbar lordosis.

6. A traction cast is probably the most effective measure. Traction of 50 pounds is used during the day and 20 pounds at night, until the acute symptoms disappear. This usually requires a week. Graduated activities in the cast are then permitted during the day as tolerated. The cast is retained for a further 5

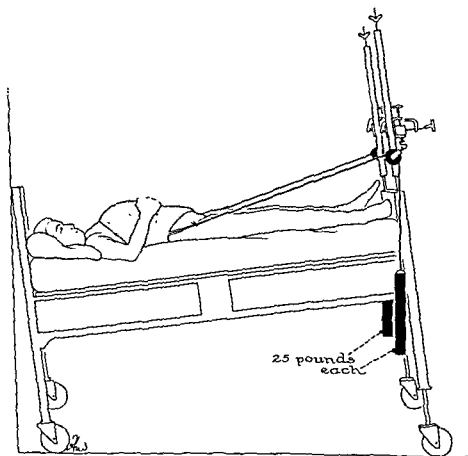


Fig 721.—Traction cast with 50 pounds continuous traction

position of flexion, with pillows under shoulders and knees, gives relief by positional reduction of the tension on the sciatic nerve

4. A flexion plaster corset is often effective. Lower lumbar flexion is obtained with the forward tilt of the pelvis when sitting. A cast applied in this position is worn for about 6 weeks. Exercises are started as soon as tolerated

weeks, with traction being used only at night. (See Fig 721.)

Operative.—Operative treatment is indicated:

1. When the symptoms are so severe or persistent that it is thought conservative measures will be ineffective. The underlying lesions are usually sequestered nuclear protrusions or very large protrusions of the annulus.

2. When adequate conservative treatment has failed to give relief.

3. When motor, sensory, and reflex changes are severe enough to threaten permanent nerve damage.

4. When restrictions about the heavy use of the back cannot be accepted.

The localization is confirmed by Pantopaque myelogram

An interlaminar approach is used, and the offending disc is excised. Usually sequestration of the nucleus or adhesions of the annulus fibrosus herniation, as a result of delay in adequate treatment, accounts for the failure of conservative treatment.

Incomplete removal of the intervertebral disc is to be avoided, as it could result in a further protrusion and recurrence of the symptoms. It is wise to finish up with a localized spinal fusion, as the long-term results are better. The writer prefers the posterior spinal fusion by an "H" graft. This maintains the normal posterior

relationships of the adjacent vertebrae, making it unnecessary to enlarge the intervertebral foramina. (The nerve root lies in the inferior part of the intervertebral foramen directly between the intervertebral disc anteriorly and the capsule of the apophyseal joint with the ligamentum flavum posteriorly.) The graft is under physiologic compression, which, by eliminating shearing strains, encourages bony union. In fact if one could be certain that there was no sequestration of the nucleus pulposus or mature adhesions, the fusion by this method would alone reduce an annulus herniation and effect a cure. The objective of the operation is to produce a permanent cure of both the nerve lesion and joint derangement. If the lesion is at L4-5, it is advisable to include the vulnerable L5-S1 space as well; otherwise a second operation will be necessary at this level in an average of 5 years. When the derangement involves the L5-S1 space, however, fusion of L4-5 is not routine.

FRACTURES AND DISLOCATIONS OF THE VERTEBRAL COLUMN

J GORDON PETRIE, M D

Emergency Management.—First aid to the person with an injury to the back or neck is of prime importance and may influence the end result. It is now generally recognized that a patient with an injured vertebral column must be moved as a whole without allowing shift of the injured part. The first doctor to examine the patient should carry out an examination of the sensory and motor functions of the extremities; tenderness and deformity of the spinous processes can be quickly detected. If shock is present, it should be treated while other examinations are being done.

The serious cervical injury presents special problems. Temperature must be watched and hyperthermia controlled. Impaired respiratory function makes it important to maintain a free airway. Simple suction may be necessary, and oxygen may lessen cyanosis. Light longitudinal traction with an improvised head halter will provide safety in transportation.

The best method of transporting the victims of thoracic and lumbar spine injuries is on

a padded board the size of the ambulance stretcher and securely lashed to the litter. Hypothermia may be present and the patient may require extra covering. Oxygen may help if there has been prevertebral hemorrhage causing impaired heart action. Catheterization and a rectal tube may reduce intra-abdominal pressure.

Serious associated injuries must not be overlooked. With sensory pathways impaired the patient may be unaware of other injuries and be unable not only to recognize them but even to help locate them. Complete notes are made and the time of the examination put down so that any change in the findings may be related to the examination and treatment.

Lumbar puncture may be done in cases showing gross neurologic damage. Lack of the normal cardiac and respiratory oscillations and rise of pressure in the manometer on jugular pressure signify that the spinal subarachnoid space is blocked. The inflation of a blood pressure cuff wrapped about the neck and pumped to various pressures provides con-

trolled, reproducible compression that permits differentiation between a partial and a complete block.

The x-ray study is as comprehensive as the patient's positioning permits. All views are taken for stereoscopic study in the anteroposterior, lateral, and often oblique positions. X-ray studies of other parts are taken if indicated.

Injuries to the Vertebral Column

Classification.—

1. Fractures of vertebral bodies
2. Fractures of muscular processes—transverse, spinous
3. Fractures of the neural arch—pedicles, articular facets, laminae
4. Dislocations of the cervical spine
5. Fracture-dislocation without paralysis
6. Fracture or fracture-dislocation with paralysis

Fractures of Vertebral Bodies

Etiology.—These injuries constitute the largest number of spinal fractures and are usually of the compression type. The fracture may be in any vertebral body, but the majority occur at the cervicothoracic and thoracolumbar junctions. They almost always occur with the spine in flexion, as in a fall from a height. It is quite common to see more than one vertebra involved with this type of injury, and occasionally there may be several normal between two or more compressed bodies.

Diagnosis.—The patient immediately complains of pain. There is muscle spasm of the erector spinae group. Prominence of a spinous process may be noticed, and localized tenderness on palpation of the involved vertebra or vertebrae is of definite significance. The roentgenograms must be as complete as the patient's positioning permits. Stereoscopic views are often valuable, especially where the neural arch is also involved. The lateral view shows irregularity of the vertebral body with a diminution in its vertical measurement. The intervertebral disc space is usually not narrowed. Compression fractures in the thoracolumbar area show more deformity than in other areas.

One must not overlook injuries to other parts of the body when a fracture of the spine has occurred, and x-ray studies of these parts should be taken if indicated.

Prognosis.—Healing of a compression fracture of a body is good, and if the deformity has been corrected, any resulting disability can be found in the muscles and ligaments.

Treatment.—One must always rule out a fracture of the pedicle or of an articular facet, in order to exclude the possibility of a fracture with dislocation. Reduction of the fracture by hyperextension is the procedure to be employed if the body is the only element involved. The technique of reduction can be carried out by one of the following methods: Davis, Rogers, or Watson-Jones. After the fracture has been reduced, a plaster corset is applied from the suprasternal notch to the symphysis pubis (see Fig. 722). Postreduction films must show perfect reduction of the deformity. Exercises are to be carried out during the immobilization, which is usually 4-6 months, depending on the severity of the fracture. The patient is allowed up and about within a few days of immobilization.

Late cases of fracture of the body or those improperly handled may be painful. These should be treated by exercises, to develop compensatory curves above and below the old injury, or by bone graft.

A serious complication of fractures of the lumbar vertebrae is retroperitoneal hemorrhage which may cause abdominal ileus.

Fractures of Muscular Processes

Spinous Processes

These are usually caused by direct violence.

Diagnosis.—Swelling and spasm of muscles occur with local tenderness at the site of injury. X-rays confirm the diagnosis and also indicate whether any other element of the spine is involved.

Treatment.—Rest for a few days, followed by exercises, is usually sufficient. In the cervical area, due to the weight of the head, a Minerva jacket may be necessary (Fig. 723).

Transverse Processes

These occur only in the lumbar area and are caused by muscular violence.

Diagnosis.—There is severe pain, especially on lateral bending of the spine, muscle spasm of the lumbar group, and local tenderness about 3" from the midline. Anteroposterior x-rays confirm the diagnosis.

Treatment.—Plaster immobilization with the body in the neutral position is carried out for 4-6 weeks. Exercises while in plaster should be done from the beginning. Union of the fracture is a fibrous one, and therefore reduction of the fracture is not attempted. Normal activity is encouraged early.

Fractures of the Neural Arch

Pedicles

Such fractures, when they occur, are usually in conjunction with those of other elements of the spine.

there is x-ray evidence of the spinal cord being encroached upon by bone, also if there is more than one element of the spine involved by the fracture. If these findings are present, it is felt that early exploration and internal fixation are necessary.

Dislocation of the Cervical Spine

Etiology.—The mechanism of production is one of hyperflexion of the spine. The dislocation may involve one or both facets and may be accompanied by varying degrees of injury to the spinal cord.

Treatment.—Skeletal traction by Crutch field's or Barton's tongs is the method of choice and has largely displaced the manual manipulative methods. (See Chapter 11, Neurosurgery.) The tongs are placed below

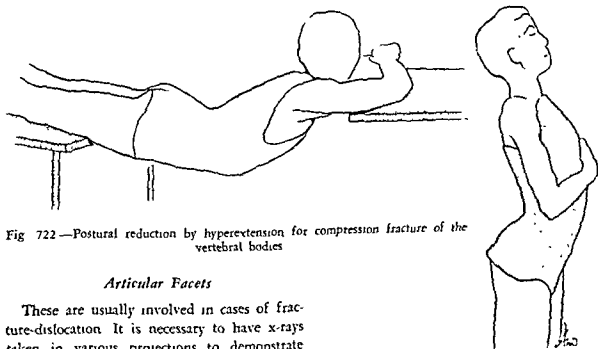


Fig 722—Postural reduction by hyperextension for compression fracture of the vertebral bodies

Articular Facets

These are usually involved in cases of fracture-dislocation. It is necessary to have x-rays taken in various projections to demonstrate these features.

Laminae

Direct violence is usually the cause of these fractures and the fragments may cause direct pressure on the spinal cord.

Treatment

One must consider whether there is a partial or complete subarachnoid block or whether

the parietal eminences and project into the outer table 3 mm. Following reduction, a Minerva jacket is applied with the head in moderate hyperextension. The jacket is worn for 2 months, after which a plaster or felt collar will give sufficient hyperextension for an additional month. X-rays must be repeated to see that the reduction is being maintained, and if it is not, a fusion operation will be necessary.

Fracture-Dislocation of the Spinal Column Without Neurologic Involvement

Etiology.—These injuries are usually caused by severe flexion or extension of the spine. The commonest findings are a compression fracture of a vertebral body with a fracture of one or both articular facets and a dislocation forward of the involved body upon the ones above and below. The pedicles, laminae, and spinous processes may be included in the fracture.

Nonsurgical Treatment.—For the cervical fracture-dislocations, skeletal traction applied

with the Crutchfield or Barton-Cone skull tongs is the best method. Halter traction is satisfactory only in the emergency management. Repeated x-rays are essential to follow the course of reduction. Manometric studies are to be carried out to see whether the traction is satisfactory when a subarachnoid block has been present.

For the thoracic and lumbar fracture-dislocations, traction on the shoulders and lower extremities can be tried with special apparatus. Manometric study should be repeated if a block has been previously demonstrated.

Surgical Treatment.—Exploration is indicated when spinal block is partial or complete. When the fracture involves more than one element of a vertebra, stabilization is required.

Exploration is carried out as soon as the condition of the patient permits. Traction is applied when the involved area has been exposed and the spinal cord is under direct inspection. When restoration of the vertebral canal is accomplished, internal fixation is secured by means of bone grafts.

Postoperative management is simplified due to the operation. A Minerva plaster jacket is used for cervical cases and bivalved casts for injuries lower in the spine.

When bladder control is lost, Munro's tidal irrigation is started at once. If urinary infection cannot be controlled by drainage, suprapubic cystostomy may be necessary.

Pressure sores can best be prevented by frequent turning; this is much safer after internal fixation of the spine.



Fig 723.—Minerva or cervicothoracic jacket. This cast includes the head, neck, and thorax, leaving the face, chin, and shoulders free for movement.

STATIC DISORDERS OF THE VERTEBRAL COLUMN

ALBERT A. BUTLER, M.D.

FIBROSITIS

Fibrositis, fibromyositis, and muscular rheumatism are terms used for pain in the soft tissues, characterized by muscle spasm and tenderness localized at "trigger points" or sites of maximum tenderness from which pain seems to be referred. Commonly there are associated tender nodules in muscle or subcutaneous tissues, which are called "fibrositic nodules."

Etiology.—The cause is unknown, lowered general resistance, exposure to cold, dampness or trauma, fatigue, mental anxiety, metabolic toxins, intestinal intoxication, bacterial toxins, gout, excessive exercise, faulty posture, endocrine disease, muscular disturbances, and so on are variously considered the cause. If we consider it a symptom complex rather than a clinical entity, we have a useful term which does not exclude a more accurate diagnosis if it can be established.

Pathology.—Sections of affected tissues show pathologic changes of a rather inconsistent nature. Inflammation of interstitial connective tissue of affected muscle, nodular in distribution, is described and is presumed to cause a spastic reaction in surrounding contractile tissue which becomes palpable. The subcutaneous nodules on biopsy appear to show fat herniation through a fibrous capsule, which on cross section is edematous and hemorrhagic.

Clinical Features.—The pain may be acute or insidious in onset. Muscular spasm is usually an outstanding feature. Certain "pain patterns" are recognized, such as *torticollis*, affecting sternomastoid, trapezius, and cervical muscles; *lumbago*, affecting lumbar and gluteal muscles, and so on. Posture is altered and movements are limited due to the muscle spasm. Passive movements are usually much freer than active ones if carried out slowly; if abrupt, they may precipitate an acute exacerbation. There may be reflex or sensory changes, and straining usually aggravates the pain.

Differential Diagnosis.—The determination of the source of pain is notoriously difficult. We must recognize that the same false localization and reflex muscular effects may occur from involvement of deep parietal structures and certain viscera. Fibrositis of the middle part of the left trapezius may produce an angular type of pain by virtue of T2, 3, 4 reflex. Involvement of cervical muscles commonly produces headaches. Fibrositis of dorsolumbar muscles may mimic appendiceal or ovarian pain, T9-12. Scalenus anticus involvement may resemble gall bladder disease, and C3, 4, 5 reflex via the phrenic nerve. Psoas fibrositis may resemble abdominal, genitourinary, or vertebral lesions.

These false localizations can be reproduced by the injection of irritating solutions at various levels into paravertebral tissues. At L1, for instance, pain will radiate to loin, inguinal, and scrotal regions, with rigidity and deep tenderness in lower abdominal wall, resembling renal colic.

Ischemia is an important factor in producing pain, and there is a great difference in the action to exercise, e.g., the intermittent indication of Buerger's disease and the pain of cardiac ischemia. If a tourniquet is used

to produce ischemia in a limb, there is much less pain at rest than with exercise, presumably due to a difference in the chemical changes in the tissues.

Investigation, to be adequate, must exclude visceral, neurologic, and skeletal disorders.

Treatment.—Heat, massage, analgesics, and local injections of Novocain are often successful in clearing up acute attacks. An exercise program and attention to general health are designed to prevent recurrences.

OSTEOARTHRITIS OF THE SPINE

Osteoarthritis of the vertebral column is generally taken to include traumatic and degenerative changes in the intervertebral and apophyseal joints. The two main types are hypertrophic spondylitis and spondyloarthritis.

Hypertrophic Spondylitis

This refers to changes in the intervertebral joints which are symphyses, and so the pathologic changes differ from those seen in diarthrodial joints. Synonyms are *spinal osteophytosis* and *degenerative disc disease*. The latter stresses one factor in a process which affects all the tissues of the joint. There is loss of the turgescence of the intervertebral disc, both with age and with degenerative processes. This dehydration of the disc is an important factor in its narrowing. As a result, there is interference with its function as a cushion between the vertebral bodies. Trauma to the adjacent bone then gives rise to peripheral ossification. This affects the bulging outer fibers of the annulus fibrosus. It probably lifts the periosteum at the attachment of these fibers and produces the hypertrophic reaction. The process is more marked anterolaterally than it is in the region of the anterior spinal ligament, presumably due to the protection that the ligament offers to the disc. The changes are absent or less marked posteriorly, because it is produced by a flexion mechanism. There is an associated reduction of spinal movement, and abnormal strains are thrown on the apophyseal joints; this may produce secondary degenerative changes there. Calcification of degenerated discs is occasionally seen.

Clinical Features.—Hypertrophic spondylitis is occasionally present to a very marked degree and entirely symptomless. Examination will show, however, that there is a good deal of limitation of spinal movement. Very often there is a complaint of pain, which is aching in character. Following strenuous exercise, or with the onset of fatigue, symptoms become more severe. If patients maintain one position for a prolonged period of time, it is difficult to start moving. After a short period of limbering up, there is a good deal of improvement. X-rays show marked beaking of the vertebral bodies with narrowing of the intervertebral space. This condition may be associated with a posterior protrusion of the nucleus pulposus, in which case that complica-

the cervical region placed lateral to the discs. Involvement of these joints is identical with that seen in other diarthrodial joints in the body. (See Chapter 37, Joints.) Histologically this is a true osteoarthritis. Degeneration of articular cartilage due to injury, rapid erosion due to overuse, slow erosion with the years, or vascular and metabolic disturbances predispose to or accelerate the process. The changes in the hyaline cartilage of the joint are quite marked before erosion to bone results. When there is bony contact, marked hypertrophic changes occur with the production of peripheral chondro-osteophytes. As one would expect from the pathologic changes, symptoms are present long before there is x-ray evidence of hypertrophic change.



Fig 724—Hypertrophic spondylitis

tion would predominate and require whatever treatment was necessary on its own merits. There may also be associated changes in the apophyseal joints. These would then probably account for the predominant symptoms.

Hypertrophic Spondyloarthritis

This is an involvement of the synovial joints of the vertebral column, which are mainly the apophyseal joints, but there are also the tiny intervertebral synovial joints in

Clinical Features.—Pain and stiffness are often severe and characteristically influenced by the weather. In fact, in many patients their joints act as an excellent barometer. A fall in pressure is associated with an exacerbation of their symptoms. Symptoms are worse in the morning but soon improve as the patients start moving about. They often discover for themselves that setting-up exercises before getting out of bed are beneficial. Symptoms are relieved by gentle exercise and aggravated by strenuous activities. The regions involved are

proportional to the sites of maximal strain. These occur most frequently at the apex of the normal curves. Lumbosacral and lumbar regions account for two thirds of the involvement; most of the others occur in the cervical region. Acute exacerbations of symptoms may produce marked muscle spasm. Reflex referral of pain occurs according to the innervation of the part concerned. Novocain infiltration may be used to relieve muscle spasm or as a paraspinal block for both diagnostic and therapeutic purposes.

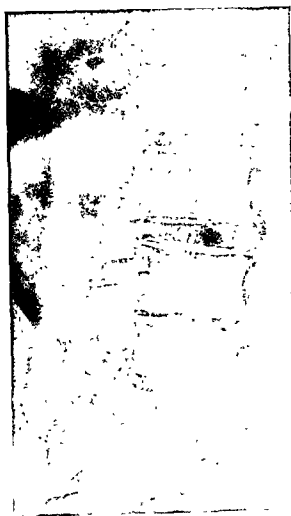


Fig. 725—Hypertrophic spondyloarthritis

Treatment.—

General Measures

1 Dietary directions may be necessary to deal with nutritional deficiency or alterations from the normal weight.

2. A regular exercise regime and regular hours of sleep should be established.

3 Mental attitude should be improved by the avoidance of worry and fatigue. The co-operation of family and associates is necessary to create a cheerful atmosphere.

4 Supplementary vitamin or hormone therapy is occasionally indicated.

5. Analgesics and sedatives are used as temporary expedients for rest and relief of pain.

Local Measures.—

1. Postural defects are corrected by instruction in remedial exercises.

2 Heat and massage are used for their soothing and palliative effects during an acute exacerbation.

3 Body casts may be used for a short period to give support while building up musculature.

4 A Harris lumbosacral brace may be used to limit painful movement.

5. Manipulation is often dramatic in giving temporary relief. Instability or nerve root involvement must be positively excluded. The principle is to place the involved joints in the positions of the extremes of their movement and then to stretch them slightly. The mechanism of relief is the snapping of light capsular adhesions.

6 Operative measures are worth while for severe and well-localized lesions. Occasionally an arthrectomy suffices and has the advantage of early mobilization. The vast majority, however, that come to operation require spinal fusion.

7. X-ray therapy is of doubtful value, but usually some temporary relief of pain is achieved.

ANKYLOSING SPONDYLITIS

This condition is characterized by obliteration of the sacroiliac joints, ankylosis of the apophyseal joints, calcification of spinal ligaments, and periphery of the intervertebral joints. It is often associated with involvement of peripheral joints. There are many synonyms. *Marie-Strümpell spondylitis* and *rheumatoid arthritis* of the spine being used fairly commonly. Others, such as *spondyloarthritis ankylopoietica* and *spondylose rhizomelique*, are occasionally used. The disease follows a fairly characteristic pattern. Ninety per cent of cases occur between the ages 15-35 years, and they

are nearly all males. It is sometimes seen in children, in association with Still's disease, which is childhood rheumatoid arthritis. It occurs in older persons as an exacerbation of the disease acquired during the usual age period. The cause of the disease is unknown. It is presumed to be an inflammatory process, as some focus of infection, recent or remote, can usually be established. There is probably an important allergic factor.

Clinical Features.—The onset is slow and characterized by pain and stiffness. There may be reflex referred pain simulating other conditions. Once started, the disease appears to be progressive, but the rate is variable, anywhere from 2-15 years, before it goes on to spontaneous remission. During this time there may

and the apophyseal joints. This is followed by involvement of the sacroiliac joint which is the first one recognizable radiologically. This is followed by calcification in the spinal ligaments and the outer layers of the annulus fibrosus. When the process is advanced the x-ray acquires the "bamboo spine" appearance. About half the cases show peripheral joint involvement to some extent. It is unusual to have involvement of the small joints of the hands and feet. In fact, the hips and knees are most commonly associated in the involvement. Histologically the process appears rheumatoid in nature. This does not explain the age and sex incidence, the response to treatment, the rarity of subcutaneous nodules, or the early calcification of periarticular tissues. Acute cases,

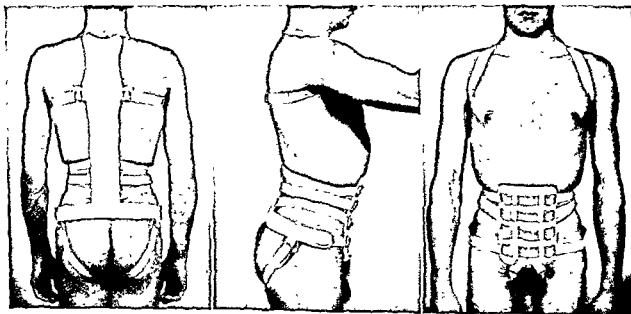


Fig. 726—Taylor brace shown in posterior, lateral, and anterior views. Useful for cases requiring support for lower dorsal, lumbar, and lumbosacral disorders.

be periods of acute exacerbation. The blood sedimentation rate is always high. Limitation of chest expansion and characteristic limitation of spinal movement are early features and may enable one to establish the diagnosis clinically before x-ray changes can be detected. The sacro-iliac joint is involved in 90% of cases. It is spared only when the disease is restricted to the upper vertebral regions. It would appear clinically that the small joints of the spine are first involved, namely, the *costovertebral*

accompanied by fever, inanition, and gross muscle wasting, may threaten life, but these are fortunately rare.

Treatment.—The general measures described for osteoarthritis of the spine are also applicable. X-ray therapy is of much greater value, and in the majority of cases it completely arrests the disease. It produces an apparent improvement due to the abolition of pain and muscle spasm. Two or three courses of therapy are necessary, at about three

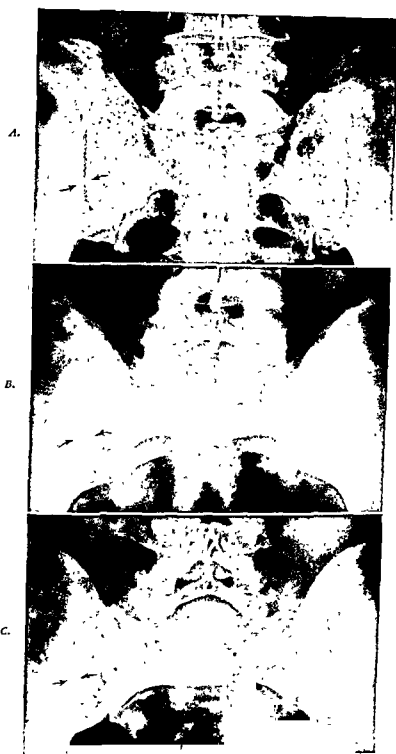


Fig 727 —Marie-Strümpell spondylitis (See opposite page for complete legend)

monthly intervals, in order to consolidate the cure. Corticosteroids have a limited use in the early cases. Occasionally in neglected cases or if the deformity appears progressive, a Taylor brace may be of value. Commonly, with adequate treatment, it is unnecessary, and if the disease is very active it is not well tolerated. A plaster bed with corrective felt pads may also be useful in the few cases in which the patients

do not respond readily to x-ray therapy. Surgery is necessary in neglected and late cases to correct deformity by osteotomy or by arthroplasty. The latter is most often indicated in the hip joint.

COCCYDYNIA

Coccydynia refers to pain in and around the coccyx and is therefore just a symptom

D.



E.

F.

Fig 727 (cont d) —Marie-Strumpell spondylitis.

A, X-ray of the normal sacroiliac joints for comparison

B, *C*, and *D*, Different degrees of sclerosis and obliteration of the sacroiliac joint. Compare with *A*

E, Bamboo appearance of the lumbar spine and also the two longitudinal lines from involvement of the facet articulations (railway track appearance)

F, Irregularly distributed calcification of the anterior longitudinal ligament.

The explanation of the rigid poker spine is apparent from these x rays

complex. It is often associated with trauma. There may be a fracture or there may be traumatic arthritis in the sacrococcygeal joint. Congenital variations in the structure of the coccyx may resemble an old fracture. Pain may be due to a soft tissue inflammatory process, either traumatic or infective in origin. The condition is commoner in females than in males. In some there are no localizing signs, and the painful coccyx is presumably psychosomatic in origin. If there is displacement of the coccyx, it may be palpated externally or by rectal examination or demonstrated in x-ray. Sacrococcygeal arthritis shows well-localized tenderness and aggravation of pain by movement of the joint. Pain with walking and exercise may indicate that the trouble lies in the soft tissue attachments of the coccyx.

Treatment.—

1 Protection from pressure may be obtained by using an air cushion or by sitting up straight on a hard chair. The weight is then taken on the ischium and the coccyx is relieved of any local pressure.

2 Short-wave diathermy and massage are occasionally beneficial.

3 Novocain infiltrations into the site of maximum tenderness are worth a trial.

4 In selected cases with a clear-cut abnormality and failure of conservative treatment, excision of the coccyx may help. It is important to avoid operation in a neurotic patient.

ADOLESCENT KYPHOSIS

This is a chronic disease producing a dorsal or dorsolumbar smooth kyphosis, commencing between the ages of 12-16 years. It may be symptomless or it may cause pain and fatigue. Occasionally the pain and stiffness are quite severe. The synonyms are *Scheuermann's disease*, *vertebral epiphysitis*, and *osteochondritis*. X-ray shows wedging of the vertebral bodies with quite a marked, smooth kyphosis. There appears to be fragmentation of the ring epiphysis and later on development of anterior marginal osteophytes. It has been thought that these changes are due to a vertebral epiphysitis and osteochondritis. It is now, however, believed that it is entirely secondary to changes in the intervertebral disc. Schmorl demonstrated

prolapse of the nucleus pulposus into the spongiosa of vertebral bodies in all his specimens of adolescent kyphotic spines, which came to dissection. This would result in loss of movement and tilting of the vertebrae. It is believed therefore that there are Schmorl's nodes at every level in the affected region. They are only seen in the x-ray if there is a reactive condensation of bone around the prolapse. This disorder is usually associated with shortened hamstring muscles.



Fig. 728—Adolescent kyphosis

Treatment.—

1. Mild cases are treated by head traction, using a head halter with 20 pounds for 20 minutes daily.

2. Intermediate cases may also require the use of a Taylor brace between exercise periods.

3. Severe cases may require recumbency for several months in a plaster bed, with gradual correction by felt pads, along with hourly hyperextension exercises. All patients probably benefit from using a plaster bed at night.

SENILE KYPHOSIS

Senile kyphosis is probably due to degenerative changes in the intervertebral disc. There may be associated nuclear protrusion or annulus bulging, but these would not be etiologic factors. There is a smooth upper and mid-dorsal kyphosis with narrowing of the intervertebral discs and a forward tilt of the vertebrae. The discs are thin and show loss of elasticity. There may be associated degenerative

as anterior fusion occurs. There may be a lot of distress in the intervening period. The remainder of the spine may be stiff. If compensatory curves cannot develop, the patient remains markedly stooped.

Treatment.—Heat, massage, and analgesics give symptomatic relief. Postural exercises are usually helpful. A Taylor brace in the day and a plaster bed at night are sometimes useful.

SENILE OSTEOPOROSIS

Senile, idiopathic, or postmenopausal osteoporosis is characterized by backache and increasing deformity as vertebrae collapse. It affects middle- or old-age persons. The decalcification is not necessarily generalized. Often the vertebral collapse occurs in several regions. The x-ray shows lozenge-shaped intervertebral discs due to the turgescence of the disc causing a smooth bulge into the soft spongiosa. It is apparently a disturbance of bone metabolism.

Clinical Features.—There are loss of movement and moderate backache during quiescent periods. Vertebral collapse causes acute exacerbations. If there is root involvement, pain may be intractable. Every movement causes severe muscle spasm and radiating nerve pain.

Treatment.—

1. Chronic cases are treated by hyperextension exercises, plus a plaster bed at night and a brace in the day.

2. Acute cases are treated by bed rest, with immobilization if necessary to relieve symptoms. Chordotomy for intractable pain may be considered.

3. High protein diet, vitamins, calcium, strontium, stilbestrol, and testosterone seem to help recalcification, but it is a slow process. Symptoms are relieved long before there is radiologic evidence of recalcification.

VERTEBRAL OSTEOPOROSIS

Vertebral osteoporosis (Calvé's disease) is an avascular necrosis in the vertebra. Presumably this is due to an infarct, but histologic studies have not been made. It is in the same group of disorders as Kienbock's disease in the semilunar, Kohler's disease in the tarsal scaphoid, etc. It affects only one vertebra, usu-



Fig 729 —Senile kyphosis

calification in some of them. Some of the vertebrae are in contact anteriorly and show fusion of the spongiosa across the intervertebral disc. Senile degeneration alone would not account for the condition. There is probably an added postural defect or occupational stress. The texture of the bone is normal unless there is an associated defect.

Clinical Features.—Pain may be local or radiating in character and is often worse when lying down. It is self-limiting, disappearing



Fig 730 —Senile osteoporosis



Fig 731 --Vertebral osteochondritis before and after treatment

ally lumbar or lower dorsal, in the 5- to 10-year age group. It is accompanied by kyphosis, pain, fatigue, tenderness, and muscular rigidity. The x-ray resembles Pott's disease of the rarer central body type. There is fragmentation with collapse of the vertebral body, but the appendages remain normal. There is tilting of the adjacent vertebrae, producing a sharp kyphosis, with the affected vertebral spinous process at the apex. The vertebral body may reduce to a wafer of bone, but with adequate treatment it becomes reconstituted to appear normal. It is distinguished from Pott's disease by the following:

- 1 Intervertebral disc space normal
- 2 No paraspinal abscess
- 3 Absence of systemic reaction
4. Negative tuberculin test

Treatment.—Bed rest in a hyperextension frame with four-point thoracic and pelvic traction, or a plaster bed with felt pads, is used till there is relief of pain and muscle spasm. A Castex corset, or spinal brace, is used until there is x-ray evidence of reconstruction of the affected vertebra.

SCOLIOSIS

Structural scoliosis is a condition in which there is a lateral curvature of the spine, associated with altered shape and rotation of the vertebrae. It is important to separate the so-called *functional scoliosis*, which is really only a postural defect, and the *simple unprogressive structural* cases, which are not significant, from the *progressive structural* ones, which if untreated go on to a horrible deformity.

Early recognition and careful follow-up is the only way to select intelligently the few cases that require radical treatment. A glance at the flexed spine in all routine examinations at school age is imperative and discloses even mild curves which should be noted and reviewed in 3 months. If there is any progression, a scoliosis record must be made. It is important to realize that there is, as yet, no substitute for continuous observation throughout the growth period. A curve may appear unprogressive during a period of slow spinal growth and then may progress rapidly and suddenly during a phase of rapid growth.

Steindler refers to three periods of rapid growth:

- 1 Birth to 2 years
- 2 5 to 10 years
- 3 Puberty to cessation

Closer observation is necessary at these times. When growth is complete, there is not likely to be any substantial change in the condition. In view of the fact that longitudinal growth of the vertebrae is by enchondral ossification, it is difficult to be sure just when it ceases. Risser has observed that ossification of the iliac apophysis is an accurate index. It ossifies anteriorly to posteriorly, and as it curves around the posterior superior spine to point toward the sacroiliac joint, vertebral ossification is complete. This varies between 12-19 years, and indicates the preferable time for spinal fusion if that is necessary.

Method of Recording.—If the periodic observations are reduced to a simple formula, it leads to more accurate records and is more easily understood if there are different observers. The x-rays should include anteroposterior views in the standing, sitting, and supine positions, extending from the iliac crests to occiput, each on one long film. A clinical photograph with the bony landmarks outlined is also taken. The *curves* are named according to the region and the side of their convexity. The apex and the limits of the curve are noted. The curve is measured by an empirical method, the last intervertebral surfaces still pointing into the concavity form the base line, and perpendiculars are drawn from these. The angle toward the axis of the spine is then measured.

Wedging is noted on a scale of 1 to 4 plus, measurements being made on the vertebra showing maximal wedging, and this is usually at the apex of the curve.

0 to $\frac{1}{6}$	= +
$\frac{1}{6}$ to $\frac{1}{3}$	= ++
$\frac{1}{3}$ to $\frac{1}{2}$	= +++
More than $\frac{1}{2}$	= ++++

Rotation of the vertebrae is also recorded where maximal, usually at the apex. It is positive if to the side of the concavity and negative if to the side of the convexity. It is measured by the relationship of the tip of the spinous process to the edge of the vertebral body.

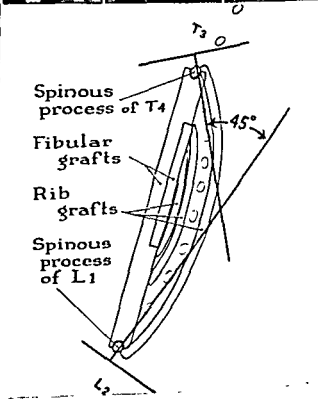
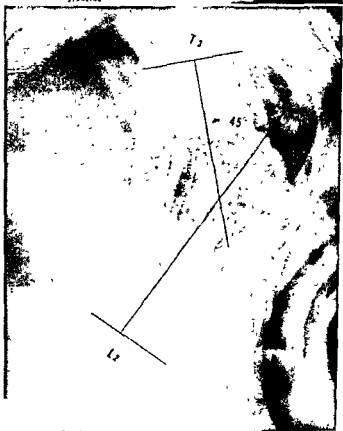
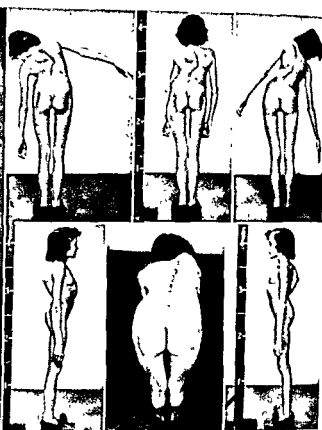
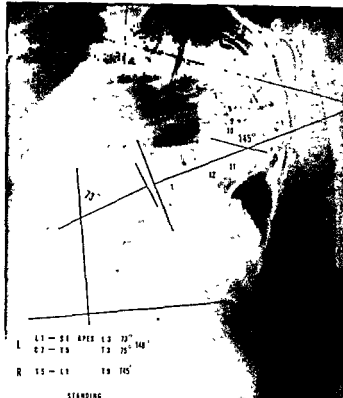


Fig 732—A, Right thoracolumbar scoliosis showing measurement of primary and secondary curves (idiopathic type)

B, The photographs show the marked thoracic cage deformity, kyphosis, and lordosis which accompany the rotational elements of a severe scoliosis

C, Correction obtained by an operation to mobilize the primary curve and turnbuckling a hinged cast.

D, The correction is maintained by bone grafts as illustrated

To concavity	To convexity
0 to $\frac{1}{3}$ = (+)	(-)
$\frac{1}{3}$ to $\frac{2}{3}$ = (++)	(--)
$\frac{2}{3}$ to edge = (+++)	(---)
Beyond edge body = (++++)	(----)

Usually there are three curves—the middle one being primary and the other two secondary. They are compensated when the sum of the curves to opposite sides is equal. The primary curve shows the most structural change and is the larger curve. Double primary curves are seen occasionally, being equal in all respects.

With high curves, *bending films* are necessary to determine the mobility of the curves. With lumbar scoliosis, this is done by the *pelvic tilt test*, the film being taken with a 3" lift under one buttock and then the other. If there is residual angulation in the compensatory curves, the total determines the limit of desirable correction in the primary curve. It is important to avoid overcorrection. Partial correction may be of remarkable value, if it restores compensation of the residual curves.

Types of Scoliosis.—

Functional scoliosis is a postural defect which can be voluntarily corrected (See postural defects.) Adaptive changes in the structure of vertebrae are absent or minimal. This differs from scoliosis due to a short leg or to other deformities of the lower extremities. If the postural defect or the deformity of the lower extremities is corrected, the scoliosis is no problem.

Structural scoliosis is due to an intrinsic defect in bone, nerve, or muscle. It cannot be voluntarily maintained in a corrected position.

Classification according to etiology is customary and the approximate incidence is noted.

- 1 *Muscle* (a) Idiopathic—80%
(b) Thoracogenic—2%
- 2 *Bone* (a) Congenital anomalies—3%
(b) Osteochondrodystrophy (Morquio's disease)—1%
- 3 *Nerve* (a) Poliomyelitis—10%
(b) Neurofibromatosis—2%
(c) Friedreich's ataxia—1%
(d) Spastics—1%

Clinical Features.—Very often scoliosis is unrecognized until someone remarks that there is a prominent hip or a high shoulder, or per-

haps it is picked up in a school medical examination. Commonly it gives no symptoms in youth. So long as mobility and good muscular strength are present, compensation for the deformity appears adequate. Later there is likely to be degenerative change in the joints at the sites of maximal curvature. In very severe deformity, cord involvement with paraplegia can occur. With extreme curves there is secondary visceral embarrassment, which shortens life. This is statistically evident in curves over 50 degrees, is proportional to the severity of the curve, and is most harmful in the thoracic region. It is therefore imperative that the condition be treated before irreversible changes occur, with such dire consequences.



Fig. 733.—The author's distraction brace.

Treatment.—

Palliative—Exercises improve the sense of well-being and compensate for the ill effects of the curvature, but they do not appreciably or permanently alter the curve.

Corrective.—

1. *Rest in recumbency* will produce appreciable correction. Albee used a Bradford frame

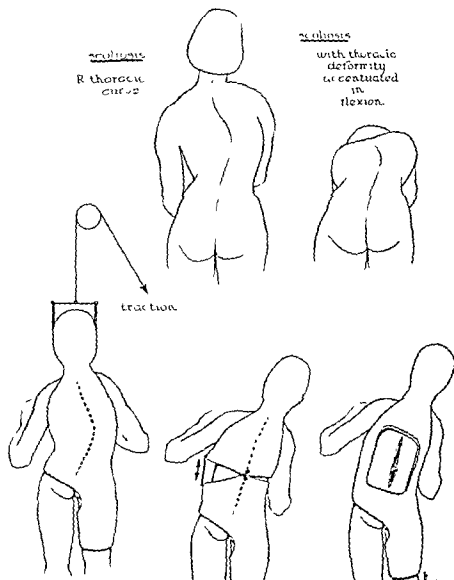
with three canvas bands to give a cross pull against the curvature.

2. A *distraction brace* gives traction on the spine between the pelvis and the occiput. It is limited by the pressure that can be tolerated on the chin and occiput but is effective in very young children. The Milwaukee brace uses fixed distraction; the Butler brace uses four elastic distraction units attached by ball-and-

socket joints to the headpiece, allowing some spinal movement.

3. *Reverse curve plaster jacket* is used on the principle that pressure against the curve may alter growth by compression. It does not appear to do this to an appreciable extent.

4. *Plaster casts* applied in corrective positions with and without crutch supports are of doubtful value. Often there is marked progres-



sion of the curvature in spite of such appliances.

Operative Treatment.—It is generally recognized that a mature spinal fusion is the only means of maintaining a correction of structural scoliosis.

Opinions differ widely, but following are Cobb's indications for spinal fusion:

1. A severe scoliosis usually over 50 degrees and correctable at least 20 degrees, in patients preferably 12-21 years of age
2. A lesser curvature which is increasing under conservative management and will become severe.
3. Unstable scoliosis, preferably in patients 13-14 years of age
4. Pain which can be relieved by fusion, this is usually in the older patient

In some cases one is forced to fuse at an early age, in which case a short fusion is done as a compromise, and it is extended after vertebral growth ceases. This is more likely to be necessary in paralytic curves and in some congenital ones with multiple anomalies.

There is a variety of methods used for correction of the curvature prior to spinal fusion:

1. **Risser Jacket**—A corrective cast is applied by
 - (a) Head traction
 - (b) Albee-Compere derotation table
 - (c) Other methods

Hinges are incorporated back and front over the apex of the curve. Turnbuckles are attached and correction carried out as tolerated. When the desired position is obtained, the cast is reinforced and a large window is cut out over the spine for the fusion. This is preferred for high and rigid curves.

2. **Transection Jacket**—In dealing with double primary curves or mobile curves, the cast may be immediately sectioned and placed in maximal correction. The cast is reinforced in this position, the window cut, and operation proceeded with.

3. **LeMesturier Hammock**.—This is an ingenious method using a fish-net hammock. The patient is suspended by overhead pulleys in the hammock, with suspension from the

upper arm and leg as well, giving a four-point suspension. When the correction is considered adequate, a padded cast is applied. The posterior window can then be cut and operation proceeded with.

The corrective jackets are usually retained for 6 months and some protection used for a further 6 months.

INFECTIOUS DISORDERS OF THE VERTEBRAL COLUMN

More than half of all bone and joint tuberculosis occurs in the vertebral column. By contrast, pyogenic infections of the spine are extremely rare, accounting for a very small proportion of all the cases of pyogenic osteomyelitis. Brucellosis, actinomycosis, typhoid

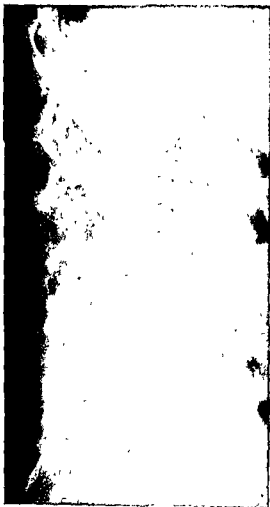


Fig. 735.—Tuberculosis of T12 with collapse.

spine, and gonococcal and syphilitic arthritis are all rarities

Tuberculosis of the Spine

Pott's disease may occur at any age, but it is most common in the young. The involvement of the spine is secondary to tuberculosis elsewhere in the body, usually a tuberculous lymphadenopathy. Minor trauma, or a localized degenerative change due to the systemic toxemia, predisposes to an endarteritis, giving rise to the initial focus. In the spine, this commonly

2. It may follow the course of muscles, nerves, and blood vessels, to reach long distances.

3. It rarely extends into the vertebral canal, giving rise to paralysis.

The cold abscess at a distance from the diseased area is found in about 20% of cases. It may account for a swelling anywhere between the occiput and the heel, but the following are the commoner types:

1. *Retropharyngeal abscess*, arising in a cervical vertebra, perforating the prevertebral

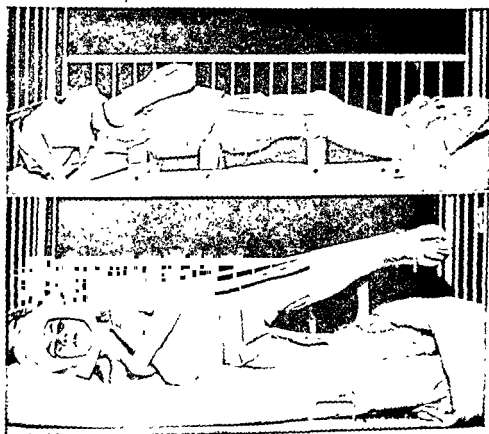


Fig. 736.—Plaster bed for treatment of tuberculosis

occurs in the vertebral body beneath the hyaline cartilage. This epiphyseal involvement accounts for two thirds of the cases. The periosteum, central vertebral body, and appendages are involved with descending frequency. Subsequent events depend on virulence, resistance, localization, complications, and treatment. Caseation in the vertebral body permits collapse and expression of tuberculous debris, forming a cold abscess.

1. This may form a paraspinal abscess

fascia, and pointing at the edge of the sternomastoid muscle.

2. *Intercostal abscess*, originating in a thoracic vertebra and following nerves or vessels.

3. *Psoas abscess*, from a lumbar vertebra, passing along the iliopsoas to the inguinal ligament on either side of the femoral artery.

Clinical Features.—The onset of the disease is often insidious, particularly in children who appear just out of sorts. The presence of a swelling, which investigation shows to be a

cold abscess, or even the development of a sharp kyphosis may be the first incidents that lead the individual to seek advice. Occasionally low back pain, with acute sciatic radiation, occurs and closely simulates the symptoms of a prolapsed intervertebral disc. Inquiry elicits the symptoms of systemic involvement, loss of stamina, malaise, loss of weight, and possibly night sweats. Localization may be indicated by a tired ache or tenderness, or the pattern of referred pain may indicate the nerve root involvement. Kyphosis, localized rigidity, tenderness, or hyperesthesia may be noted. The x-ray in advanced cases shows collapse of the affected vertebral bodies and a paraspinal abscess. With early involvement, the loss of intervertebral space may be difficult to interpret, but this is soon followed by decalcification, with a blurring of bony trabeculation. The hemogram may show increased sedimentation rate, lymphocytosis, and leukopenia. Muscle spasm is always present, and there is painful limitation of spinal movement. A young child will pick up an object by crouching on the hands and knees instead of bending the back. Paraplegia is a serious complication which is not uncommon when the disease is in the dorsal region.

Treatment.—It is essential that treatment be correlated with an understanding of the disease in all its phases.

1 *Systemic treatment* by bed rest, controlled exposure to sun and wind, and intelligent diet is as fundamental as adequate immobilization of the diseased area. Mental health is achieved by education, occupational therapy, and an interesting environment, with positive training in the philosophy of contentment. Supportive therapy, iron, vitamins, blood transfusions, etc., and antibiotics are used as required. The response to this regime is more effective in children than in adults, and therefore surgery as an aid to recovery is used accordingly.

2 *Streptomycin and other antituberculosis drugs* systemically and locally in wounds and sinuses have improved the scope of surgery in Pott's disease.

3. *Arthrodesis* of the affected region achieves a more effective local immobilization than external means but does not in itself cure the disease. Delay for several months is

essential in early disease to determine the number of vertebrae involved. Multiple involvement is a distinct possibility. Fusion is a delayed aid to recovery, only becoming effective when union is sound. One must be sure that the response to the general regime will be adequate for several months to make the procedure worth while. Operation, when performed too early, increases the mortality rate; if too late, just prolongs the illness! Bradford frames or similar orthopedic appliances are commonly used to maintain the child in the position of hyperextension. Plaster beds with

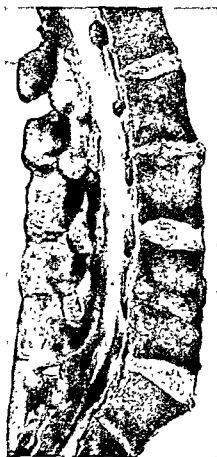


Fig 737—Museum specimen showing tuberculosis of lumbar spine

turning shells are adaptable to any age or condition. There are many methods of spinal fusion based on the original Albee and Hibbs procedures. The most popular are twin tibial grafts and "H" shaped iliac grafts. When the systemic disease is arrested and the vertebral lesion is recalcified, graduated convalescence

is permitted This consists of recumbency without plaster, increased exercises, sitting up in bed, and so on, to a gradual resumption of full activity over several months. Close supervision is required to guard against a possible relapse

Excision of the Nidus of Infection.—Extirpation of an abscess with bony and cartilaginous sequestra is performed when simpler measures fail or when, due to great size, chronicity, or mixed infection, a conservative approach is likely to lead to failure.

Diodrast may be used to outline the size and contour of the abscess cavity Tryptic enzymes such as Varidase have been used to remove inspissated exudate from a draining sinus prior to this examination It is used, but in smaller dosage, when there is a closed abscess, because of the severe reactions it may produce. When the abscess wall is totally excised, the vertebrae and discs are then exposed, and carious material can be removed Cancelous bone chips and streptomycin may then be inserted as an aid to anterior fusion

Antibiotics and antituberculosis drugs are efficient protection against dissemination of the disease by the operative procedure Permanent healing is less likely with incision and drainage because of constriction within the tract and irregular pouching, preventing free drainage There may also be retained sequestra at the primary focus or along the sinus.

Sacroiliac Tuberculosis

Tuberculosis of the sacroiliac joints is not uncommon It is chiefly seen in young adults and is serious if associated with multiple lesions elsewhere in the body. Multiple sinus formation is also an unfortunate characteristic in this region

Clinical Features.—The diagnosis is usually missed until a painful limp develops Pain may be localized to the sacroiliac region, but very often it is a diffuse low back pain. Occasionally it may be referred into the gluteal region and posterior aspect of the spine A sciatic type of scoliosis is often seen Either this or the muscle spasm in the gluteal region accounts for the limp Abscesses are commonly seen and may present anywhere around the pelvis, most frequently pointing posteriorly

over the joint. The usual systemic manifestations of the disease are present. X-rays show erosion of the joint surface and a zone of decalcification around this, but in the early stages it is difficult to detect any change

Treatment.—Systemic treatment is the same as for tuberculosis elsewhere in the body. A sacroiliac joint is immobilized by the use of a plaster spica If there are sinuses, a plaster bed is used Streptomycin locally as well as systemically is very effective in obtaining healing of the sinuses and quiescence of the disease This is followed by arthrodesis of the joint by one of the extra-articular methods

Osteomyelitis of the Spine

Pyogenic infections of the vertebrae are most commonly due to *Staphylococcus aureus*. It is usually a blood-borne infection, rarely a complication of lumbar puncture, spinal operation, or injury It may occur as a complication of septicemia in children, or it may localize in the vertebral body or the appendages of the vertebra as the result of a bacteremia *Acute cases*, usually in the appendages, are characterized by fever, local tenderness, muscular spasm, and swelling of the overlying soft tissues Oblique x-rays may show involvement of the interarticular joints The *chronic type* is more often in the vertebral body, is slow in onset, and resembles tuberculosis. Blood culture, hemogram, and tuberculin test often help to establish the diagnosis The x-ray changes are late, they first show narrowing of the intervertebral space and then collapse of the vertebral body Subsequent sequestration and reactive changes in the bone, with thickening and bridging of the vertebrae, become characteristic The complication of mediastinal abscess, empyema, suppurative pericarditis, or extradural abscess may be serious.

Treatment.—Antibiotics are used in large dosage, combined with general supportive measures such as blood transfusions and parenteral fluids The spine is protected in a plaster cast or plaster bed Abscesses may have to be drained, and if there is sequestration in an inaccessible location, drainage may persist. The hypertrophic reaction to the disease usually makes spinal fusion unnecessary, although it has been used in some of the chronic cases to give local support.

Gonorrheal Spine

Gonorrheal arthritis is not uncommon, but its occurrence in the spine is rare. It is suspected when there is pain and rigidity of the vertebral column following a neisserian urethritis. The likelihood is increased if other joints are involved and if the complement fixation test is positive. It may produce a marked ankylosis.

Treatment.—The nidus of infection in the genitourinary tract must be eliminated first. Antibiotics, particularly penicillin, and the sulfonamides are of inestimable value. Local treatment of the spinal lesion may be symptomatic, with bed rest, followed by a plaster corset, until completely cleared.

Syphilitic Spine

(See Fig 562)

Syphilis is becoming a rare disease, and syphilitic involvement of the vertebral column is a great rarity. There is a variety of possible lesions:

1. Localized periostitis
2. Congenital syphilitic epiphysitis
3. Syphilitic osteochondritis
4. Gummatous lesions
5. Extension from a syphilitic ulcer
6. Neurotrophic changes producing a Charcot spine

Cervical involvement is most likely by extension from a syphilitic pharynx. It is said to simulate Pott's disease. Other stigmas of syphilitic infection—Hutchinson teeth, nasal catarrh, suppurative otitis, interstitial keratitis, blood Wassermann, and x-ray appearance—help to establish the diagnosis.

Treatment.—The systemic disease is treated by penicillin and other antisyphilitic drugs as indicated. The local condition may require mechanical support. If there is instability, as with a Charcot spine, spinal fusion is indicated.

Other Infections of the Spine

1. *Typhoid fever* sometimes produces a periostitis and osteitis of the spine.
2. *Undulant fever* may produce abscesses in the vertebral body and intervertebral disc, or

a hypertrophic periostitis. The general symptoms of the disease, plus *Brucella agglutination*, low white count, fever, and high sedimentation rate, aid diagnosis.

3. *Actinomycosis* of the spine shows extensive bone destruction and numerous draining sinuses. Biopsy of the granulation tissue, with discovery of the ray fungus, establishes the diagnosis. Treatment is with penicillin, sulfonamides, usually combined with surgical drainage and mechanical support of the spine. Occasionally iodides and/or x-ray therapy are beneficial.

4. *Blastomycosis* resembles Pott's disease; the diagnosis is established by biopsy and microscopic findings. The prognosis is poor.

5. *Echinococcus cysts* of the spine require resection of the part, followed by mechanical support.



Fig 738—Metastatic carcinoma of L2 with collapse

NEOPLASTIC DISORDERS OF THE VERTEBRAL COLUMN

Tumors of the vertebral column are rather rare. Those that do occur are mostly malignant and usually metastatic. Metastases occur from the prostate, breast, kidney, gastrointestinal tract, female genital organs, thyroid, and lung, in descending order. The preponderant opinion is that the tumors are blood-borne, the malignant emboli lodging in marrow capillaries.

Primary Benign Tumors

1. Osteomas.
2. Osteochondromas—these are usually symptomless and no treatment is required.
3. Chordomas—the remnants of the embryonic notochord.
4. Hemangiomas—usually in one vertebral body, common but rarely give symptoms; x-ray shows coarse trabeculation.
5. Dermoids, in connection with spina bifida occulta.

Primary Malignant Tumors

1. Fibrosarcoma—resembles Pott's disease, but there is more rapid destruction.
2. Endothelioma—this shows hematogenous metastases.
3. Multiple myelomas—these are recognized by plasma cells in a marrow biopsy, Bence Jones protein in the urine, and x-ray showing collapse of the vertebral bodies.

Metastatic Malignant Tumors

1. Carcinomas—there is gradual erosion of vertebrae with collapse.

2. Sarcomas—the erosion is more rapid.
3. Multiple myelomas.

The treatment of malignant tumors is symptomatic. Occasionally mechanical or operative support is indicated. Chordotomy is occasionally used to relieve severe pain.

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Title	Running Time	Sound or Silent	Procureable From
Surgical Approaches to the Joints of the Spine and Sacroiliac (1952) (By LeRoy C. Abbott, M.D., Donald C. Smith, M.D., and J. B. de C. M. Saunders, M.B., San Francisco)	28 min	Sound Color	Central Office Film Library, Veterans Administration Vermont Ave. and H St., N.W. Washington 25, D. C.

Film References—Cont'd

<i>Title</i>	<i>Running Time</i>	<i>Sound or Silent</i>	<i>Procurable From</i>
Scoliosis Method of Correction and Fusion (Shows the steps taken in correcting a case of idiopathic scoliosis and carefully documents each stage in the progress of one patient) (1951) (By Philip D. Wilson, M.D., and John R. Cobb, M.D., New York)	28 min	Sound Color	Sturgis-Grant Productions, Inc. 322 E. 44th St. New York 17, N. Y.
Protruded Cervical Intervertebral Disk Diagnosis and Treatment (1951)	10 min	Silent Color	J. Grafton Love, M.D. Rochester, Minn.
Sciatic Pain and the Intervertebral Disk (1945)	25 min	Sound Color	Audio Visual Training Section Bureau of Medicine and Surgery U. S. Navy Department Washington 25, D. C.

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